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)

Copyright 2011 American Society of Clinical Oncology
### Special Award Lecture Abstracts

Abstracts will be presented on June 14 at 9:00 a.m.

**1s**

### Plenary Abstracts (LBA1 – LBA5)

Abstracts will be presented on June 13 through June 16.

**5s**

### Trials in Progress Poster Session

Scheduled presentations (Abstracts TPS100 – TPS249)

**7s**

### Breast Cancer—HER2/ER

Scheduled presentations (Abstracts 500 – 636)

**45s**

### Breast Cancer—Triple-negative/Cytotoxics/Local Therapy

Scheduled presentations (Abstracts 1000 – 1135)

**80s**

### Cancer Prevention/Epidemiology

Scheduled presentations (Abstracts 1500 – 1604)

**114s**

### Central Nervous System Tumors

Scheduled presentations (Abstracts LBA2000 – 2101)

**140s**

### Developmental Therapeutics—Clinical Pharmacology and Immunotherapy

Scheduled presentations (Abstracts CRA2500 – 2614)

**165s**

### Developmental Therapeutics—Experimental Therapeutics

Scheduled presentations (Abstracts 3000 – 3106)

**194s**

### Gastrointestinal (Colorectal) Cancer

Scheduled presentations (Abstracts 3500 – 3636)

**221s**

### Gastrointestinal (Noncolorectal) Cancer

Scheduled presentations (Abstracts 4000 – 4132)

**256s**

### Genitourinary Cancer

Scheduled presentations (Abstracts 4500 – 4670)

**289s**

### Gynecologic Cancer

Scheduled presentations (Abstracts 5000 – 5111)

**332s**

### Head and Neck Cancer

Scheduled presentations (Abstracts 5500 – 5594)

**360s**

### Health Services Research

Scheduled presentations (Abstracts 6000 – 6136)

**384s**

### Leukemia, Myelodysplasia, and Transplantation

Scheduled presentations (Abstracts 6500 – 6632)

**419s**

### Lung Cancer—Local-Regional and Adjuvant Therapy/Small Cell

Scheduled presentations (Abstracts 7000 – 7091)

**453s**
Lung Cancer—Metastatic/Non-small Cell
Scheduled presentations (Abstracts 7500 – 7611) ................................................................. 476s

Lymphoma and Plasma Cell Disorders
Scheduled presentations (Abstracts 8000 – 8086) ....................................................................... 504s

Melanoma/Skin Cancers
Scheduled presentations (Abstracts 8500 – 8597) ....................................................................... 526s

Patient and Survivor Care
Scheduled presentations (Abstracts 9000 – 9140) ....................................................................... 550s

Pediatric Oncology
Scheduled presentations (Abstracts 9500 – 9581) ....................................................................... 585s

Sarcoma
Scheduled presentations (Abstracts 10000 – 10098) ..................................................................... 605s

Tumor Biology
Scheduled presentations (Abstracts 10500 – 10632) ..................................................................... 630s

Author Index .................................................................................................................................. 664s
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)
Journal of Clinical Oncology (ISSN 0732–183X) is published 36 times a year, three times monthly, by the American Society of Clinical Oncology, 2318 Mill Road, Suite 800, Alexandria, VA 22314. Periodicals postage is paid at Alexandria, VA, and at additional mailing offices.

Postmaster: Send all changes of address for Journal of Clinical Oncology subscribers to:
JCO Customer Service
2318 Mill Road, Suite 800
Alexandria, VA 22314

Editorial Correspondence (manuscript-related inquiries):
Daniel G. Haller, MD, Editor-in-Chief
Journal of Clinical Oncology
2318 Mill Road, Suite 800
Alexandria, VA 22314
Telephone: 703-797-1900; Fax: 703-684-8720
E-mail: jco@asco.org; Internet: www.jco.org

American Society of Clinical Oncology membership-related queries should be addressed to:
ASCO Member Services
2318 Mill Road, Suite 800
Alexandria, VA 22314
Telephone: 703-299-0158; Toll-free: 888-282-2552
Fax: 703-299-0255
E-mail: membermail@asco.org; Internet: www.asco.org

Hours: Monday-Friday, 8:30 a.m.-5:00 p.m. Eastern Time

Customer Service, Subscriptions, and Changes of Address:
JCO Customer Service
2318 Mill Road, Suite 800
Alexandria, VA 22314
Telephone: 703-519-1430; Toll-free: 888-273-3508; Fax: 703-518-8155
E-mail: jcoservice@asco.org
Internet orders/renewals: www.jco.org/subscriptions

Orders and Payments
P.O. Box 37211
Baltimore, MD 21279-3211

2011 Subscription Rates

<table>
<thead>
<tr>
<th>Category</th>
<th>Domestic (Print + Online)</th>
<th>Domestic (Online Only)</th>
<th>International (Print + Online)</th>
<th>International (Online Only)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Individuals — 1 year</td>
<td>$578.00</td>
<td>N/A</td>
<td>$802.00</td>
<td>N/A</td>
</tr>
<tr>
<td>Individuals — 2 years</td>
<td>$1,098.00</td>
<td>N/A</td>
<td>$1,524.00</td>
<td>N/A</td>
</tr>
<tr>
<td>Individuals in training</td>
<td>$289.00</td>
<td>N/A</td>
<td>$401.00</td>
<td>N/A</td>
</tr>
<tr>
<td>Tier 1 Institutions</td>
<td>$849.00</td>
<td>$735.00</td>
<td>$1,174.00</td>
<td>$735.00</td>
</tr>
<tr>
<td>Tier 2 Institutions</td>
<td>$989.00</td>
<td>$856.00</td>
<td>$1,312.00</td>
<td>$856.00</td>
</tr>
<tr>
<td>Tier 3 Institutions</td>
<td>$1,427.00</td>
<td>$1,236.00</td>
<td>$1,740.00</td>
<td>$1,236.00</td>
</tr>
<tr>
<td>Tier 4 Institutions</td>
<td>$1,573.00</td>
<td>$1,361.00</td>
<td>$1,885.00</td>
<td>$1,361.00</td>
</tr>
<tr>
<td>Tier 5 Institutions</td>
<td>Call for quote</td>
<td>Call for quote</td>
<td>Call for quote</td>
<td>Call for quote</td>
</tr>
</tbody>
</table>

Important Tiers and Pricing Notes
1. All tier assignments are subject to periodic evaluation by the Publisher.
2. Institutions are assigned to Tiers 1-3 if they operate from a single site at a single geographic location. A single institution with more than one site or location (eg, a research university with a teaching hospital or with multiple geographically dispersed campuses) is classified as Tier 4. Individually negotiated multisite licenses (Tier 5) are required by consortia, corporations, and other institutions having shared network access.
3. Prices are in effect from September 1, 2010, through August 31, 2011. Prices are subject to change.
4. Print-only subscriptions or additional print subscriptions are available for $702 in the US and $1,014 outside the US.
5. Institutional online access, whether an online-only or bundled subscription, is for a single-site license, which allows an unlimited number of concurrent users from that site.
6. For multisite licenses, please contact the appropriate agent for a quote.
7. Subscribers outside the US, add $100 per print subscription for expedited delivery.
9. Prices quoted are in US dollars and payments must be made in US dollars.
10. Except on Tier 5 orders, the publisher allows for a 5% discount to recognized subscription agents.

2011 Institutional Subscription Tiers

Tier 1
- Primary or secondary school
- Private practice office or clinic
- General reference, nonresearch public library

Tier 2
- Associate, undergraduate, or masters degree–level college or university
- Residency program, nursing school, or allied health training program
- Community or military hospital or clinic
- Independent research library
- Health policy or advocacy nonprofit or charitable organization
- Local or regional government agency or ministry
- Professional society, trade union, or industry trade association

Tier 3
- Medical or pharmacy school
- Doctorate-granting research university
- Teaching or research hospital
- Nonprofit, nongovernmental research institute
- Local government agency or ministry
- Government laboratory
- Local, independent, for-profit organization (eg, consulting partnership or law firm)

Tier 4
- Regional health care network
- Regional government agency or ministry
- Regional corporation
- Multicampus, centrally administered academic institution

Tier 5
- Consortia
- National health care network
- Regional or national university network
- National government agency or ministry
- Government research institute or national research library
- National or multinational corporation
Prices are subject to change without notice. Current prices are in effect for back volumes and back issues. Single issues, both current and back, exist in limited quantities and are offered for sale subject to availability. Back issues sold in conjunction with a subscription are on a prorated basis.

USA, Canada, Europe, and India Licenses and Consortia
David Charles
eLicensing
92 Avenue du General de Gaulle
78600 Maisons-Laffitte, France
Telephone/Fax: +33-1-39-12-29-29
E-mail: dc.licensing@orange.fr

Japan
USACO Corporation
2-17-12 Higashi-Azuba Minato-ku
Tokyo, Japan 106-0044
Telephone: +81-3-3505-3529; Fax: +81-3-3505-6284
E-mail: import@usaco.co.jp; Internet: www.usaco.co.jp

Maruzen Co., Ltd.
Subscription Department
4-13-14 Higashi Shinagawa, Shinagawa-ku
Tokyo, Japan 140-0002
Telephone: +81-3-6367-6047
Fax: +81-3-6367-6160
E-mail: Journal@maruzen.co.jp

China: Orders should be placed through:
Charlesworth China
Beijing Modern Palace Building, 12th Floor
No. 20, Dongsanhuan Nanlu
Chaoyang District
Beijing 100022
PR China
Telephone: +86-10-6779-1601; Fax: +86-10-6779-9806
E-mail: sales@charlesworth.com.cn
Internet: www.charlesworth.com.cn (in Mandarin)
and www.charlesworth.com

Indonesia, Malaysia, Philippines, Singapore, South Korea, Thailand, Taiwan, and Vietnam
EBSCO EMpact
5724 Highway 80 East
Birmingham, AL 35242
Telephone: +1-205-980-6676
Fax: +852-2573-8822
E-mail: jnccdaniel@ebSCO.com

Central/South America, The Caribbean
Accucoms (US), Inc
West Point Commons
1816 West Point Pike, Suite 201
Lansdale, PA 19446
Telephone: 215-395-5026
Fax: 215-660-5042
E-mail: anouk.snijders@accucoms.com
Internet: www.accucoms.com

Display Advertising/Classifieds/Commercial Reprints/Supported Subscriptions
The Walchi Tauber Group, Inc.
225 Old Emmorton Road, Suite 201
Bel Air, MD 21015
Telephone: 443-512-8899; Fax: 443-512-8909
Internet: www.wt-group.com

Permissions Requests should be sent to:
Licensing, Rights, and Permissions Division
American Society of Clinical Oncology
2318 Mill Road, Suite 800
Alexandria, VA 22314
Telephone: 571-483-1722; Fax: 703-518-5094
E-mail: permissions@asco.org

Free Public Access: JCO provides free online access to original research articles older than one year at www.jco.org. Online readers can access JCO research that is more than 12 months old without a subscription; this includes articles published from January 1999 to the present. Additionally, all ASCO Special Articles, all Editorials, all Comments and Controversies papers, the Art of Oncology series, and the Correspondence section are free immediately upon publication.

Disclaimer: The ideas and opinions expressed in Journal of Clinical Oncology (JCO), do not necessarily reflect those of the American Society of Clinical Oncology (ASCO). The mention of any product, service, or therapy in this publication or in any advertisement in this publication should not be construed as an endorsement of the products mentioned. It is the responsibility of the treating physician or other health care provider, relying on independent experience and knowledge of the patient, to determine drug dosages and the best treatment for the patient. Readers are advised to check the appropriate medical literature and the product information currently provided by the manufacturer of each drug to be administered to verify approved uses, the dosage, method, and duration of administration, or contraindications. Readers are also encouraged to contact the manufacturer with questions about the features or limitations of any products. ASCO assumes no responsibility for any injury or damage to persons or property arising out of or related to any use of the material contained in this publication or to any errors or omissions.

Copyright © 2011 by American Society of Clinical Oncology unless otherwise indicated. All rights reserved. No part of this publication may be reproduced or transmitted in any form or by any means now or hereafter known, electronic or mechanical, including photocopy, recording, or any information storage and retrieval system, without permission in writing from the Publisher. Printed in the United States of America.

The appearance of the code at the bottom of the left column of the first page of an article in this journal indicates the copyright owner’s consent that copies of the article may be made for personal or internal use, or for the personal or internal use of specific clients, for those registered with the Copyright Clearance Center Inc. (222 Rosewood Drive, Danvers, MA 01923; 978-750-8400; www.copyright.com). This consent is given on the condition that the copier pay the stated per-copy fee for that article through the Copyright Clearance Center, Inc. for copying beyond that permitted by Sections 107 or 108 of the US Copyright Law. This consent does not extend to other kinds of copying, such as copying for general distribution, for advertising or promotional purposes, for creating new collective works, or for resale. Absence of the code indicates that the material may not be processed through the Copyright Clearance Center Inc.

CPT© is a trademark of the American Medical Association.
ASCO Abstracts Policy

Public Release of Abstracts
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.
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.

For clinical trials that began accrual on or after April 29, 2004, ASCO’s Policy places some restrictions on the financial relationships of principal investigators (J Clin Oncol. 2006 Jan 20;24[3]:521). If a principal investigator holds any restricted relationships, his or her abstract will be ineligible for placement in the 2011 Annual Meeting unless the ASCO Ethics Committee grants an exception. Among the circumstances that might justify an exception are that the principal investigator (1) is a widely acknowledged expert in a particular therapeutic area; (2) is the inventor of a unique technology or treatment being evaluated in the clinical trial; or (3) is involved in international clinical oncology research and has acted consistently with recognized international standards of ethics in the conduct of clinical research. NIH-sponsored trials are exempt from the Policy restrictions. Abstracts for which authors requested and have been granted an exception in accordance with ASCO’s Policy are designated with a caret symbol (^) in the Annual Meeting Proceedings.

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-beta, canonical Wnts, and noncanonical Wnts. When converging on carcinoma cells, these signals, working in concert, are able to induce expression of the...
Does breast cancer in Asian geriatric patients have the same biological characteristics as in their Western counterparts? A comparison between Shanghai and Vienna.

**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 differences of breast cancer older than 69 years in Asia and Europe. The incidence of breast cancer in Asia is especially Asian women in Western countries.

**Methods:** 198 Austrian women seen during 2005 and 2010 were reviewed. The demographics were confirmed by chart review. One-way ANOVA was used to test for differences in age, tumor stage, anatomic site and sex. Frequency distribution and median OS were calculated using the Kaplan-Meier method with the log-rank test. The influence of age, tumor stage, anatomic site and sex on survival were analyzed using Cox regression analysis.

**Results:**
- The mean age of patients was 71 years (range: 52-80) in Vienna vs. 8.3% (n=36) in Shanghai and 70 years (range: 52-80) in Vienna vs. 8.3% (n=36) in Shanghai.
- The incidence of breast cancer in Vienna is higher than in Austria (73.7% vs. 52.5%). The 10-year mortality rate is 15% higher in Austria (73.7% vs. 52.5%).
- The 5-year mortality rate is 5% higher in Austria (73.7% vs. 52.5%).
- The median OS in Vienna and Austria is 101 months vs. 106 months.
- The median OS in Vienna and Austria is 101 months vs. 106 months.

**Conclusions:**
Breast cancer in Asian geriatric patients has a different biological profile compared to their Western counterparts. The differences in biology may be due to differences in tumor characteristics, such as tumor stage, anatomic site, and sex. Further research is needed to understand these differences and to develop targeted therapies for these patients.

---

**Stomach cancer median OS* (months) (95% CI)**

<table>
<thead>
<tr>
<th>Tumor Stage</th>
<th>White</th>
<th>71</th>
<th>72</th>
</tr>
</thead>
<tbody>
<tr>
<td>Localized</td>
<td>38</td>
<td>40</td>
<td>42</td>
</tr>
<tr>
<td>Regional</td>
<td>12</td>
<td>13</td>
<td>14</td>
</tr>
<tr>
<td>Distant</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>Median age (yrs)</td>
<td>67</td>
<td>69</td>
<td>69</td>
</tr>
</tbody>
</table>

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

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. | ER/PR- | Triple negative | Basal type | p53+ | Grade 3
--- | --- | --- | --- | --- | ---
Race | OR | 95% CI | OR | 95% CI | OR | 95% CI | OR | 95% CI
AA/non-AA | 1.11 0.77-1.59 | 1.29 0.87-1.96 | 1.65 1.07-2.69 | 0.92 0.60-1.42 | 1.04 0.73-1.48 | 0.98 0.76-1.34 | 0.98 0.76-1.34 | 0.98 0.76-1.34
Age (per year) | 0.98 0.96-0.99 | 0.98 0.96-0.99 | 0.97 0.95-0.99 | 0.97 0.91-1.00 | 0.99 0.91-1.00 | 0.99 0.91-1.00 | 0.99 0.91-1.00 | 0.99 0.91-1.00
Comorbidity (+) | 0.79 0.54-1.15 | 0.71 0.48-1.07 | 0.68 0.42-1.11 | 0.97 0.62-1.56 | 0.96 0.65-1.40 | 0.82 0.55-1.42 | 0.79 0.51-1.24 | 0.73 0.43-1.24
Family history (+) | 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 | 0.86 0.62-1.49 | 0.94 0.58-1.51 | 0.83 0.43-1.61
BMI (≥ 30/ <) | 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 | 1.08 0.62-1.90 | 1.13 0.71-1.83 | 1.10 0.70-1.73
Parity (per child) | 1.00 .99-1.01 | 1.01 1.00-1.02 | 1.01 1.00-1.02 | 1.00 0.98-1.03 | 1.02 1.00-1.04 | 1.01 0.99-1.03 | 1.01 0.99-1.03 | 1.01 0.99-1.03
Race (AA/non-AA) | 0.98 0.76-1.34 | 0.98 0.76-1.34 | 0.97 0.95-0.99 | 0.97 0.91-1.00 | 0.99 0.91-1.00 | 0.99 0.91-1.00 | 0.99 0.91-1.00 | 0.99 0.91-1.00
Poverty status (≥ 1999 U.S. mean/s) | 1.56 1.06-2.57 | 0.81 0.51-1.34 | 1.13 0.83-1.58 | 1.02 0.66-1.61 | 1.16 0.76-1.77 | 1.59 0.99-2.53 | 1.86 1.10-3.17 | 1.98 1.03-3.84
Education status (≥ 1999 U.S. mean/s) | 1.79 0.97-3.32 | 0.91 0.58-1.42

1555 General Poster Session (Board #40), 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 compared to white women (adjusted odd ratio (OR) = 0.40, 95% CI: 0.19-0.83). Regardless of race, the most significant determinant of compliance was perception 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. | White | Black | P value
--- | --- | --- | ---
Age (mean; range) | 59.72 | 67.59 | 0.005
> some college education | 113 (80.4) | 95 (67.3) | 0.001
Income ≥60K | 95 (67.3) | 17 (25.3) | 0.04
Regressed therapy compliance | Never took | 4 (2.8) | 5 (7.8)
Missed > 1/week | 0 (0) | 3 (4.7)
Missed > 1/month | 1 (7.8) | 1 (7.8)
Missed ≤ 1 month | 29 (20.6) | 22 (32.8)
Missed never dose | 97 (68.5) | 32 (48.4)

1556 General Poster Session (Board #4), 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 92%. 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 and long-term experience with a shift of misuser group of patients who repeatedly misrepresent SR tobacco use. Weekly handsonhade 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.

Visit abstract.asco.org and search by abstract for the full list of abstract authors and their disclosure information.
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. M. Hepp, University Dusseldorf, Düsseldorf, 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. The SUCCESS Trial compared FEC-docetaxel (Doc) vs. FEC-Doc-gemcitabine (Doc-G) regime and 2 vs. 5 year treatment with zoledronate in 3754 patients with primary breast cancer (N+: high risk). CA27.29 has been measured with ST AIA-PACK Ca27.29 reagent using MUC-1 or AIA-6001 (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 8.95% 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.

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, histologic 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 bilateralty of many cases of breast cancer of a type, and to a possible shared etiology between cancers in the two contralateral breasts.

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 MyQ module. ΔΔct method was used to calculate the relative ASS mRNA level. Levels of ASS mRNA were correlated with that of GAPDH and normalized with that of β-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 2/9 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 μg/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 immunohistochemistry 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.

Thymidylate synthase gene copy number as predictive marker of capecitabine efficacy in patients with breast cancer. Presenting Author: R. Audeh, V M Institute of Research, Montreal, QC, Canada

Background: Expression of key enzymes of the thymidylate synthase pathway may affect the efficacy of 5-FU and the pro-drug capecitabine (C). The gene copy number of thymidylate 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 histologically confirmed breast cancer and locally advanced or metastatic disease were treated with C 1000 mg/m2 BID days 1-14 of a 28 day cycle. The SUCCESS trial. V. M Institute of Research, Montreal, QC, Canada

Methods: The gene copy number of TS, TP, DHFR and ASS was measured in 866 tumor samples using FISH. The copy number was correlated with TTP and PFS using Cox proportional hazard models in the overall population and in hormone receptor positive (ER, HER2) and HER2 negative patients. With further refinement, 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.
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 metastasize. CD44 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 high 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 high histologic grade were favorable prognostic factors for survival in patients with breast cancer.

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 76Br- and 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-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 Met abundance. In the first study, overall detection of CK+ cells was 83% with EpCAM alone and 93% with antibody cocktail. In the second study, CK+ cells were detected in 43 of 54 cases (80%). Among the 43 cases in which CK+ cells were detected, high concordance (93%) in HER2 status between primary tumor and CTCs was observed with Her2 amplification noted in both CK+ and CK- cells (presumably EMT 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 CTCs is being missed by current staging criteria. Data also demonstrate that recovery of CTCs from peripheral blood using the CEE platform is efficient and suitable for FISH-based testing.

Visit abstract.asco.org and search by abstract for the full list of abstract authors and their disclosure information.
Publication-only Abstracts

Publication-only abstracts, which are selected to be published in conjunction with the 2011 Annual Meeting, but not to be presented at the Meeting, can be found online in full-text, fully searchable versions at abstract.asco.org and JCO.org.

The publication-only abstracts are not included in the print volume, but are citable to this *Journal of Clinical Oncology* supplement. Please refer to the following example when citing publication-only abstracts:

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

Note: Numerals refer to abstract number. Accepted abstracts not presented at the meeting (designated by "e") are available in full-text versions on ASCO.org.
<table>
<thead>
<tr>
<th>Name</th>
<th>ID</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zu, Zhifei</td>
<td>5074</td>
</tr>
<tr>
<td>Zuber, Emmanuel P H</td>
<td>e21150</td>
</tr>
<tr>
<td>Zuehlke, Helmut</td>
<td>3630</td>
</tr>
<tr>
<td>Zub, Zhifei</td>
<td>5074</td>
</tr>
<tr>
<td>Zuber, Emmanuel P H</td>
<td>e21150</td>
</tr>
<tr>
<td>Zuehlke, Helmut</td>
<td>3630</td>
</tr>
<tr>
<td>Zu, Zhifei</td>
<td>5074</td>
</tr>
<tr>
<td>Zuber, Emmanuel P H</td>
<td>e21150</td>
</tr>
<tr>
<td>Zuehlke, Helmut</td>
<td>3630</td>
</tr>
<tr>
<td>Zu, Zhifei</td>
<td>5074</td>
</tr>
<tr>
<td>Zuber, Emmanuel P H</td>
<td>e21150</td>
</tr>
<tr>
<td>Zuehlke, Helmut</td>
<td>3630</td>
</tr>
<tr>
<td>Zu, Zhifei</td>
<td>5074</td>
</tr>
<tr>
<td>Zuber, Emmanuel P H</td>
<td>e21150</td>
</tr>
<tr>
<td>Zuehlke, Helmut</td>
<td>3630</td>
</tr>
</tbody>
</table>

Numerals refer to abstract number
This publication is supported in part by

Pfizer Oncology