Volume 44 Supplement 1 April 2019 ISSN 0960 9776

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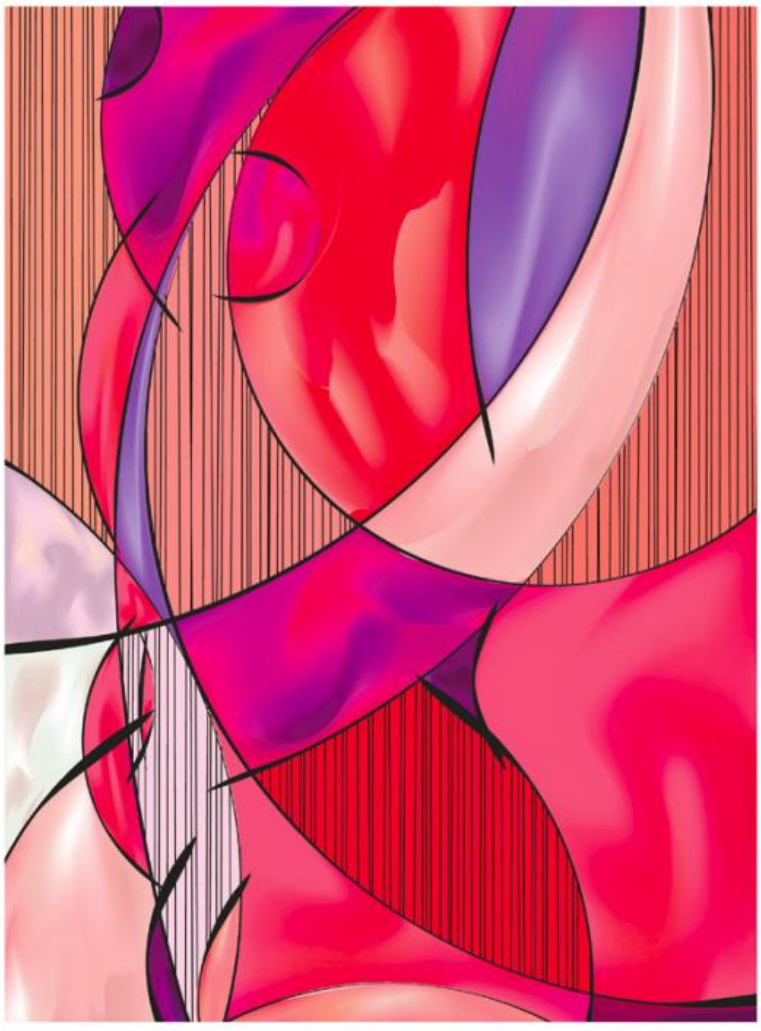
IN THIS SUPPLEMENT

PRIMARY THERAPY OF EARLY BREAST CANCER
16TH ST.GALLEN INTERNATIONAL BREAST CANCER CONFERENCE
VIENNA, AUSTRIA, 20–23 MARCH 2019

# THE BREAST

An Associate Journal of the Australasian Society for Breast Disease
Affiliated with the European Society of Breast Cancer Specialists
Official Journal of the Breast Centres Network



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# THE BREAST

## PRIMARY THERAPY
## OF EARLY BREAST CANCER
## Evidence, Controversies, Consensus

## 16th St.Gallen International Breast Cancer Conference
## Vienna/Austria, 20–23 March 2019

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## *The Breast*: Aims and Scope

*The Breast* is an international, multidisciplinary journal for researchers and clinicians, which focuses on translational and clinical research for the advancement of breast cancer prevention, diagnosis and treatment of all stages. The Editors welcome the submission of original research articles, systematic reviews, and viewpoint/commentary and debate articles, and correspondence on all areas of pre-malignant and malignant breast disease, including:

- Epidemiology and prevention
- Translational research, encompassing the use of new technologies, molecular biology, genetics and pathology
- Screening, early diagnosis, follow-up and response assessment: use of imaging, nuclear medicine and other technologies
- Medical oncology
- Radiation oncology
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- Psycho-oncology
- Quality of life
- Survivorship
- Supportive care
- Palliative and end-of-life care
- Advocacy
- Breast Nursing
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**Publication information.** *The Breast* (ISSN 0960-9776). For 2019, volumes 43–48 (6 issues) are scheduled for publication. Subscription prices are available upon request from the Publisher or from the Elsevier Customer Service Department nearest you or from this journal's website (http://www.elsevier.com/brst). Further information is available on this journal and other Elsevier products through Elsevier's website: (http://www.elsevier.com). Subscriptions are accepted on a prepaid basis only and are entered on a calendar year basis. Issues are sent by standard mail (surface within Europe, air delivery outside Europe). Priority rates are available upon request. Claims for missing issues should be made within six months of the date of dispatch.
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**USA mailing notice:** *The Breast* (ISSN 0960-9776) is published bimonthly by Elsevier Ltd. (P.O. Box 211, 1000 AE Amsterdam, The Netherlands). Periodicals postage paid at Jamaica, NY 11431 and additional mailing offices.
**USA POSTMASTER:** Send address changes to *The Breast*, Elsevier, Journal Returns, 1799 Highway 50 East, Linn, MO 65051, USA.

**AIRFREIGHT AND MAILING** in USA by Air Business Ltd., c/o Worldnet Shipping Inc., 156-15, 146th Avenue, 2nd Floor, Jamaica, NY 11434, USA.



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*The Breast* has no page charges.

♾The paper used in this publication meets the requirements of ANSI/NISO Z39.48-1992 (Permanence of Paper).

Printed in the United Kingdom by Henry Ling Limited, at the Dorset Press, Dorchester, DT1 1HD.

BCC 2021

# 17th St. Gallen International Breast Cancer Conference 2021

Primary Therapy of Early Breast Cancer
Evidence, Controversies, Consensus

17–20 March 2021, Vienna/Austria

## First Announcement 2021

### First Announcement


**Information**
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VOLUME 44 S1 APRIL 2019

# THE BREAST

## Speakers' Abstracts



## Poster Abstracts I

## Poster Abstracts II



## Author Index



**Guide for Authors**

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## Predictive and prognostic factors

### P209

### Mammographic breast density: visual and automated measurement, its role in tumor size and prognostic factors

M. Izquierdo*, J. Browne, S. Garcia, F. Tresserra, M. Garcia, S. Baulies, C. Ara, M.A. Pascual, R. Fabregas, Hospital Universitari Dexeus
*Barcelona, Spain*

**Goals:** Study the visual and automatic measurement of mammographic breast density and its implications as a prognostic factor.
**Methods:** Study the visual and automatic measurement of mammographic breast density according to the breast imaging data system (BI-RADS) in 212 patients with invasive unifocal breast cancer (not microinvasive) who did not perform neoadjuvant chemotherapy and surgery before.
Analyze the tumor size globally and with the BIRADS mammographic breast density categories, comparing the histological tumor size, versus the clinical size, ultrasound size, mammographic size and size of the magnetic resonance, a regression is made to study which test values the size better, and the correlation of DMR with prognostic factors (RE, RP, HER2, Ki67, p53).
**Results:** The comparison of Visual DMR and Automatic DMR, visual DMR 2 the DMR Automatic matches in 40.6% (41/101), in 58.4% (59/101) the DMR is 1, the visual DMR 3 matches with DMR 3 automatic in 32.1% (9/28), in the DMR 3 automatic 64.3% (18/28) is lower ($p < 0.001$). When comparing Visual DMR and Automatic DMR, visual DMR 2 the DMR Automatic matches in 40.6% (41/101), in 58.4% (59/101) the DMR is 1, the visual DMR 3 matches with DMR 3 automatic in 32.1% (9/28), in the DMR 3 automatic 64.3% (18/28) is lower ($p < 0.001$). The study of BMI with DMR, a BMI> 30 there are 0 cases DMR BIRADS 4 (visual and automatic), BMI 15–29.9 there are 0 cases DMR BIRADS 4 automated and 4 cases (14.8%) with DMR BIRADS 4 visual. DMR is not correlated ($p = ns$) with prognostic factors (ER, PR, HER2, Ki67, Histological Grade). The study of size using linear regression shows us a better estimate with less variability with ultrasound and magnetic resonance. ($\bar{x} + 1.96\ \sigma$)
**Conclusions:** *Visual measurement* overestimate MBD versus automatic measurement according BIRADS categories. Ultrasound and magnetic resonance estimate tumor size better with less variability. MBD is not related to tumor prognostic factors.
**Conflict of Interest:** No significant relationships.

### P210

### The mathematical model for predicting the *earliest diagnostics period* of the secondary distant metastases growth process of breast cancer

E. Tyuryumina*, A. Neznanov. *International Laboratory for Intelligent Systems and Structural Analysis, National Research University Higher School of Economics, Moscow, Russian Federation*

**Goals:** Previously, the mathematical models (CoMPaS and CoM-III) of primary tumor (PT) growth and secondary distant metastases (sdMTS) growth of breast cancer (BC) considering TNM classification have been presented (Tyuryumina E., Neznanov A.; 2017, 2018).
**Goal**: To detect the *earliest diagnostics period* of visible sdMTS via CoMPaS and CoM-III.
**Methods:** The models CoMPaS and CoM-III rest on exponential growth model and complementing formulas and correspond to TNM classification. The CoMPaS and CoM-III allow for calculating: (1) the tumor volume doubling time (TVDT) of the PT and the sdMTS (CoMPaS); (2) the correction coefficient of the sdMTS spreading rate in patients with lymph node MTS related with the PT growth rate (CoM-III); (3) the earliest diagnostics periods of the sdMTS. The CoMPaS model reflects the stages I-II (T1-3N0M0), the growth processes of PT and sdMTS in BC patients **without** lymph nodes MTS. The CoM-III describes the stages II-III (T1-3N1-3M0), the growth processes of PT and sdMTS in BC patients **with** lymph nodes MTS.
**Results:** The critical growth periods have been defined via the models CoMPaS and CoM-III: (1) the non-visible growth period of PT of BC; (2) the non-visible growth period of sdMTS of BC; (3) the visible growth period of sdMTS of BC. The CoMPaS and CoM-III correctly describe the growth period of PT and corresponds to TNM classification (parameter T), the growth period of the sdMTS (parameter M) and the 10–15-year survival of BC patients considering TNM classification. The CoMPaS correctly describes the growth of PT in BC patients with T1-3N0M0 stages and helps to calculate the period in which the sdMTS might appear (M1). The CoM-III correctly describes the growth of PT in BC patients with T1-3N1-3M0 stages and helps to calculate the period in which the sdMTS might appear (M1).
**Conclusions:** The models CoMPaS and CoM-III and the corresponded software tool can help: (1) to optimise the process of detecting the *earliest diagnostics periods* of sdMTS in BC patients (T1-3N0-3M0) considering TNM classification and the growth rate of PT and sdMTS of BC; (2) to improve the effectiveness of the *earliest diagnostics* and to start the earliest treatment of small sdMTS in BC patients (T1-3N0-3M0); (3) to increase the survival of BC patients with sdMTS (T1-3N0-3M0).
**Conflict of Interest:** No significant relationships.

### P211

### Integration of whole-genome sequencing and functional screening identifies a prognostic signature for lung metastasis in triple-negative breast cancer

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**Goals:** Lung metastasis is one of the leading causes of death for triple-negative breast cancer (TNBC). We sought to characterize the genetic alterations underlying TNBC lung metastases by integrating whole-genome sequencing and functional screening. Further, we aimed to develop a metastasis-related gene signature for TNBC patients to improve risk stratification.
**Methods:** In this prospective observational study, we first conducted whole-genome sequencing of paired primary tumor and lung metastasis from one TNBC patient to identify potential genetic driver alterations. An *in vivo* gain-of-function screening using an amplified open reading frame library was then employed to screen candidate genes promoting lung metastasis. Finally, we applied Cox proportional hazard regression modeling to develop a prognostic gene signature from 14 candidate genes in TNBC.
**Results:** Compared with the primary tumor, copy number amplifications of chromosomes 3q and 8q were identified in the lung metastasis. We discovered an enrichment of 14 genes from chromosomes 3q and 8q in mouse lung metastases model. We further developed and validated a four-gene signature (*ENY2*, *KCNK9*, *TNFRSF11B* and *KCNMB2*) that predicts recurrence-free survival and lung metastasis in TNBC. Our data also demonstrated that upregulated expression of *ENY2* could promote invasion and lung metastasis of TNBC cells both *in vitro* and *in vivo*.
**Conclusions:** In conclusion, our study reveals functional genes with copy number amplifications among chromosome 3q and 8q in lung metastasis of TNBC. And we develop a functional gene signature that can effectively stratify patients into low- and high-risk subgroups of recurrence, helping frame personalized treatments for TNBC.
**Conflict of Interest:** No significant relationships.