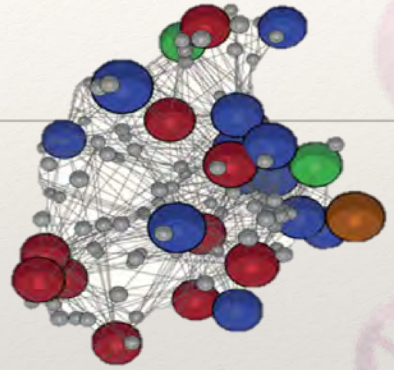
Towards the Future: Prognostic & Predictive Markers



Sunil R Lakhani

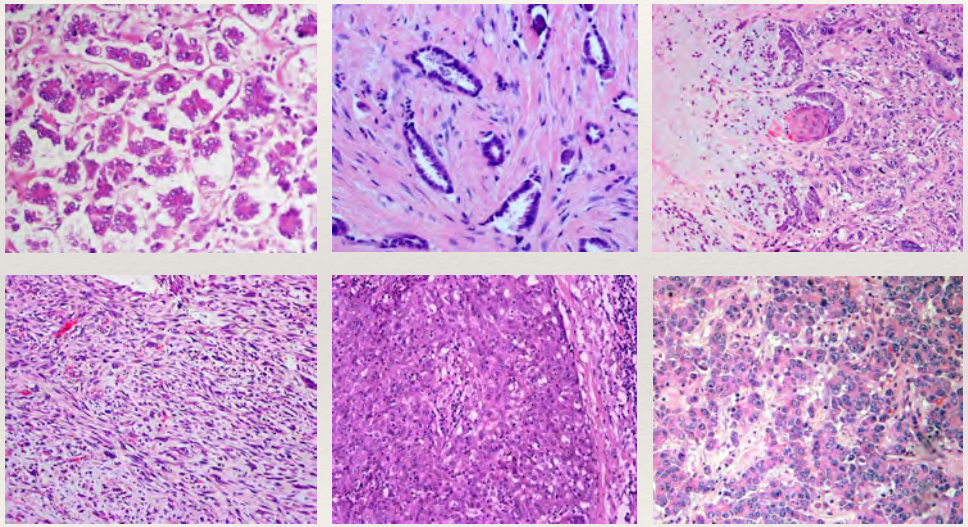
The University of Queensland & Pathology Queensland, Brisbane, Australia

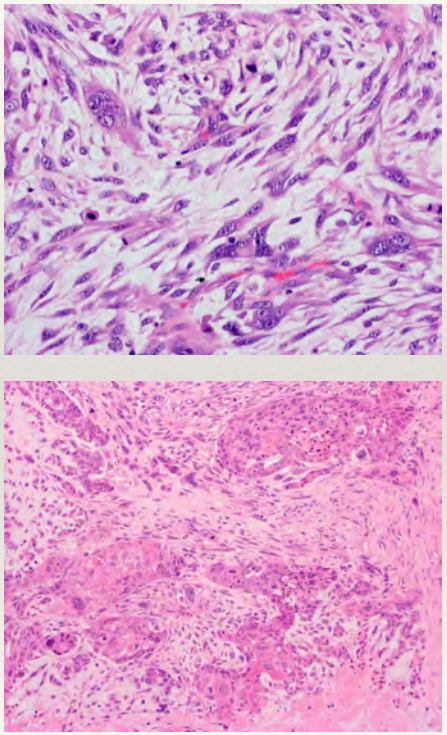
The University Of Queensland AUSTRALIA
Create change

pathology queensland

Breast Cancer – many diseases

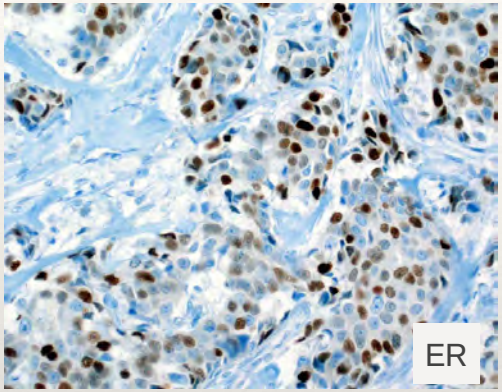
Morphology

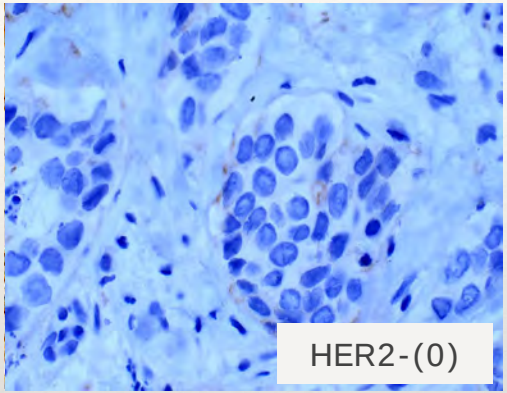


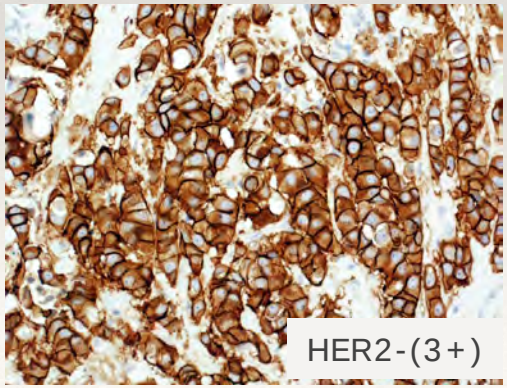


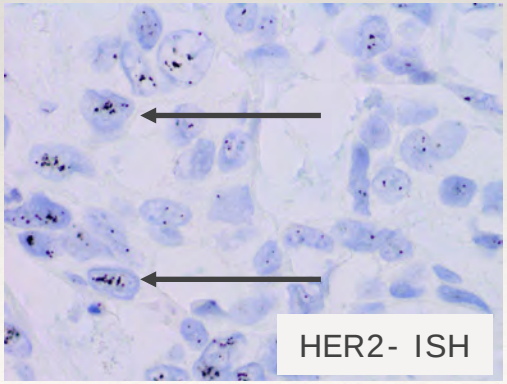
Metaplastic Ca

Current Standard - Biomarkers









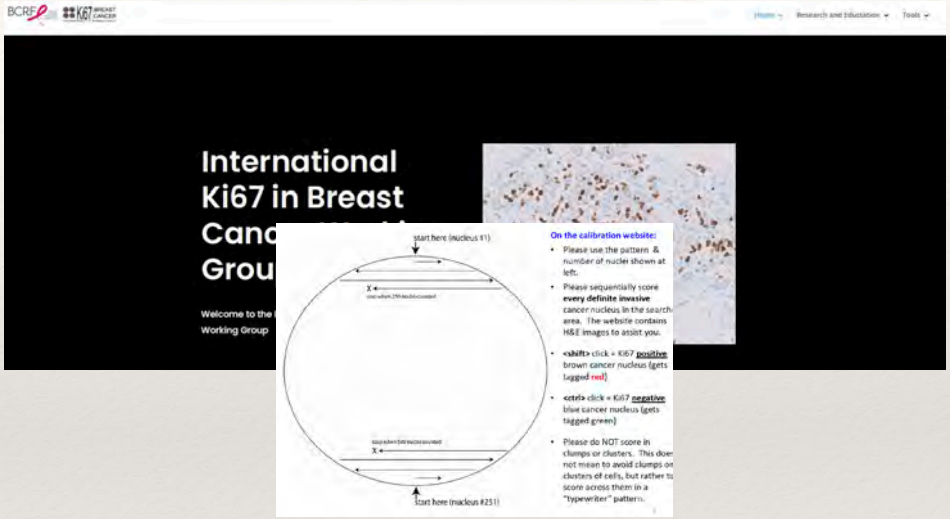
Ki-67

DOI: 10.1093/jnci/djr393
Advance Access publication on September 29, 2011.

COMMENTARY

Assessment of Ki67 in Breast Cancer: Recommendations from the International Ki67 in Breast Cancer Working Group

Mitch Dowsett, Torsten O. Nielsen, Roger A'Hern, John Bartlett, R. Charles Coombes, Jack Cuzick, Matthew Ellis, N. Lynn Henry, Judith C. Hugh, Tracy Lively, Lisa McShane, Soon Paik, Frederique Penault-Llorca, Ljudmila Prudkin, Meredith Regan, Janine Salter, Christos Sotiriou, Ian E. Smith, Giuseppe Viale, Jo Anne Zujewski, Daniel F. Hayes



- Post-Analytical
- Interpretation
- Average vs Hot spots
- How many nuclei
- Eyeball vs Image analysis

Breast Cancer Research and Treatment
https://doi.org/10.1007/s10549-023-07197-3

Online 29.12.23

ORIGINAL LABORATORY INVESTIGATION

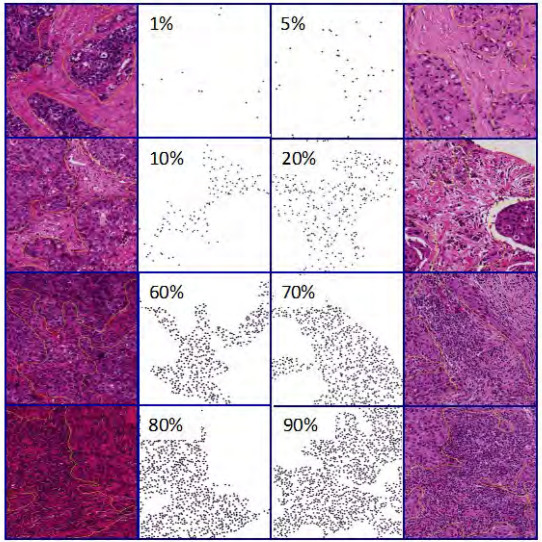
Subspecialized breast pathologists have suboptimal interobserver agreement in Ki-67 evaluation using 20% as the cutoff

Di Ai[1] · Gulisa Turashvili[1] · Sandra Gjorgova Gjeorgjievski[1] · Qun Wang[1] · Abdulwahab M. Ewaz[1] · Yuan Gao[1] · Thi Nguyen[1] · Chao Zhang[2] · Xiaoxian Li[1]

Tumour Infiltrating Lymphocytes (TILs)

TILs in Invasive Breast Carcinoma:
Reference Scoring Sheet

Reproduced from Salgado et al 2015 with permission from Oxford University Press on behalf of the European Society for Medical Oncology.



npj | Breast Cancer

www.nature.com/npjbcancer

REVIEW ARTICLE OPEN

Check for updates

The tale of TILs in breast cancer: A report from The International Immuno-Oncology Biomarker Working Group

npj Breast Cancer (2021)7:150 ; https://doi.org/10.1038/s41523-021-00346-1

Virchows Archiv (2022) 480:147–162
https://doi.org/10.1007/s00428-022-03276-w

REVIEW

Incorporation of TILs in daily breast cancer care: how much evidence can we bear?

Anne-Vibeke Laenkholm[1] · Grace Callagy[2] · Marcelo Balancin[3] · John M. S. Bartlett[4] · Christos Sotiriou[5] · Caterina Marchio[6,7] · Marleen Kok[8] · Carlos Henrique Dos Anjos[9] · Roberto Salgado[10,11]

biomedicines

MDPI

Article

The Value of Tumor Infiltrating Lymphocytes (TIL) for Predicting the Response to Neoadjuvant Chemotherapy (NAC) in Breast Cancer According to the Molecular Subtypes

Ionut Flaviu Faur [1,2], Amadeus Dobrescu [1,2,*], Adelina Ioana Clim [3], Paul Pasca [1], Catalin Prodan-Barbulescu [1,2], Bogdan Daniel Gherle [4], Cristi Tarta [1,2], Alexandru Isaic [1,2], Dan Brebu [1,2], Ciprian Duta [1,2], Bogdan Totolici [5,6] and Gabriel Lazar [7,8]

Biomedicines **2023**, *11*, 3037. https://doi.org/10.3390/biomedicines11113037

Immunotherapy in TNBC – PD-L1

- Atezoluzimab (anti-PD-L1) approved in advanced TNBC 2019
- Based on IMpassion130 (using SP142 assay)
- PD-L1+ IMMUNE CELLS in ≥1% of TUMOUR AREA
- Problem for Pathology

  Criteria and Abs different for different tumours and drugs

Am J Surg Pathol • Volume 45, Number 8, August 2021

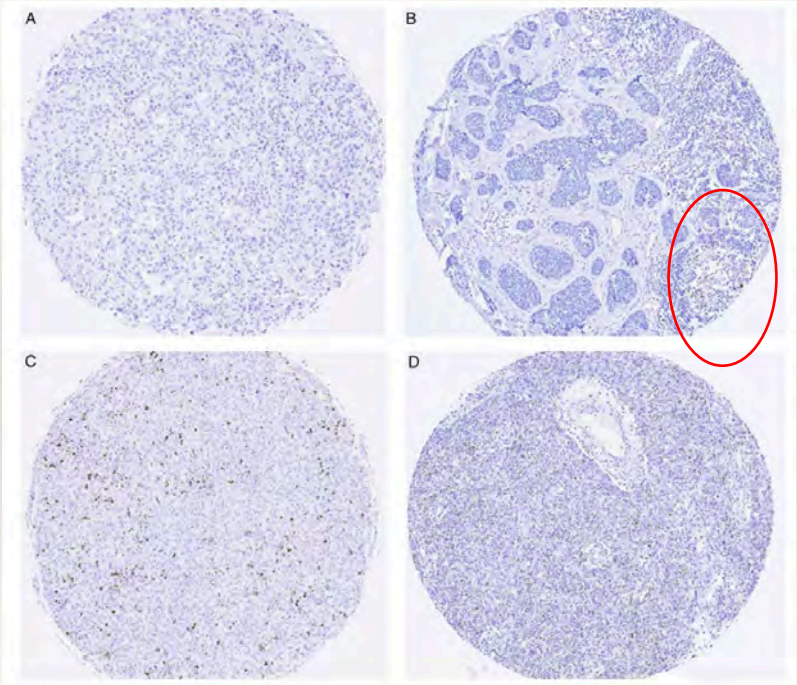


FIGURE 1. SP142 PD-L1 staining of inflammatory cells in tumor-associated stroma. A, <1%. B, 1% to <5%. C, 5% to <10%. D, ≥10%.

HER2 Low Breast Cancers: T-DXd


The NEW ENGLAND JOURNAL of MEDICINE

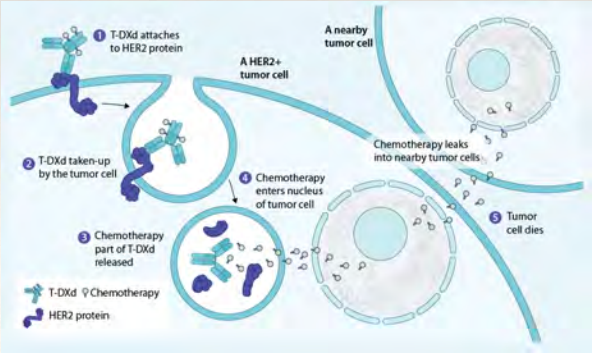
ESTABLISHED IN 1812 JULY 7, 2022 VOL. 387 NO. 1

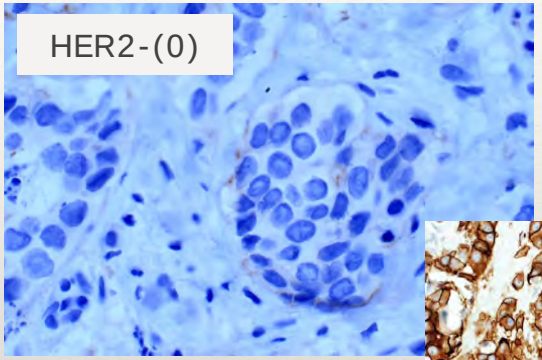
Trastuzumab Deruxtecan in Previously Treated HER2-Low Advanced Breast Cancer

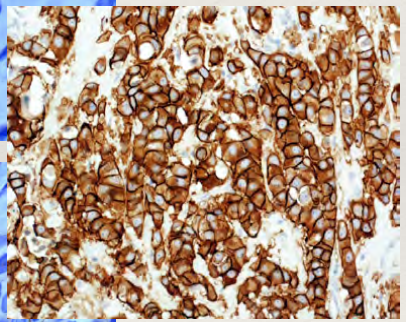
S. Modi, W. Jacot, T. Yamashita, J. Sohn, M. Vidal, E. Tokunaga, J. Tsurutani, N.T. Ueno, A. Prat, Y.S. Chae, K.S. Lee, N. Niikura, Y.H. Park, B. Xu, X. Wang, M. Gil-Gil, W. Li, J.-Y. Pierga, S.-A. Im, H.C.F. Moore, H.S. Rugo, R. Yerushalmi, F. Zagouri, A. Gombos, S.-B. Kim, Q. Liu, T. Luo, C. Saura, P. Schmid, T. Sun, D. Gambhire, I. Yung, Y. Wang, J. Singh, P. Vitazka, G. Meinhardt, N. Harbeck, and D.A. Cameron, for the DESTINY-Breast04 Trial Investigators*

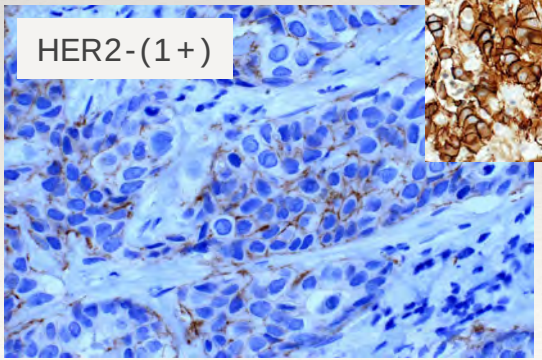
HER2-low mBC is defined by IHC scores of 1+ or 2+/ISH–

T-DXd is the first HER2-*targeted* therapy to demonstrate statistically significant and clinically meaningful improvement in PFS and OS versus TPC

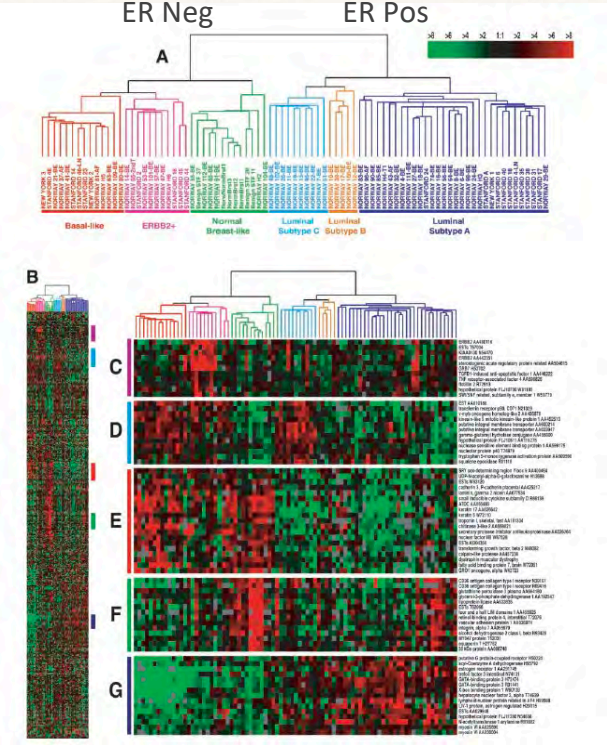


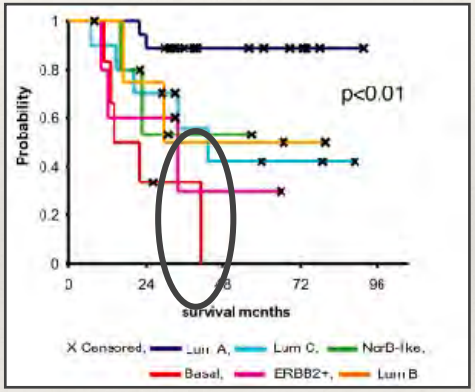






Genomic Revolution

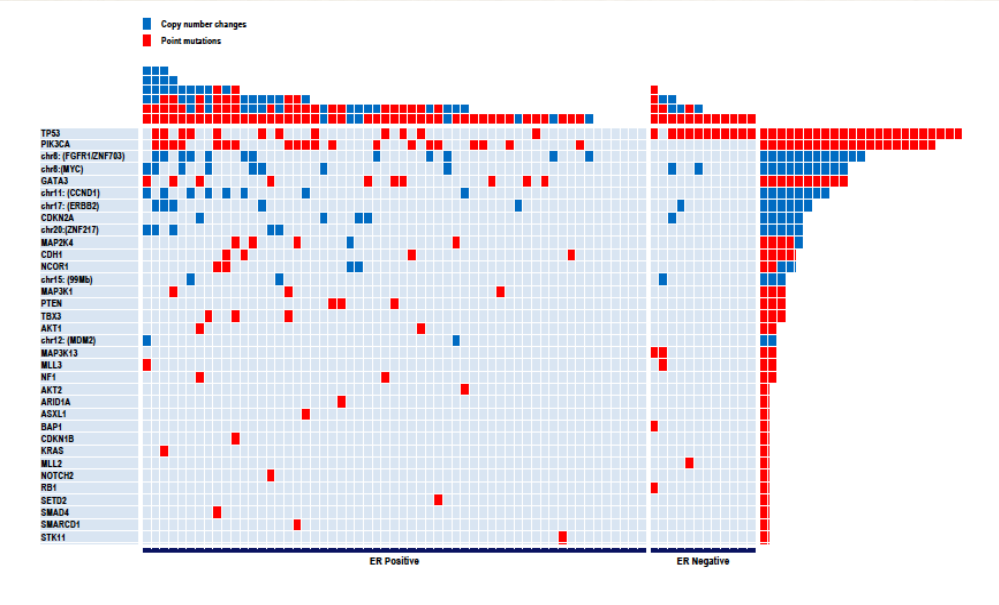




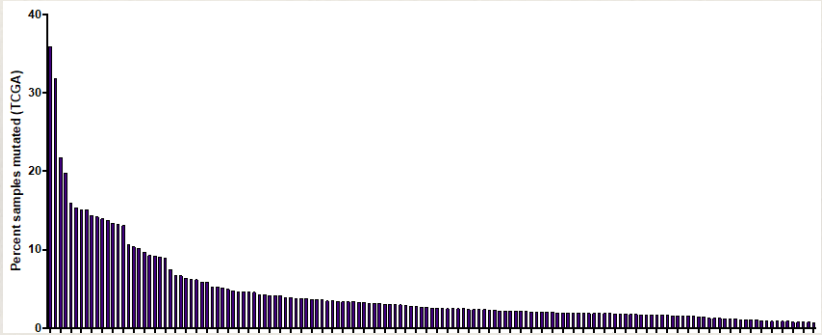
PNAS | September 11, 2001 | vol. 98 | no. 19 | 10869–10874

Inter-patient genomic heterogeneity

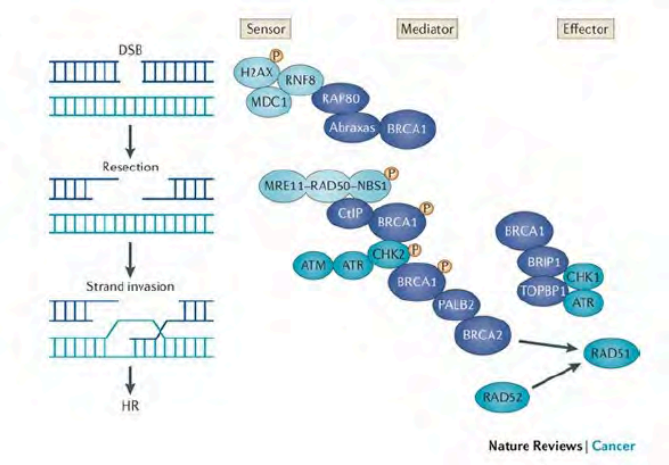
ICGC – International Cancer Genome Consortium

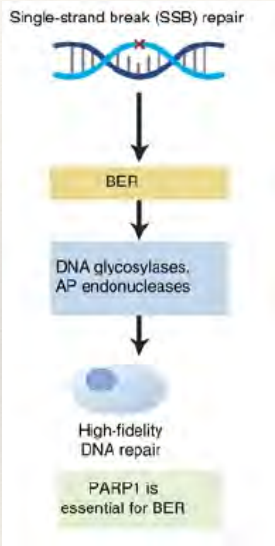


Proportion of breast cancer samples with driver gene mutations (n=147) - TCGA

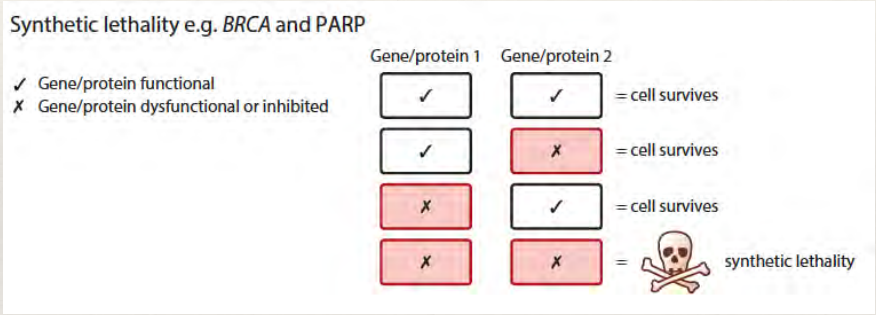


BRCA 1&2: DNA Repair & Synthetic Lethality





British Journal of Cancer (*Br J Cancer*)



NATURE REVIEWS | CANCER VOLUME 16 | FEBRUARY 2016

OlympiAD and OlympiA

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Olaparib for Metastatic Breast Cancer in Patients with a Germline

Mark Robson, M.D., Seock-Ah Im, M.D., Ph.D., Eli
Binghe Xu, M.D., Ph.D., Susan M. Domchek, M.D., N
Suzette Delaloge, M.D., Wei Li, M.D., Na
Anne Armstrong, M.D., Ph.D., Wenting Wu, Ph.D
Sarah Runswick, Ph.D., and Pierfranc

ABSTRACT

BACKGROUND

Olaparib is an oral poly(adenosine diphosphate–ribo
has promising antitumor activity in patients with m
germline *BRCA* mutation.

METHODS

We conducted a randomized, open-label, phase 3 tr
therapy was compared with standard therapy in pa
mutation and human epidermal growth factor rece
metastatic breast cancer who had received no mor
therapy regimens for metastatic disease. Patients wer
ratio, to receive olaparib tablets (300 mg twice dai
single-agent chemotherapy of the physician's choic
vinorelbine in 21-day cycles). The primary end point
which was assessed by blinded independent central
an intention-to-treat basis.

RESULTS

Of the 302 patients who underwent randomization,
olaparib and 97 were assigned to receive standard t
free survival was significantly longer in the olaparib
therapy group (7.0 months vs. 4.2 months; hazard ra
death, 0.58; 95% confidence interval, 0.43 to 0.80; P<
59.9% in the olaparib group and 28.8% in the standa
grade 3 or higher adverse events was 36.6% in the
the standard-therapy group, and the rate of treatmen
effects was 4.9% and 7.7%, respectively.

CONCLUSIONS

Among patients with HER2-negative metastatic breas
mutation, olaparib monotherapy provided a signif
therapy; median progression-free survival was 2.8 m
disease progression or death was 42% lower with ola
standard therapy. (Funded by AstraZeneca; OlympiA
NCT02000622.)

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Talazoparib in Patients with Advanced Breast Cancer and a Germline *BRCA* Mutation

Jennifer K. Litton, M.D., Hope S. Rugo, M.D., Johannes Ettl, M.D.,
Sara A. Hurvitz, M.D., Anthony Gonçalves, M.D., Ph.D., Kyung-Hun Lee, M.D., Ph.D.,
Louis Fehrenbacher, M.D., Rinat Yerushalmi, M.D., Lida A. Mina, M.D.,
Miguel Martin, M.D., Ph.D., Henri Roché, M.D., Ph.D., Young-Hyuck Im, M.D., Ph.D.,
Ruben G.W. Quek, Ph.D., Denka Markova, Ph.D., Iulia C. Tudor, Ph.D.,
Alison L. Hannah, M.D., Wolfgang Eiermann, M.D., and Joanne L. Blum, M.D., Ph.D.

ABSTRACT

BACKGROUND

The poly(adenosine diphosphate–ribose) inhibitor talazoparib has shown antitumor activity in patients with advanced breast cancer and germline mutations in *BRCA1* and *BRCA2* (*BRCA1/2*).

METHODS

We conducted a randomized, open-label, phase 3 trial in which patients with advanced breast cancer and a germline *BRCA1/2* mutation were assigned, in a 2:1 ratio, to receive talazoparib (1 mg once daily) or standard single-agent therapy of the physician's choice (capecitabine, eribulin, gemcitabine, or vinorelbine in continuous 21-day cycles). The primary end point was progression-free survival, which was assessed by blinded independent central review.

RESULTS

Of the 431 patients who underwent randomization, 287 were assigned to receive talazoparib and 144 were assigned to receive standard therapy. Median progression-free survival was significantly longer in the talazoparib group than in the standard-therapy group (8.6 months vs. 5.6 months; hazard ratio for disease progression or death, 0.54; 95% confidence interval [CI], 0.41 to 0.71; P<0.001). The interim median hazard ratio for death was 0.76 (95% CI, 0.55 to 1.06; P=0.11 [57% of projected events]). The objective response rate was higher in the talazoparib group than in the standard-therapy group (62.6% vs. 27.2%; odds ratio, 5.0; 95% CI, 2.9 to 8.8; P<0.001). Hematologic grade 3–4 adverse events (primarily anemia) occurred in 55% of the patients who received talazoparib and in 38% of the patients who received standard therapy; nonhematologic grade 3 adverse events occurred in 32% and 38% of the patients, respectively. Patient-reported outcomes favored talazoparib; significant overall improvements and significant delays in the time to clinically meaningful deterioration according to both the global health status–quality-of-life and breast symptoms scales were observed.

CONCLUSIONS

Among patients with advanced breast cancer and a germline *BRCA1/2* mutation, single-agent talazoparib provided a significant benefit over standard chemotherapy with respect to progression-free survival. Patient-reported outcomes were superior with talazoparib. (Funded by Medivation [Pfizer]; EMERACA ClinicalTrials.gov number, NCT01945775.)

From the University of Texas M.D. Anderson Cancer Center, Houston (J.K.L.), and the Texas Oncology–Baylor Charles A. Sammons Cancer Center, US Oncology Network, Dallas (J.L.B.) — both in Texas; University of California, San Francisco, Helen Diller Family Comprehensive Cancer Center (H.S.R.), and Pfizer (R.G.W.Q., D.M., I.C.T., A.L.H.), San Francisco, University of California, Los Angeles, Los Angeles (S.A.H.), and Kaiser Permanente, Northern California, Vallejo (L.F.) — all in California; the Department of Obstetrics and Gynecology, Klinikum Rechts der Isar, Technische Universität München (J.E.), and Interdisziplinäres Onkologisches Zentrum München (W.E.) — both in Munich, Germany; Institut Paoli–Calmettes, Marseille (A.G.), and Institut Claudius Regaud, Institut Universitaire du Cancer Toulouse, Toulouse (H.R.) — both in France; Seoul National University Hospital (K.-H.L.) and Samsung Medical Center (Y.-H.I.) — both in Seoul, South Korea; Rabin Medical Center, Beilinson Hospital, Petah Tikva, Israel (R.Y.); Banner M.D. Anderson Cancer Center, Gilbert, AZ (L.A.M.); and Instituto de Investigación Sanitaria Gregorio Marañón, Centro de Investigación Biomédica en Red Oncología, Grupo Español de Investigación en Cáncer de Mama, Universidad Complutense, Madrid (M.M.). Address reprint requests to Dr. Litton at Breast Medical Oncology, University of Texas M.D. Anderson Cancer Center, 1515 Holcombe Blvd., Unit 1354, Houston, TX 77030, or at jlitton@mdanderson.org.

This article was published on August 13, 2018, and updated on August 17, 2020, at NEJM.org.

N Engl J Med 2018;379:753-63.
DOI: 10.1056/NEJMoa1802905

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Adjuvant Olaparib for Patients with *BRCA1*- or *BRCA2*-Mutated Breast Cancer

A.N.J. Tutt, J.E. Garber, B. Kaufman, G. Viale, D. F
R.D. Gelber, E. de Azambuja, A. Fielding, J. Balm
K.A. Gelmon, S.J. Hollingsworth, L.A. Korde, R. Lir
E. Senkus, J.M. Suga, Z. Shao, A.W. Pippas, Z. Nowec
P.C. Lucas, N. Baker, S. Loibl, R. McConnell, M. P
G.G. Steger, J.P. Costantino, A. Arahmani, N. Wo
V. Karantza, S.R. Lakhani, G. Yothers, C. Campbel
for the OlympiA Clinical Trial Steering Committe

ABSTRACT

BACKGROUND

Poly(adenosine diphosphate–ribose) polymerase inhibit
fects in homologous recombination repair by synthetic l
needed to reduce recurrence in patients with *BRCA1* or
associated early breast cancer.

METHODS

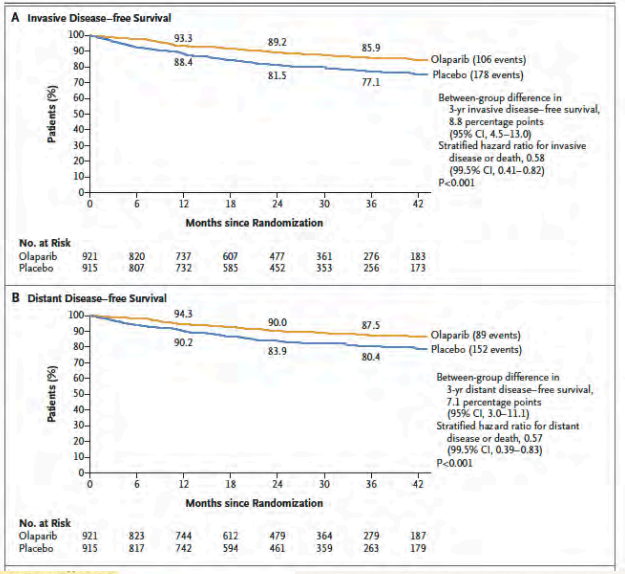
We conducted a phase 3, double-blind, randomized tr
human epidermal growth factor receptor 2 (HER2)–n
with *BRCA1* or *BRCA2* germline pathogenic or likely pat
risk clinicopathological factors who had received local
or adjuvant chemotherapy. Patients were randomly assig
of oral olaparib or placebo. The primary end point was in

RESULTS

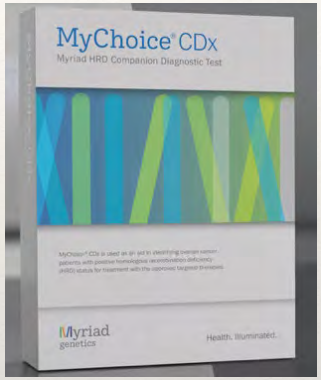
A total of 1836 patients underwent randomization. At
interim analysis with a median follow-up of 2.5 years, t
free survival was 85.9% in the olaparib group and 77
(difference, 8.8 percentage points; 95% confidence inte
ard ratio for invasive disease or death, 0.58; 99.5% CI,
3-year distant disease–free survival was 87.5% in the ol
the placebo group (difference, 7.1 percentage points; 9
ratio for distant disease or death, 0.57; 99.5% CI, 0.39
was associated with fewer deaths than placebo (59 an
ratio, 0.68; 99% CI, 0.44 to 1.05; P=0.02); however, the
was not significant at an interim-analysis boundary of
Safety data were consistent with known side effects of ol
ous adverse events or adverse events of special interest.

CONCLUSIONS

Among patients with high-risk, HER2-negative early b
BRCA1 or *BRCA2* pathogenic or likely pathogenic variar
completion of local treatment and neoadjuvant or adju
sociated with significantly longer survival free of invasive or distant disease than
was placebo. Olaparib had limited effects on global patient-reported quality of life.
(Funded by the National Cancer Institute and AstraZeneca; OlympiA ClinicalTrials
.gov number, NCT02032823.)



Genomic Signatures and Tools for HRD



nature medicine

HRDetect is a predictor of *BRCA1* and *BRCA2* deficiency based on mutational signatures

Helen Davies[1,32], Dominik Glodzik[1,32], Sandro Morganella[1], Lucy R Yates[1,2], Johan Staaf[3], Xueqing Zou[1], Manasa Ramakrishna[1,4], Sancha Martin[1], Sandrine Boyault[5], Anieta M Sieuwerts[6], Peter T Simpson[7], Tari A King[8], Keiran Raine[1], Jorunn E Eyfjord[9], Gu Kong[10], Åke Borg[3], Ewan Birney[11], Hendrik G Stunnenberg[12], Marc J van de Vijver[13], Anne-Lise Børresen-Dale[14,15], John W M Martens[6], Paul N Span[16,17], Sunil R Lakhani[7,18], Anne Vincent-Salomon[19,20], Christos Sotiriou[21], Andrew Tutt[22,23], Alastair M Thompson[24], Steven Van Laere[25,26], Andrea L Richardson[27,28], Alain Viari[29,30], Peter J Campbell[1], Michael R Stratton[1] & Serena Nik-Zainal[1,31]

Approximately 1–5% of breast cancers are attributed to inherited mutations in *BRCA1* or *BRCA2* and are selectively sensitive to poly(ADP-ribose) polymerase (PARP) inhibitors. In other cancer types, germline and/or somatic mutations in *BRCA1* and/or *BRCA2* (*BRCA1/BRCA2*) also confer selective sensitivity to PARP inhibitors. Thus, assays to detect *BRCA1/BRCA2*-deficient tumors have been sought. Recently, somatic substitution, insertion/deletion and rearrangement patterns, or 'mutational signatures', were associated with *BRCA1/BRCA2* dysfunction. Herein we used a lasso logistic regression model to identify six distinguishing mutational signatures predictive of *BRCA1/BRCA2* deficiency. A weighted model called HRDetect was developed to accurately detect *BRCA1/BRCA2*-deficient samples. HRDetect identifies *BRCA1/BRCA2*-deficient tumors with 98.7% sensitivity (area under the curve (AUC) = 0.98). Application of this model in a cohort of 560 individuals with breast cancer, of whom 22 were known to carry a germline *BRCA1* or *BRCA2* mutation, allowed us to identify an additional 22 tumors with somatic loss of *BRCA1* or *BRCA2* and 47 tumors with functional *BRCA1/BRCA2* deficiency where no mutation was detected. We validated HRDetect on independent cohorts of breast, ovarian and pancreatic cancers and demonstrated its efficacy in alternative sequencing strategies. Integrating all of the classes of mutational signatures thus reveals a larger proportion of individuals with breast cancer harboring *BRCA1/BRCA2* deficiency (up to 22%) than hitherto appreciated (~1–5%) who could have selective therapeutic sensitivity to PARP inhibition.

A small fraction of breast cancers (~1–5%)[1–3] are attributed to familial mutations in the *BRCA1* and *BRCA2* cancer susceptibility genes. Heterozygous germline mutations in *BRCA1* and *BRCA2* confer elevated lifetime risks of breast, ovarian and other cancers[4,5]. BRCA1 and BRCA2 proteins have multiple, distinct roles in maintaining genome integrity, particularly through homologous recombination (HR)-mediated double-strand break (DSB) repair[6]. These classical tumor-suppressor genes usually lose the wild-type allele during tumorigenesis to become fully inactivated[7]. *BRCA1*- and *BRCA2*-null tumors are thus deficient in HR and are selectively sensitive to compounds that increase the demand on HR[8]. PARP inhibitors are an example of therapeutic compounds that cause replication fork stalling and collapse, leading to increased DSBs[9]. The inability to perform HR-dependent DSB repair ultimately leads to selective tumor cell death[10,11].

Preclinical studies and phase I and II breast and ovarian cancer clinical trials[12,13] have shown PARP inhibitor efficacy in familial *BRCA1*- and *BRCA2*-mutant patients. However, PARP inhibition has applications beyond the treatment of germline-mutated tumors[14]. Effective PARP inhibition maintenance therapy has been demonstrated in high-grade serous ovarian cancer with germline or somatic *BRCA1* or *BRCA2* mutations[15]. Thus, extensive efforts have been put into identifying the molecular features of tumors that are *BRCA1* or *BRCA2* deficient—a defect historically referred to as 'BRCAness'—whether the genes are inactivated through germline, somatic or secondary means, including promoter DNA hypermethylation or inactivation of a related gene in the HR pathway.

Gene-specific sequencing strategies, including sequencing all known HR genes, multiplex-ligation-dependent probe amplification (MLPA)[16], promoter hypermethylation assays[17], identification of transcriptional metagene signatures[18–20], copy-number-based methods (for example, to determine the homologous recombination deficiency (HRD) index and genomic 'scars')[21–23] and functional assays of HR competence[24], have been developed to detect *BRCA1/BRCA2* deficiency. However, the indices from these methods have had limited predictive success. A recent review suggests that a good predictor of the biological status of an HR-deficient tumor is essential, as the cohort of tumors that demonstrate BRCAness and could be selectively sensitive to PARP inhibitors is likely not limited to the small proportion of familial breast and ovarian cancers with *BRCA1* or *BRCA2* mutations but extends to a larger fraction of sporadic breast and ovarian cancers, as well as other cancer types[25].

Recent advances in sequencing technology[26] have greatly reduced sequencing costs, permitting whole-genome sequencing (WGS) for

A full list of affiliations appears at the end of the paper.

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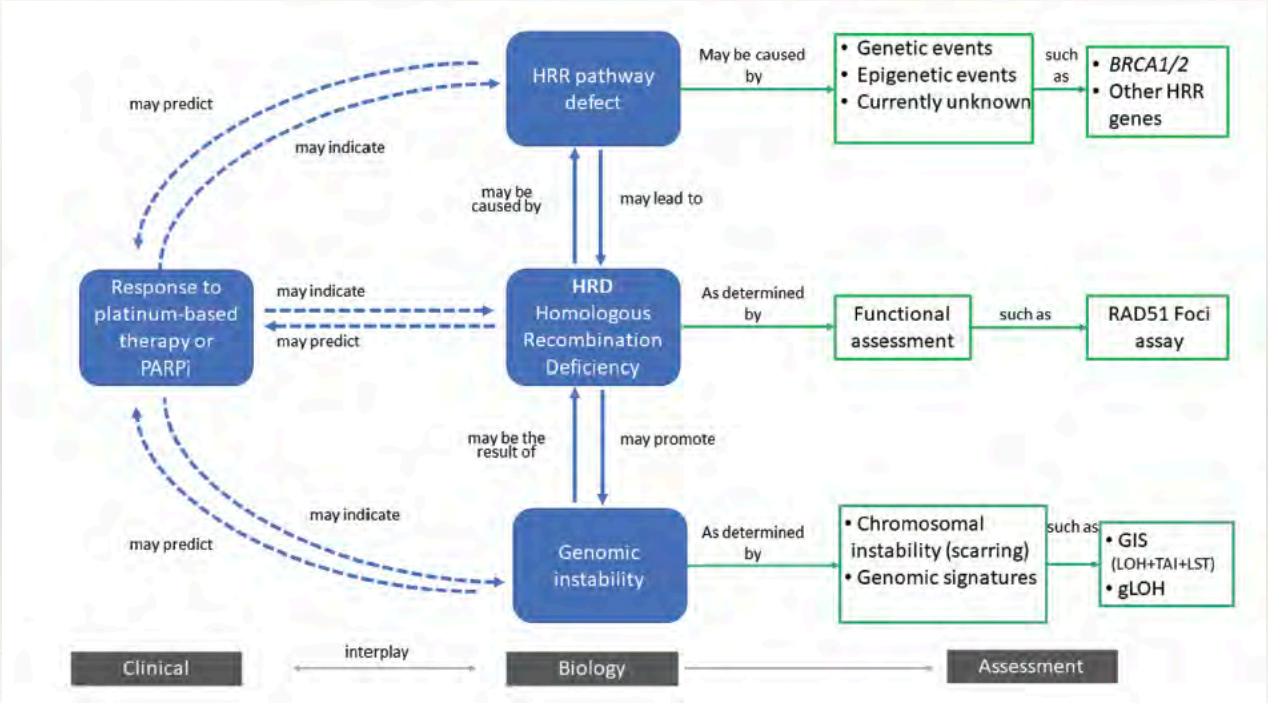
Homologous Recombination Deficiency



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Q-IMPROvE

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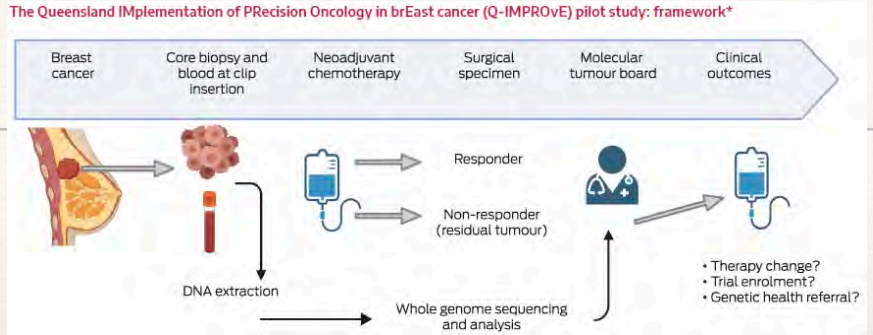
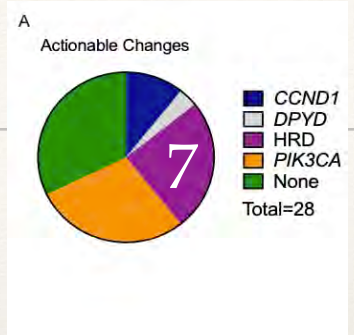
doi: 10.5694/mja2.51975





ER-, PR-, HER2-

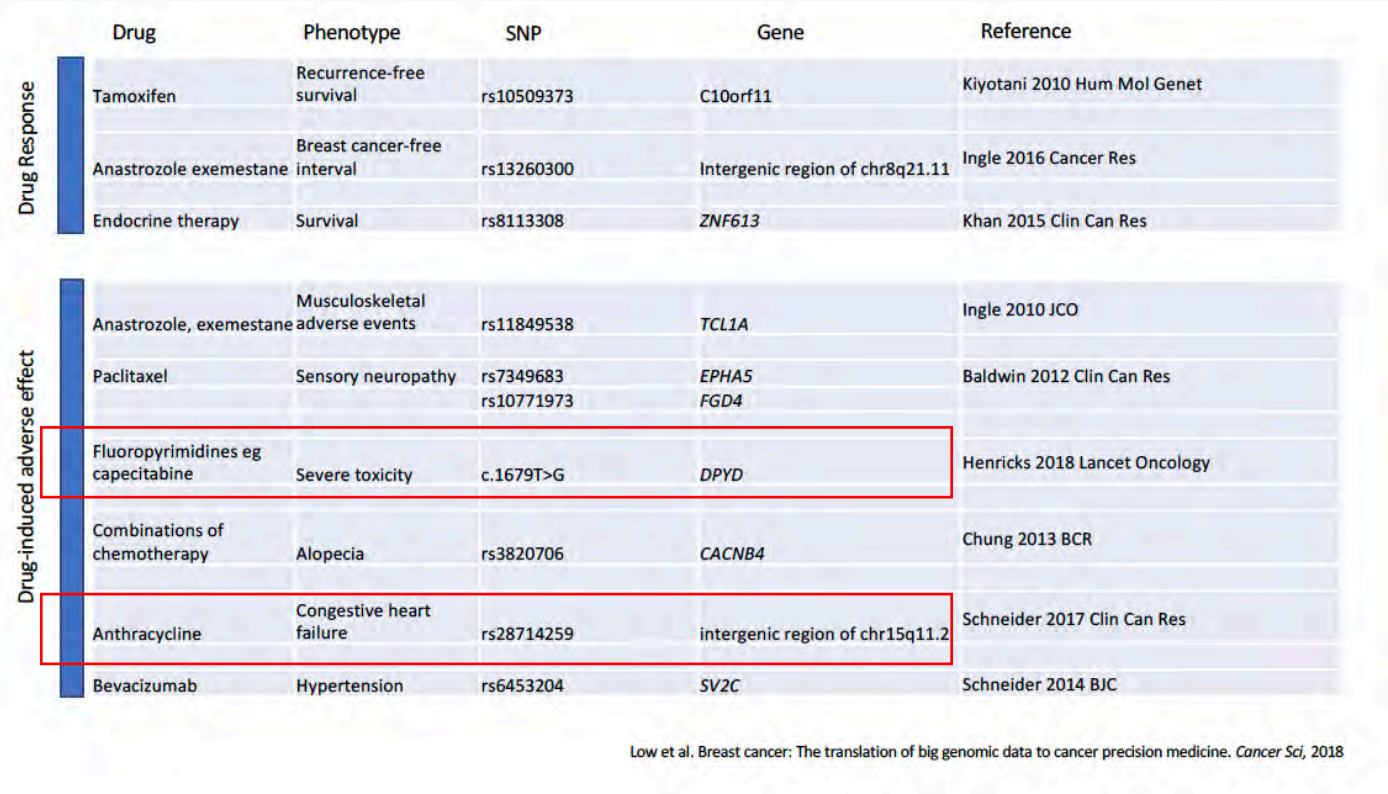
HR Detect score (>0.7)	0
HRD score (>=42)	43

ER+, PR+, HER2+

HR Detect score (>0.7)	0.03
HRD score (>=42)	54

Neither showed canonical HRD signature Sig3/Sig 8

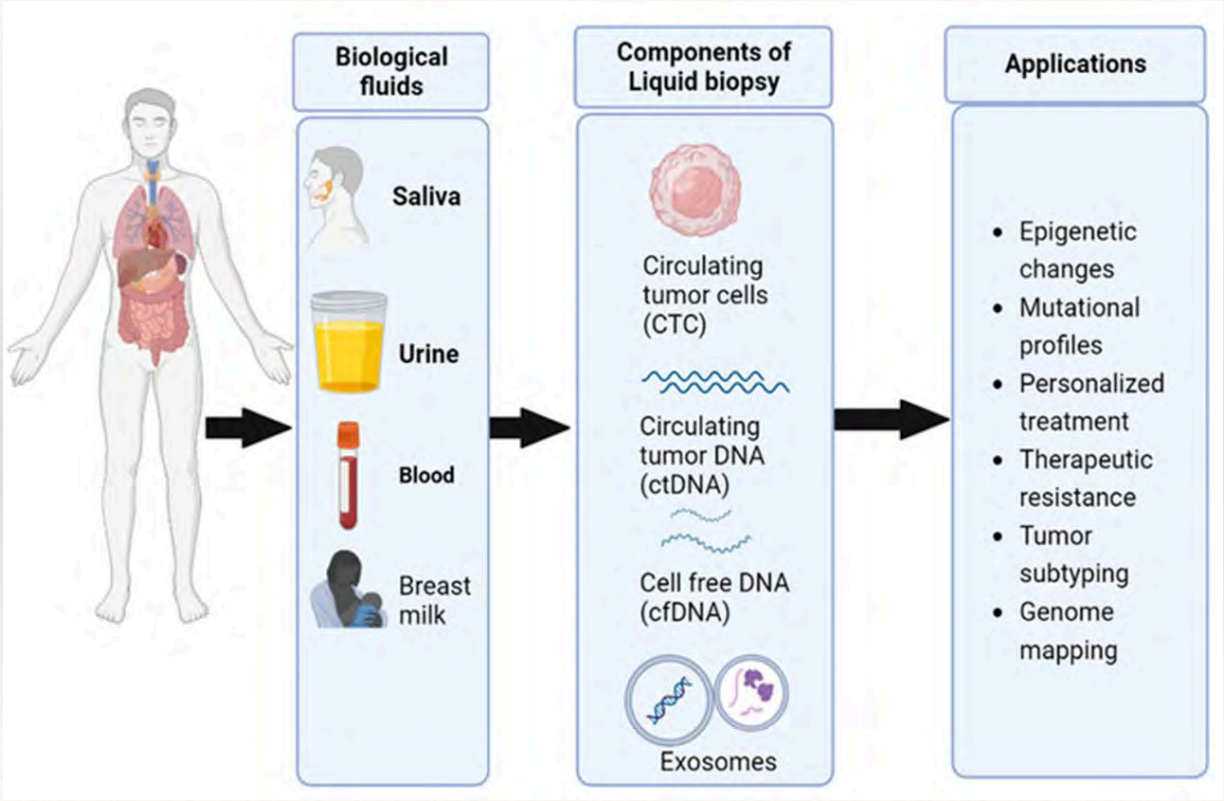
Pharmacogenomics



	Drug	Phenotype	SNP	Gene	Reference
Drug Response	Tamoxifen	Recurrence-free survival	rs10509373	C10orf11	Kiyotani 2010 Hum Mol Genet
	Anastrozole exemestane	Breast cancer-free interval	rs13260300	Intergenic region of chr8q21.11	Ingle 2016 Cancer Res
	Endocrine therapy	Survival	rs8113308	*ZNF613*	Khan 2015 Clin Can Res
Drug-induced adverse effect	Anastrozole, exemestane	Musculoskeletal adverse events	rs11849538	*TCL1A*	Ingle 2010 JCO
	Paclitaxel	Sensory neuropathy	rs7349683 rs10771973	*EPHA5* *FGD4*	Baldwin 2012 Clin Can Res
	Fluoropyrimidines eg capecitabine	Severe toxicity	c.1679T>G	*DPYD*	Henricks 2018 Lancet Oncology
	Combinations of chemotherapy	Alopecia	rs3820706	*CACNB4*	Chung 2013 BCR
	Anthracycline	Congestive heart failure	rs28714259	intergenic region of chr15q11.2	Schneider 2017 Clin Can Res
	Bevacizumab	Hypertension	rs6453204	*SV2C*	Schneider 2014 BJC

Low et al. Breast cancer: The translation of big genomic data to cancer precision medicine. *Cancer Sci*, 2018

‘Liquid biopsies’



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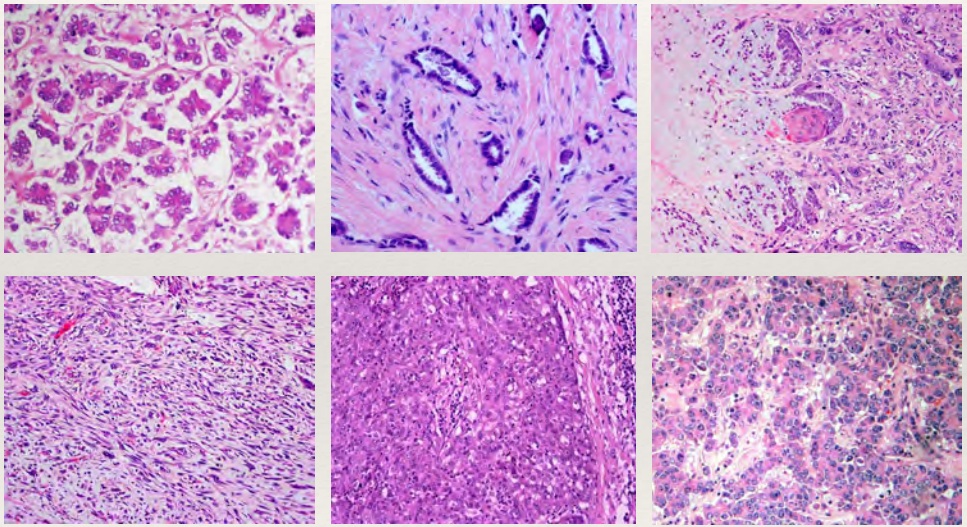
Digital Pathology

Morphology

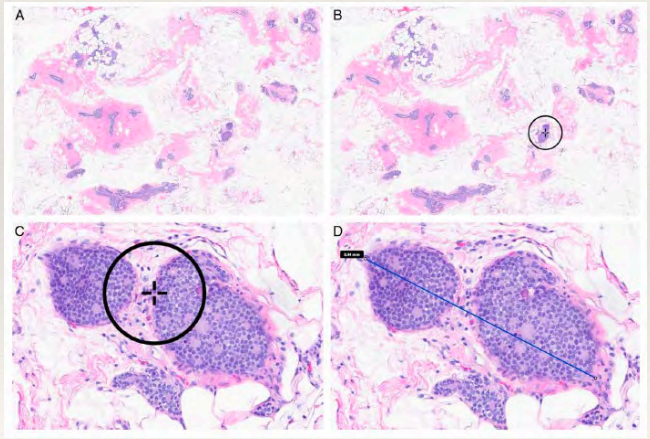


Digital Pathology – Machine Learning

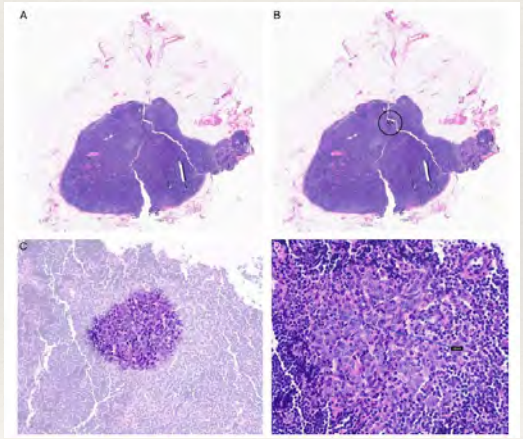
Future Practices of Breast Pathology Using Digital and Computational Pathology

Matthew G. Hanna, MD and Edi Brogi, MD, PhD

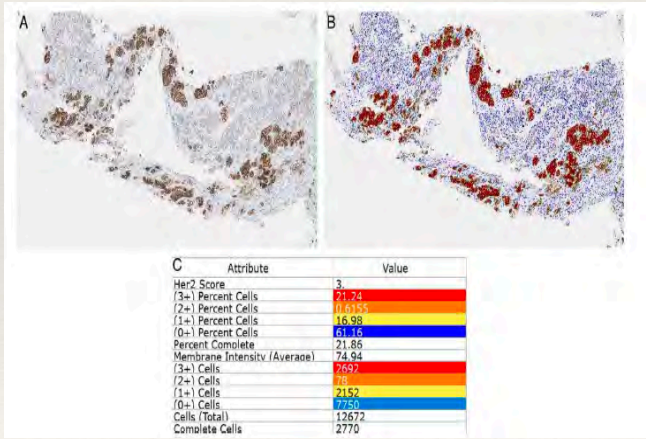
Adv Anat Pathol • Volume 30, Number 6, November 2023



ADH



LN – Micro met



HER2 - heterogenous

Machine Learning & AI

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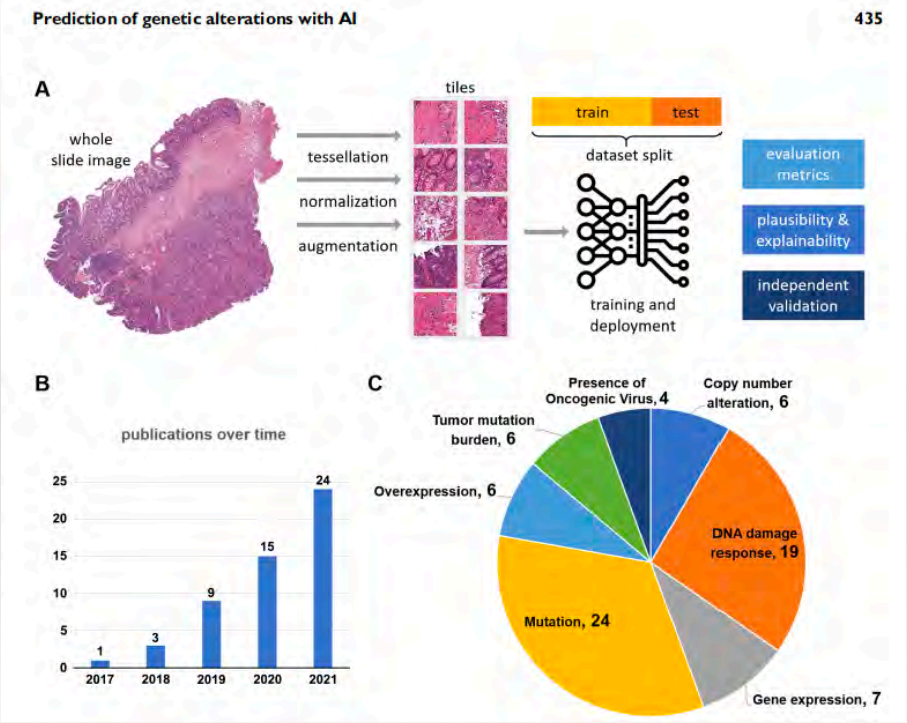
INVITED REVIEW

Artificial intelligence to identify genetic alterations in conventional histopathology

Didem Cifci[1], Sebastian Foersch[2] and Jakob Nikolas Kather[1,3,4*]



Data Integration

Pathology

Imaging

Genomic

Clinical

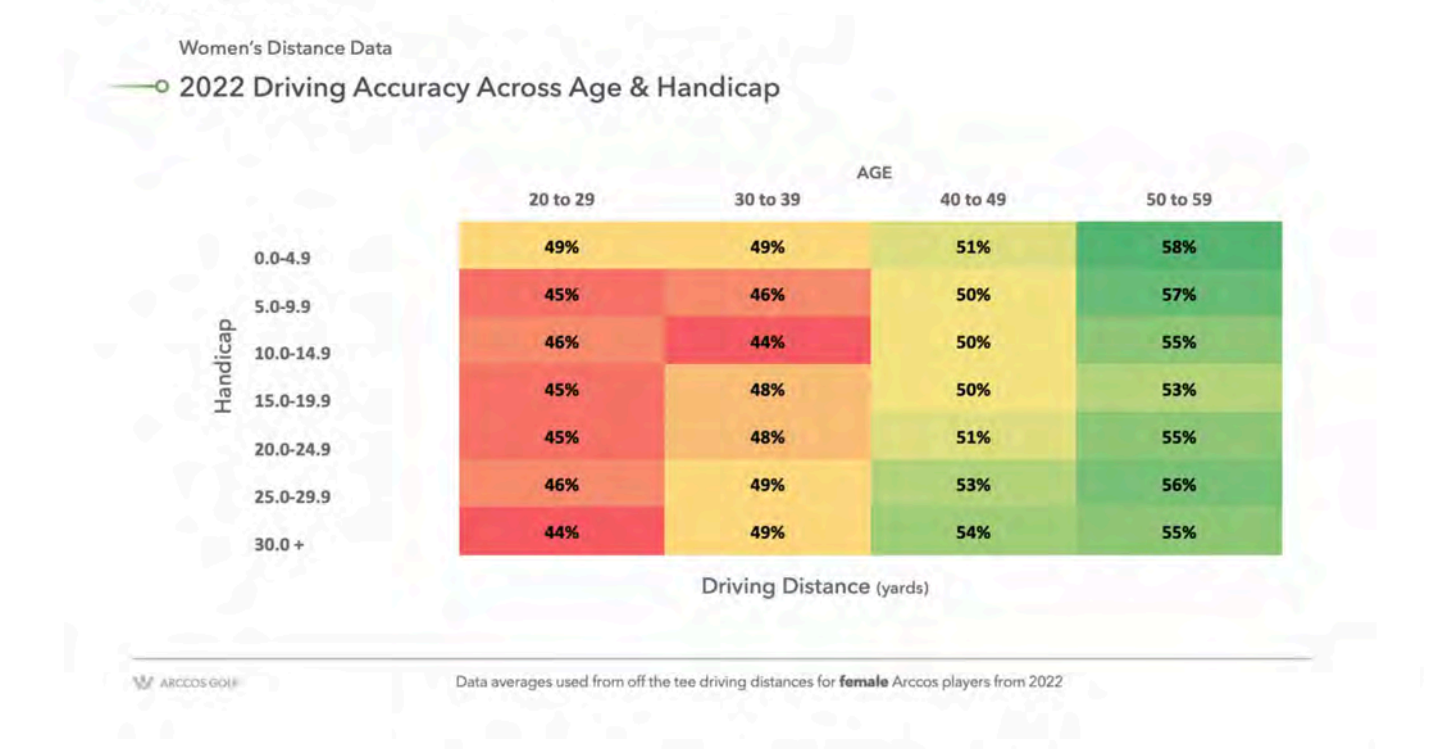
Lifestyle

Familial

Outcome

Integrate datasets from millions of people

Data Integration



Summary

- Morphology and Immunohistochemistry are mainstay of delivering prognostic/predictive markers
- Ki67, TILs, PDL-1 and others are rapidly being incorporate
- Genomic methods (WES, WGS, proteomics, metabolomics) will add new levels of information to manage patients
- Ability to utilize liquid biopsies is providing ‘non-invasive’ tools
- Digital pathology – machine learning – artificial intelligence are entering workflows and will further enhance and impact precision medicine

Thank you