How Long Should We Treat Patients with Metastatic Breast Cancer? Is Cure Possible?

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Medical Director, UNIMED Medical Institute, Hong Kong

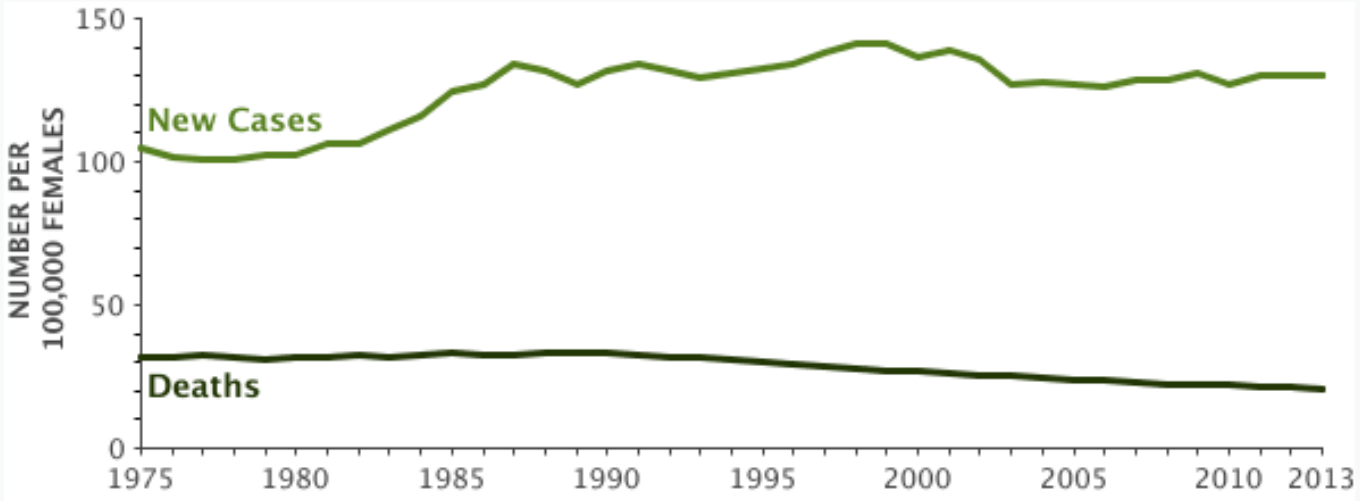
How Long Should We Treat Patients With Metastatic Breast Cancer?

NATIONAL CANCER INSTITUTE

Surveillance, Epidemiology, and End Results Program

New Cases, Deaths and 5-Year Relative Survival

View Data Table



Year	1975	1980	1985	1990	1995	2000	2004	2008
5-Year Relative Survival	75.2%	74.9%	78.4%	84.6%	86.8%	90.2%	89.9%	90.6%

SEER 9 Incidence & U.S. Mortality 1975-2013, All Races, Females. Rates are Age-Adjusted.

Death rates have been falling on average 1.9% each year

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How Many People Survive 5 Years Or More after Being Diagnosed with Female Breast Cancer?

Relative survival statistics compare the survival of patients diagnosed with cancer with the survival of people in the general population who are the same age, race, and sex and who have not been diagnosed with cancer. Because survival statistics are based on large groups of people, they cannot be used to predict exactly what will happen to an individual patient. No two patients are entirely alike, and treatment and responses to treatment can vary greatly.

Percent Surviving 5 Years

89.7%

Based on data from SEER 18 2006–2012. Gray figures represent those who have died from female breast cancer. Green figures represent those who have survived 5 years or more.

But, how about MBC?

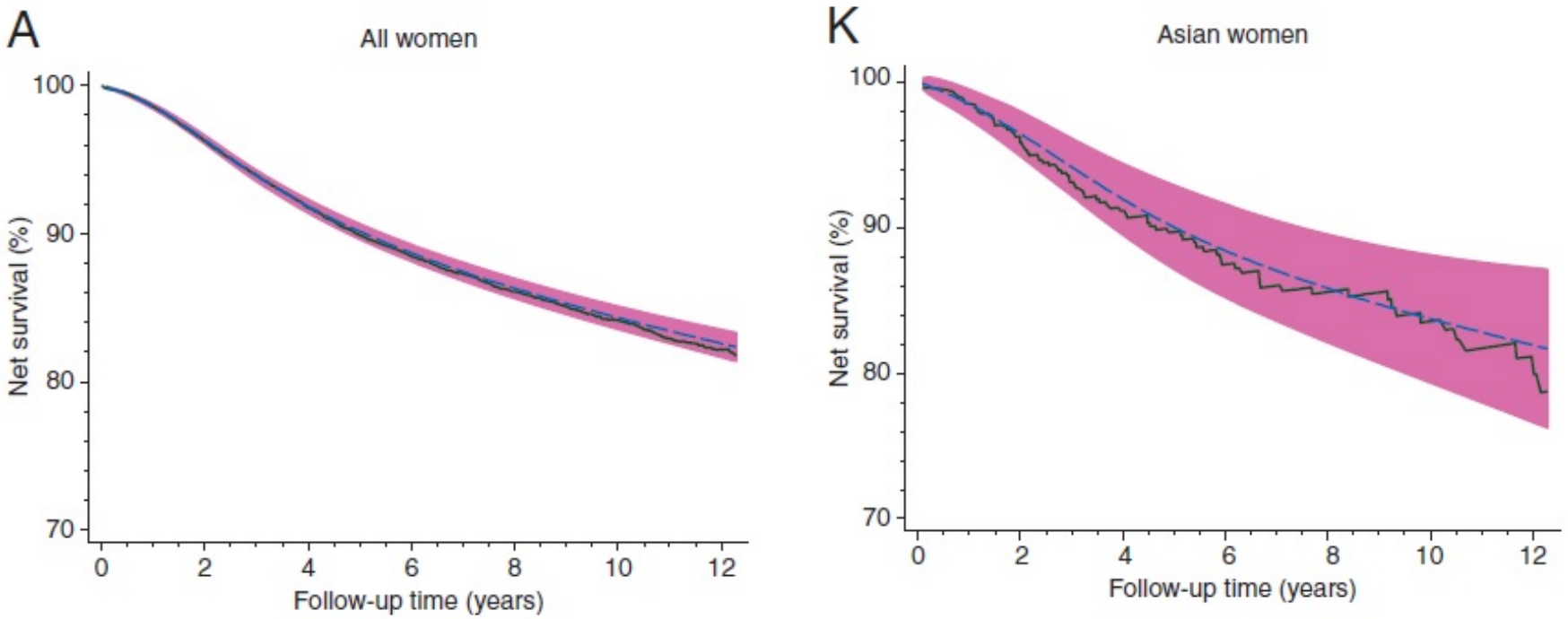
No ‘cure’ within 12 years of diagnosis among breast cancer patients who are diagnosed via mammographic screening: women diagnosed in the West Midlands region of England 1989–2011

L. M. Woods, M. Morris* & B. Rachet

Cancer Research UK Cancer Survival Group, Faculty of Epidemiology and Population Health, Department of Non-Communicable Disease Epidemiology, London School of Hygiene and Tropical Medicine, London, UK

- Cancer registry data on 19 800 women aged 50–70, diagnosed with a **primary, invasive, non-metastatic breast cancer** between 1 April 1989 and 31 March 2011.

- Results: There was an overwhelming lack of evidence for ‘cure’.



Even for non-MBC, there was a continuous decrease in net survival over time, with no obvious asymptotic tendency within 12 years of follow-up. Model-based analyses confirmed this observation.

Woods LM, et al. Ann Oncol. 2016; 27(11): 2025–2031.

The goals of treating MBC are to control tumor growth and prolong life while also maintaining quality of life
(The Susan G. Komen Breast Cancer Foundation)

Hence, a balance between the
treatment regimen and quality of life is needed

Disease control

=

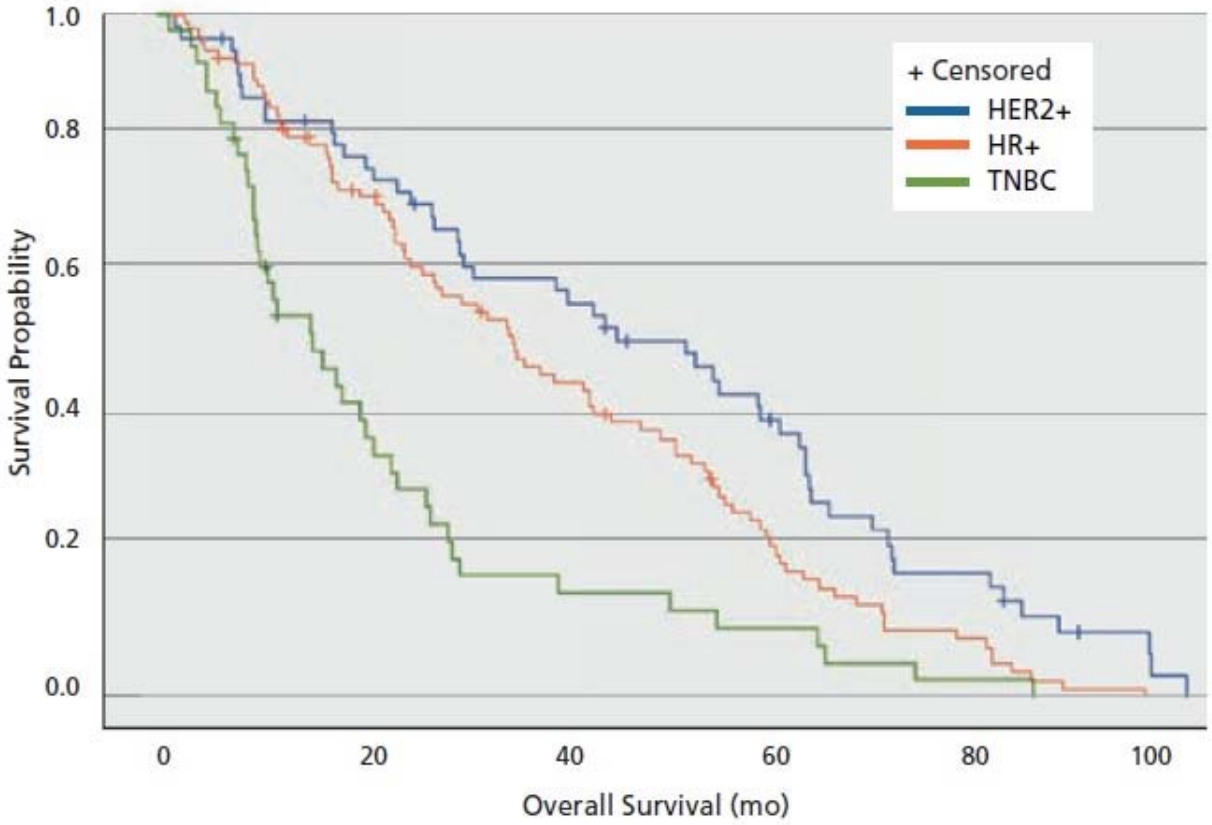
Make cancer a chronic disease

Strategies

- Administering potent MTD treatments for a fixed period?
- Administering biological drugs together with cytotoxic regimens, continuously until progression occurs?
- Administering better tolerated regimens for prolonged time – metronomic strategy?

Tumor subtypes influence survival

Data from MBC patients at Dana-Farber Cancer Institute



Overall survival time was longest in patients with HER2-amplified disease, followed by HR+ and then TNBC disease (54 vs 36 vs 17 months, respectively; P<.0001)

Seah DS, et al. J Natl Compr Canc Netw. 2014;12(1):71-80.

Calculating survival times for patients with MBC

Data from 15 trials with 4,798 patients

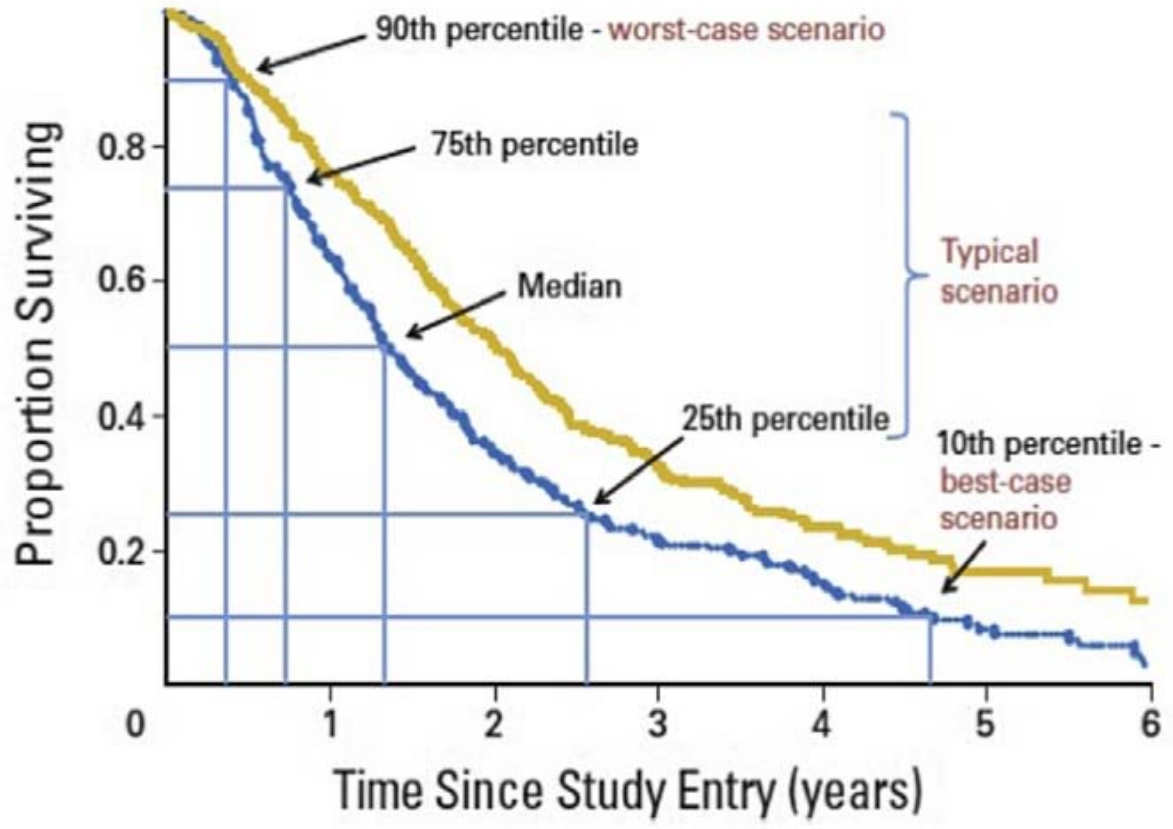
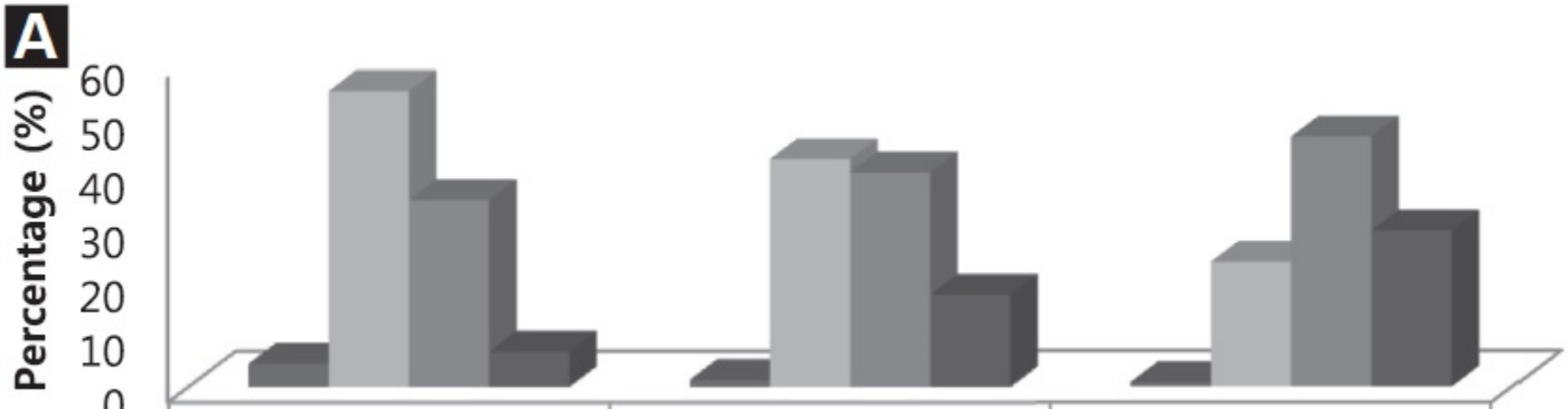


Fig. 1. Kaplan–Meier OS curve percentiles and their corresponding scenarios.

Vasista A, et al. Breast. 2017;31:99-104.

Effects of subsequent CT on HR+ HER2- MBC patients
Data from National Cancer Center Korea

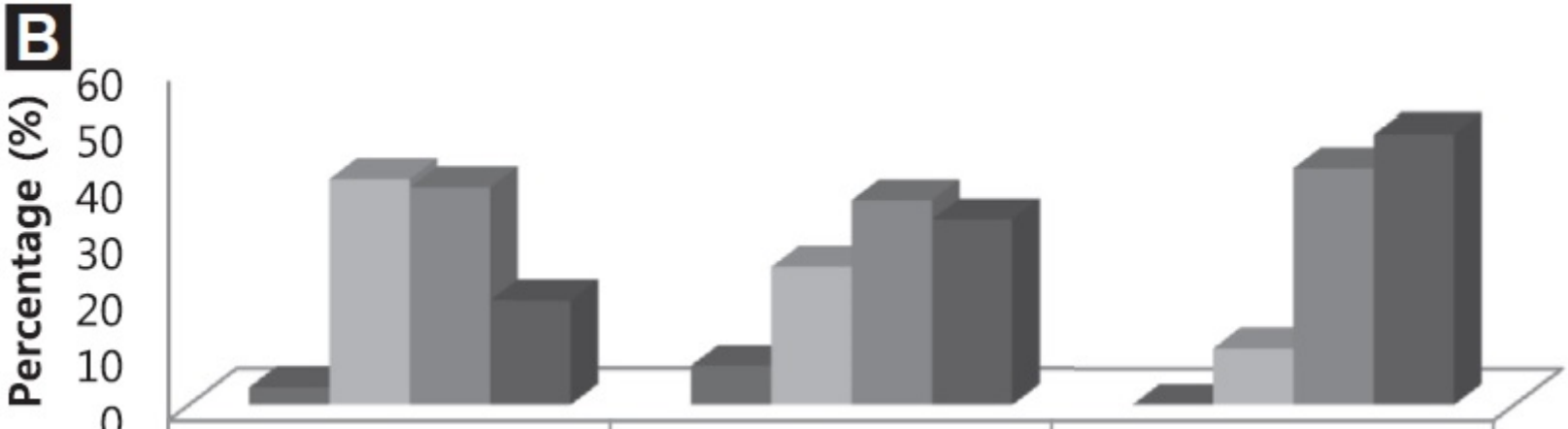


	First line (N=177)	Second line (N=151)	Third line (N=116)
CR	4.3	1.3	0.8
PR	54.7	42.1	23.1
SD	34.5	39.6	46.3
PD	6.5	17	28.9

CR = complete response; PR = partial response; SD = stable disease; PD = progressive disease

Park IH, et al. Clin Breast Cancer. 2015;15(1):e55-62.

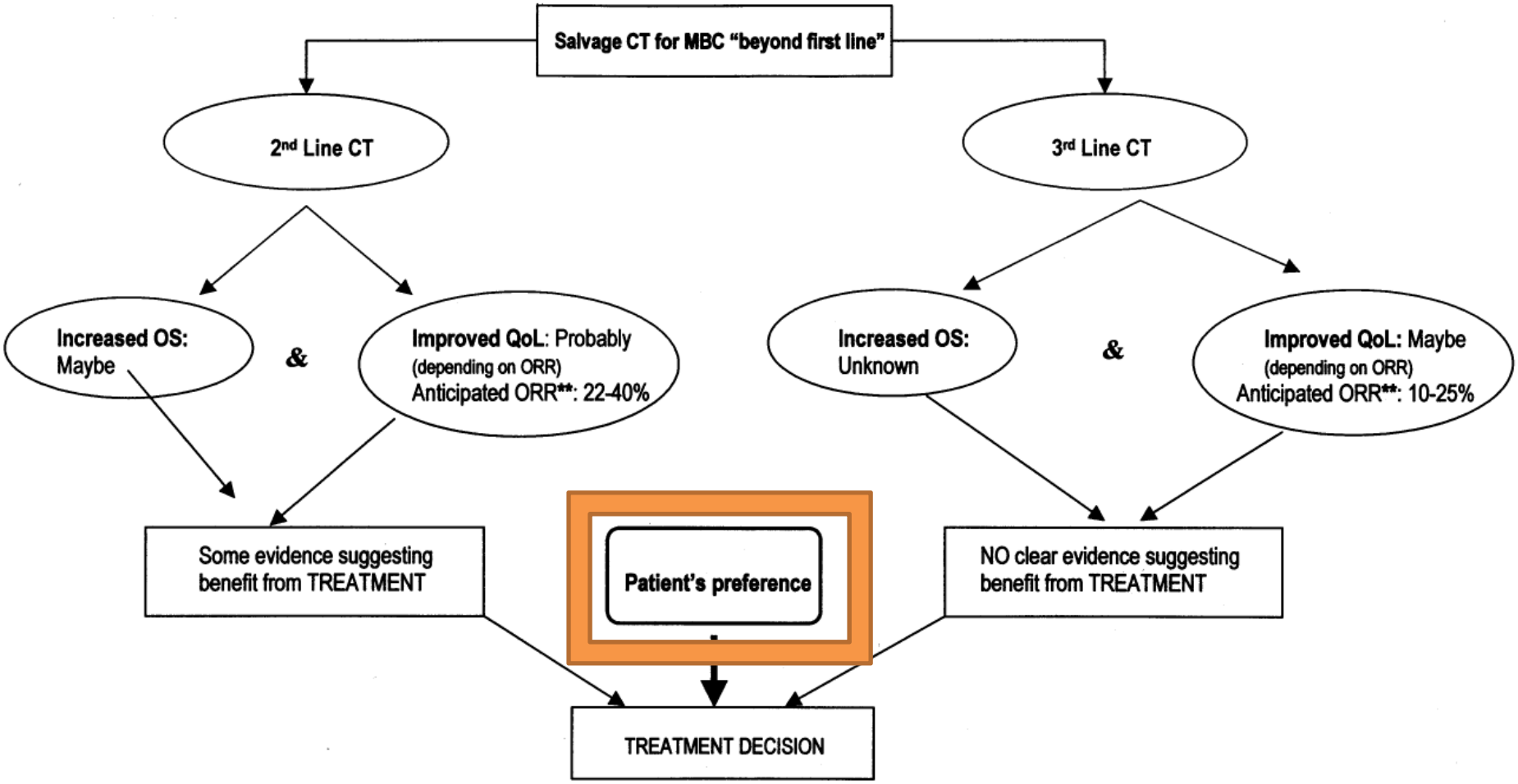
Effects of subsequent CT on HR- HER2- MBC patients
Data from National Cancer Center Korea



	First line (N=63)	Second line (N=58)	Third line (N=50)
CR	3.1	6.9	0
PR	40	24.1	10
SD	38.5	36.2	42
PD	18.5	32.8	48

CR = complete response; PR = partial response; SD = stable disease; PD = progressive disease

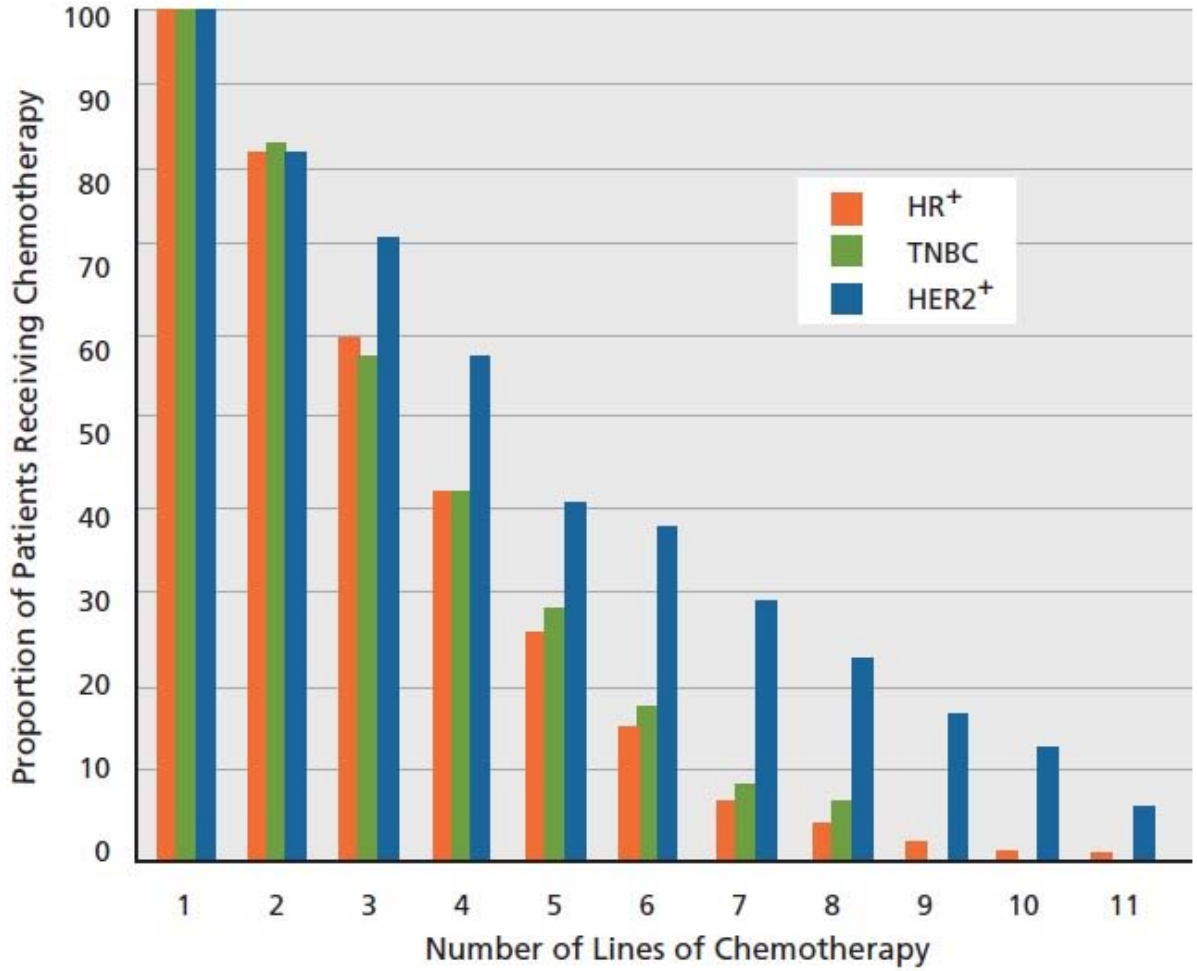
Park IH, et al. Clin Breast Cancer. 2015;15(1):e55-62.



Cardoso F, et al. Ann Oncol. 2002;13(2):197-207.

Number of lines of chemotherapy by line and subtype

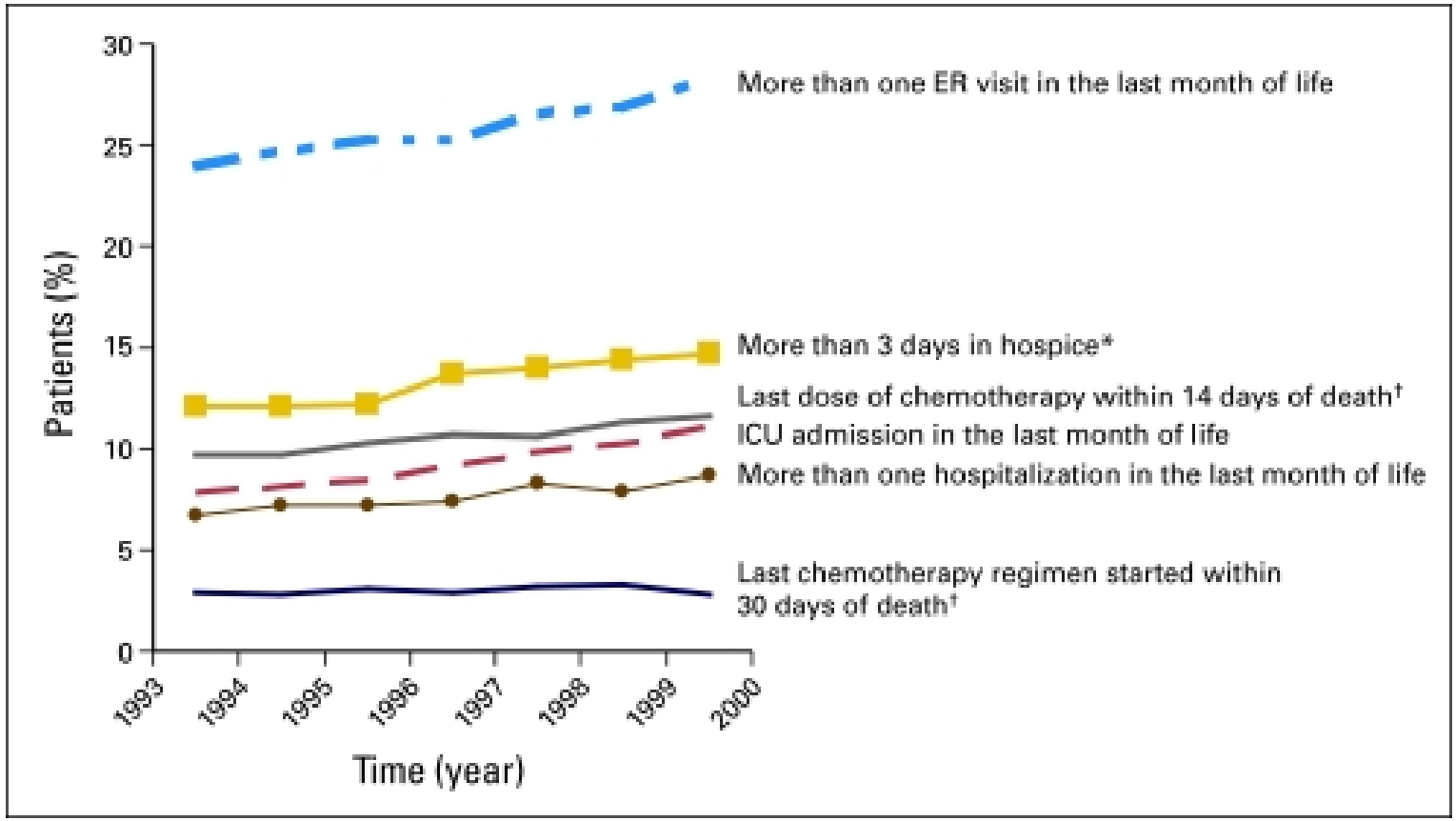
Data from MBC patients at Dana-Farber Cancer Institute



Seah DS, et al. J Natl Compr Canc Netw. 2014;12(1):71-80.

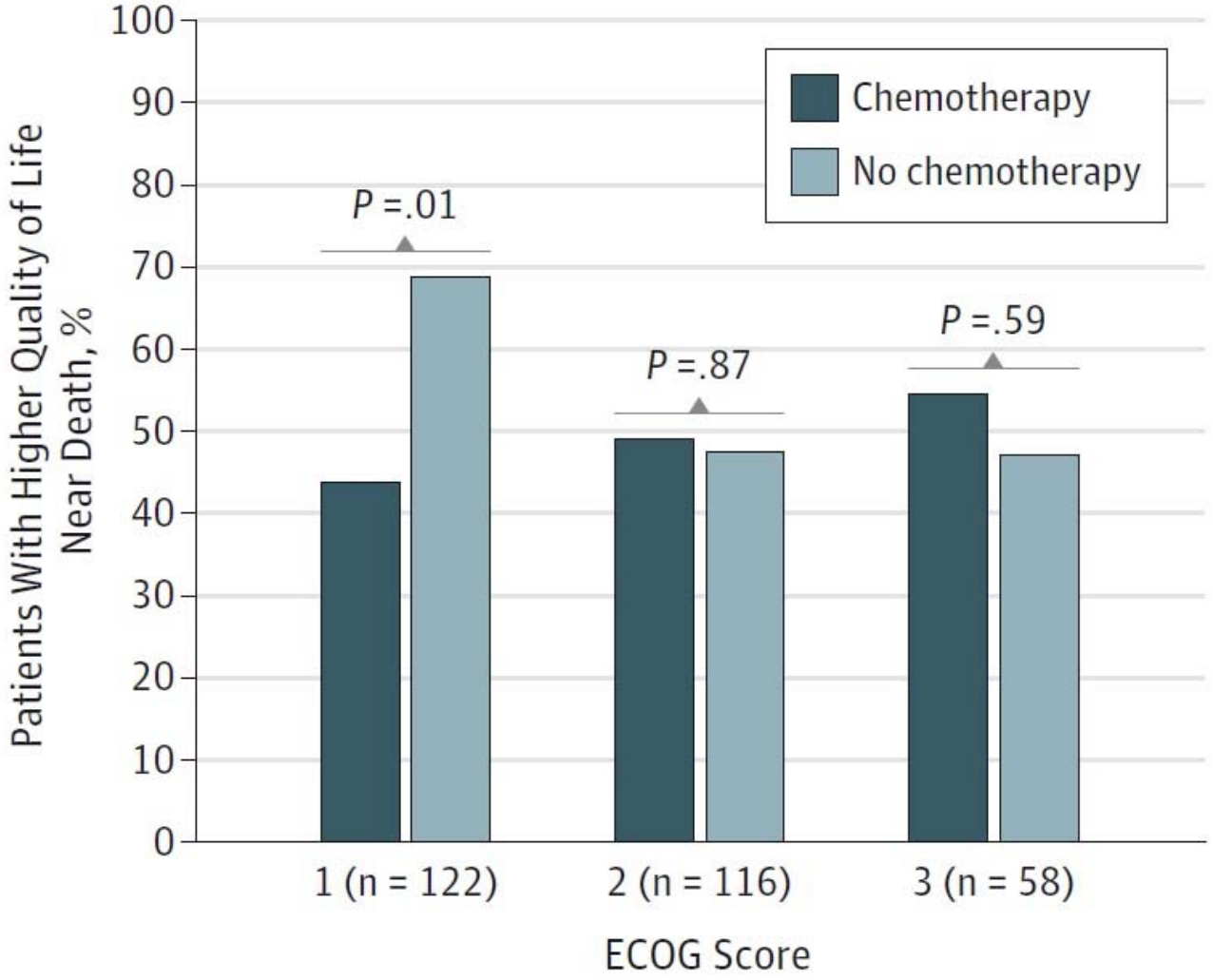
Avoid Cancer Overtreatment

ASCO Quality Oncology Practice Initiative: CT cessation in the last 2 weeks of life to improve clinical practice



McNiff KK, et al. J Clin Oncol. 2008;26(23):3832-7; Earle CC, et al. J Clin Oncol 2008;26:3860–3866.

QOL near death can be harmed by CT use near death



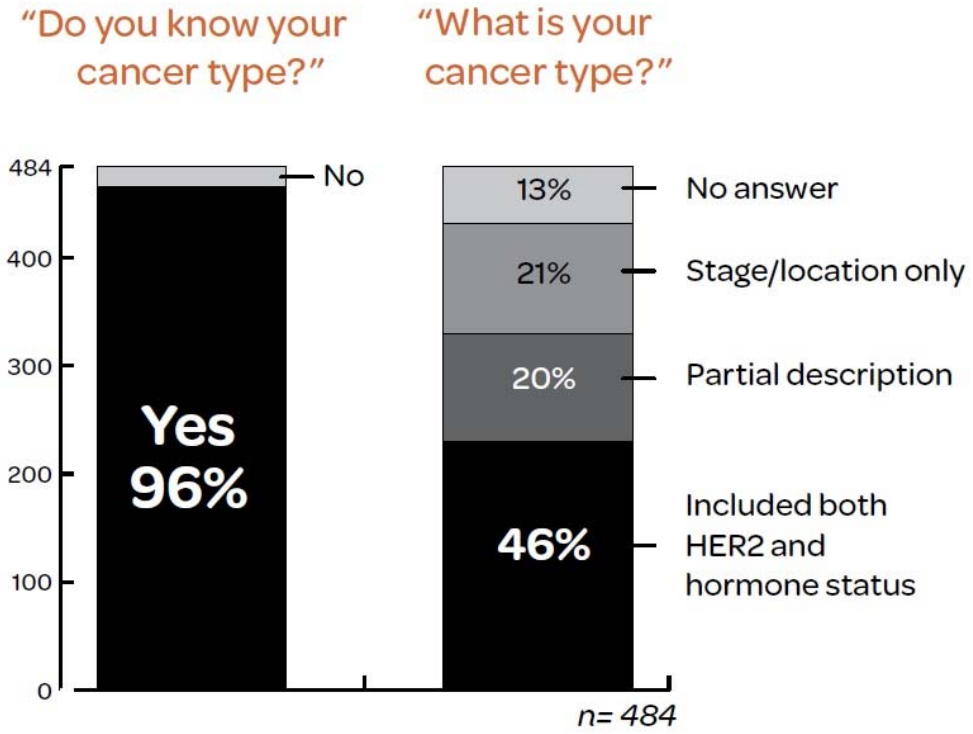
Prigerson HG, et al. JAMA Oncol. 2015;1(6):778-84.

MBCalliance's survey on 500 MBC patients

Patients may not have enough knowledge about their disease

Patients likely overestimate their knowledge.

Not realizing they know less than they should, they also likely overestimate their ability to participate in decision making.



33%

felt they don't have enough knowledge to participate in decision making

58%

felt rushed and that starting treatment was urgent; 24% got a second opinion; 38% did not research prior to starting treatment

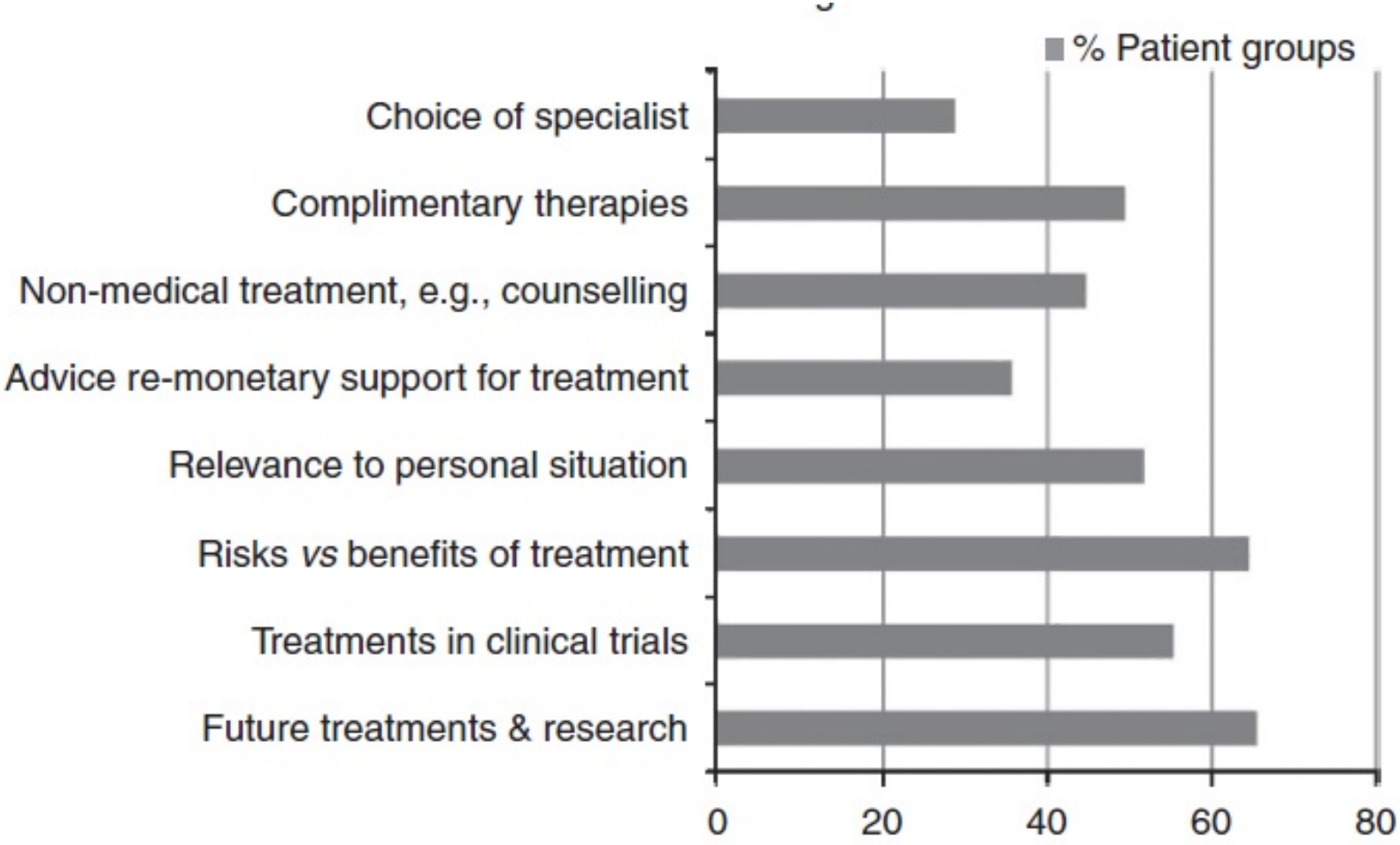
71%

did not recall goals and hobbies as part of pretreatment discussions

http://www.mbcalliance.org/education-access-initiatives/dandelion

Treatment information needs of patients living with metastatic breast cancer

Data from pan-European patient survey



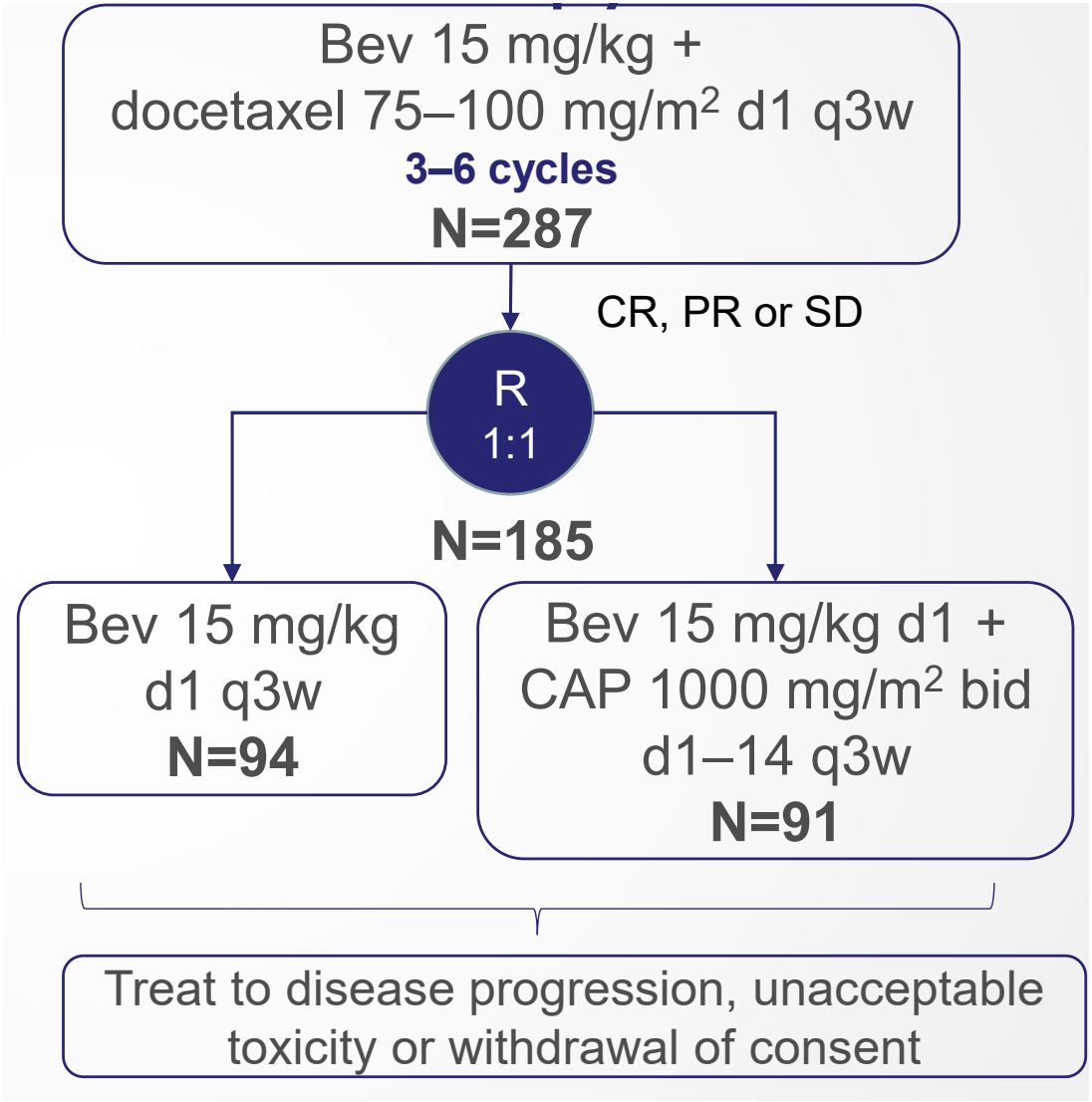
Harding V, et al. Br J Cancer. 2013;109(6):1543-8.

MTD regimens for a fixed period

- Taxane – based therapy, preferably as single-agents, are usually considered as first-line treatments of choice in HER2-negative MBC
- Prolongation of taxane exposure until disease progression is unrealistic because of cumulative toxic effects

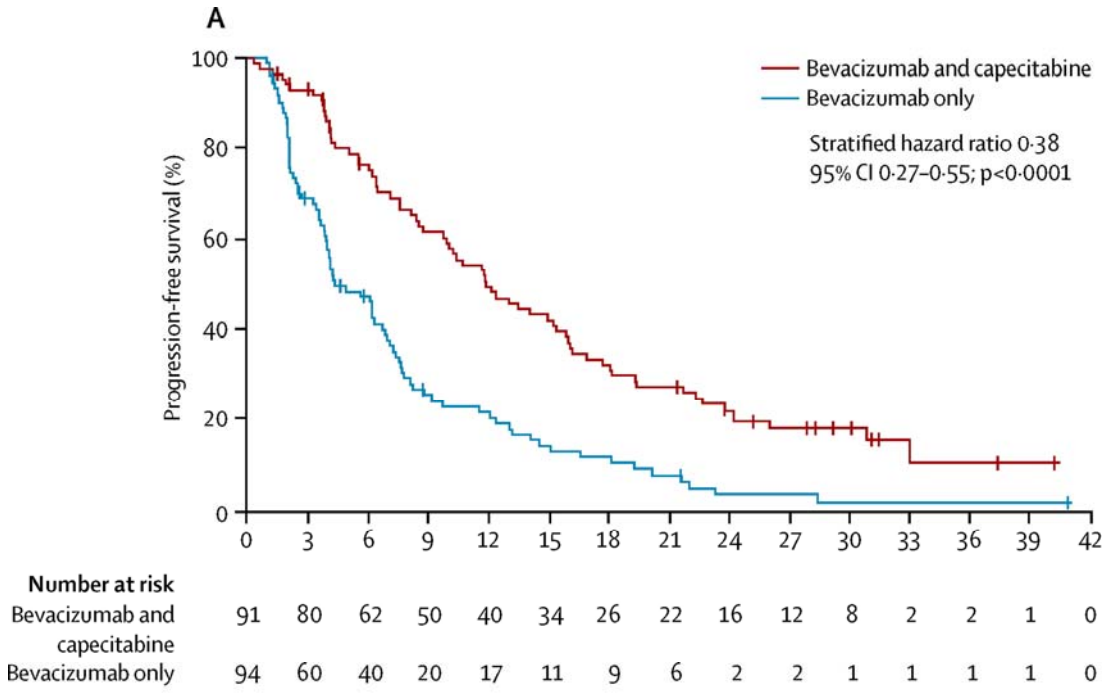
IMELDA: Evaluation of prolonged treatment with maintenance chemotherapy

- Open-label, randomised study
- Women with HER2-negative
- metastatic breast cancer
- No previous chemotherapy for metastatic breast cancer
- Stratification factors:
  - Oestrogen receptor status (positive vs. negative)
  - Visceral metastases (present vs. absent)
  - Response status (SD vs. response vs. non-measurable disease)
  - LDH concentration (≤1.5 vs. >1.5 x ULN)

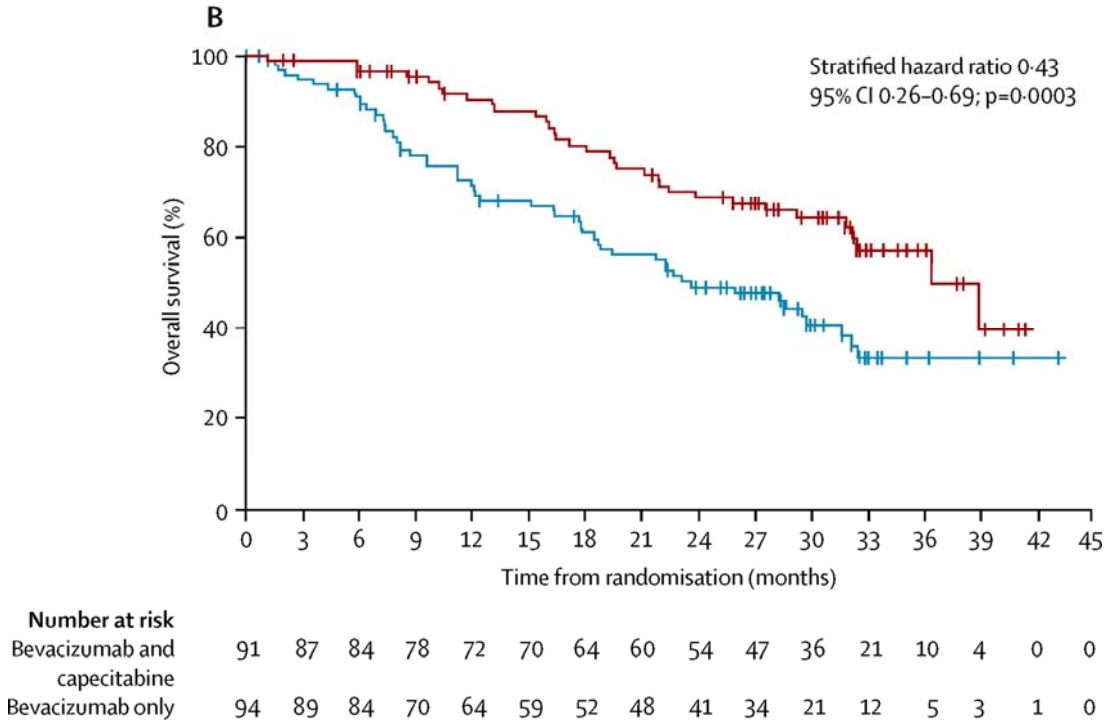


Gligorov J et al. Lancet Oncol 2014

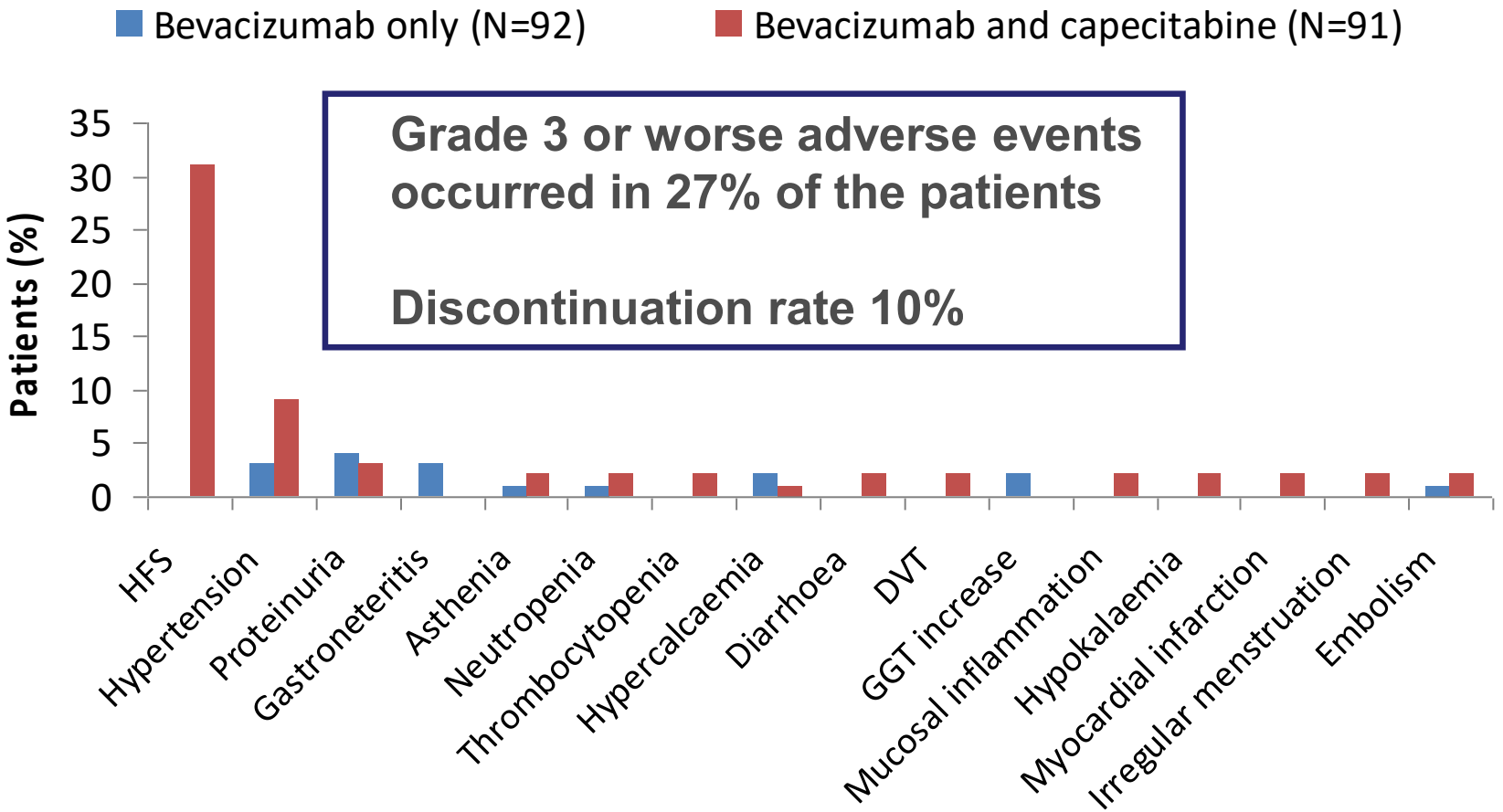
PFS


A
Bevacizumab and capecitabine
Bevacizumab only
Stratified hazard ratio 0·38
95% CI 0·27–0·55; p<0·0001
Progression-free survival (%)
100
80
60
40
20
0
0 3 6 9 12 15 18 21 24 27 30 33 36 39 42
Number at risk
Bevacizumab and capecitabine 91 80 62 50 40 34 26 22 16 12 8 2 2 1 0
Bevacizumab only 94 60 40 20 17 11 9 6 2 2 1 1 1 1 0

OS

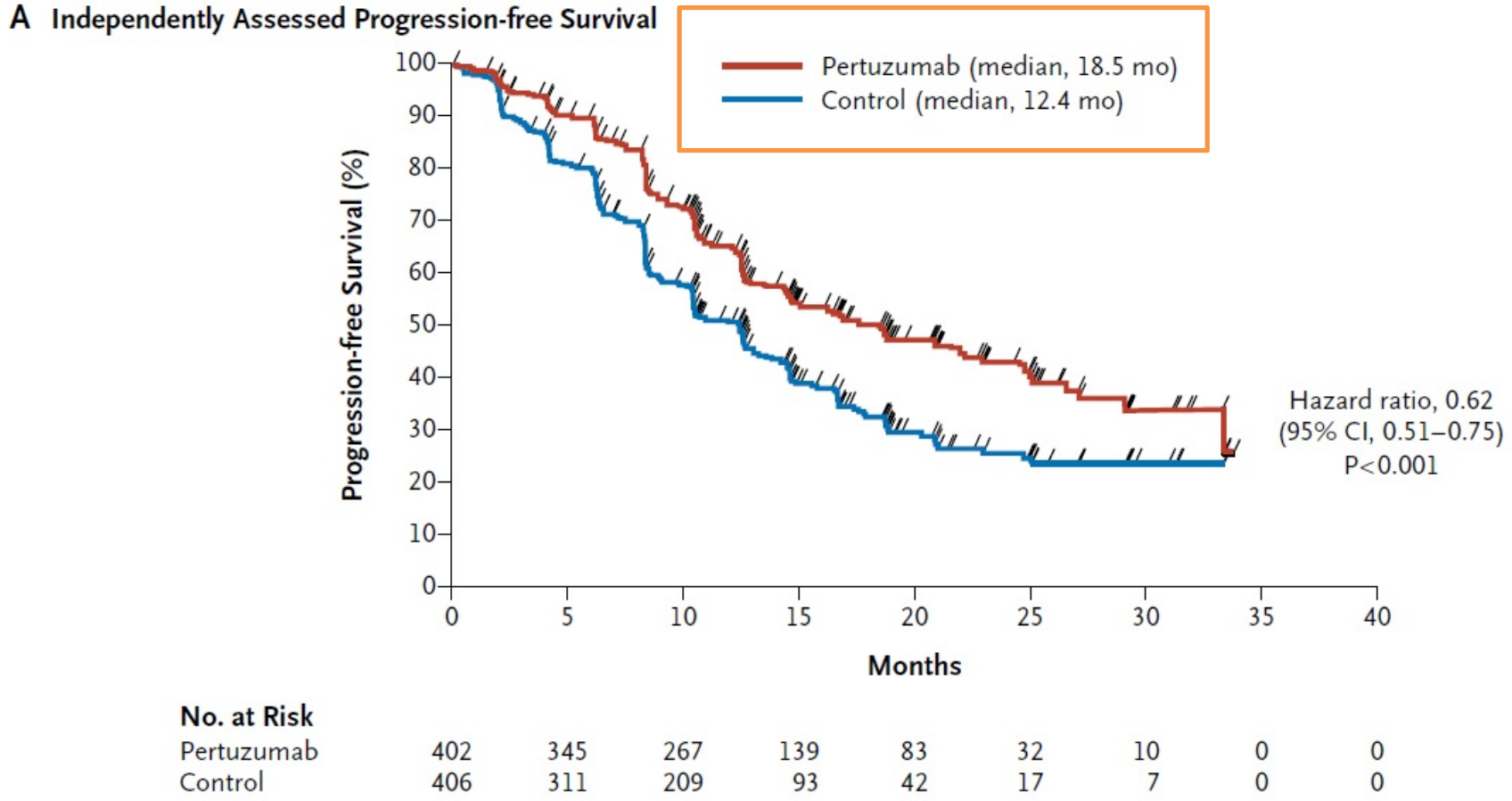

B
Stratified hazard ratio 0·43
95% CI 0·26–0·69; p=0·0003
Overall survival (%)
100
80
60
40
20
0
0 3 6 9 12 15 18 21 24 27 30 33 36 39 42 45
Time from randomisation (months)
Number at risk
Bevacizumab and capecitabine 91 87 84 78 72 70 64 60 54 47 36 21 10 4 0 0
Bevacizumab only 94 89 84 70 64 59 52 48 41 34 21 12 5 3 1 0

Adverse Events



CLEOPATRA trial

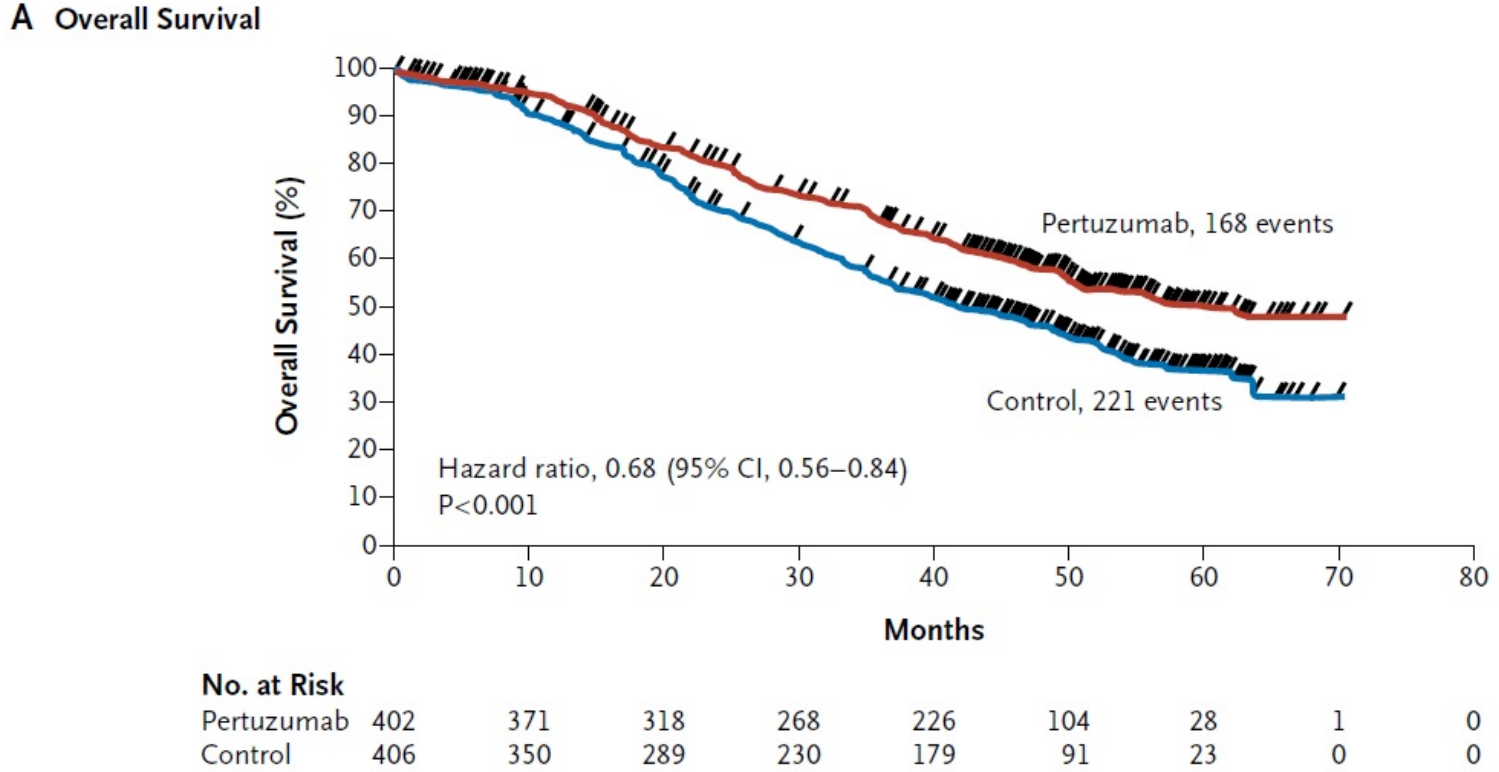
Pertuzumab, Trastuzumab, and Docetaxel in HER2+ MBC



Baselga J, et al. N Engl J Med. 2012;366(2):109-19.

CLEOPATRA trial

Pertuzumab, Trastuzumab, and Docetaxel in HER2+ MBC

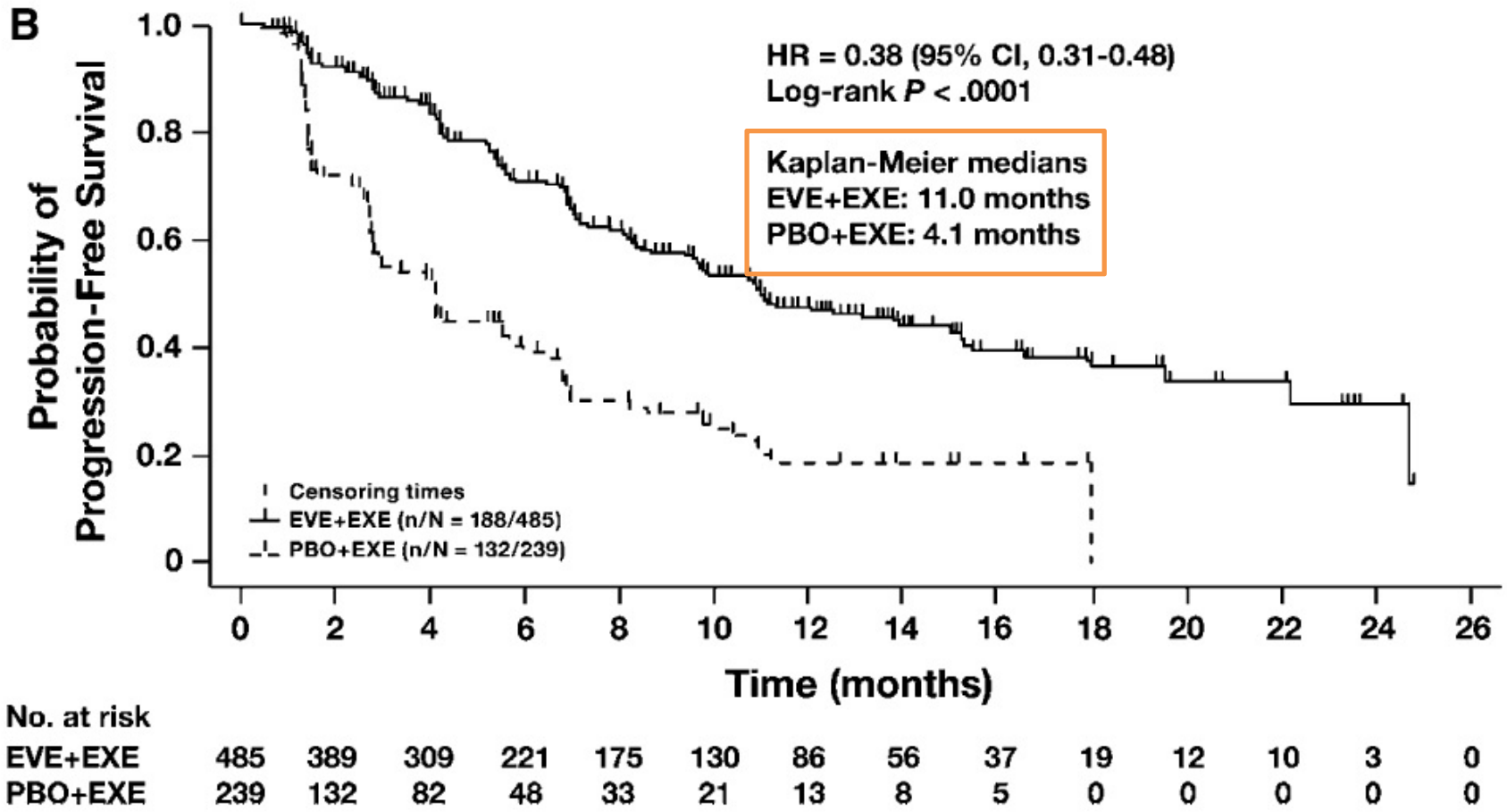


Median OS was **15.7 months longer** in the pertuzumab group (56.5 vs. 40.8 months)

Swain SM, et al. N Engl J Med. 2015;372(8):724-34.

BOLERO-2 trial

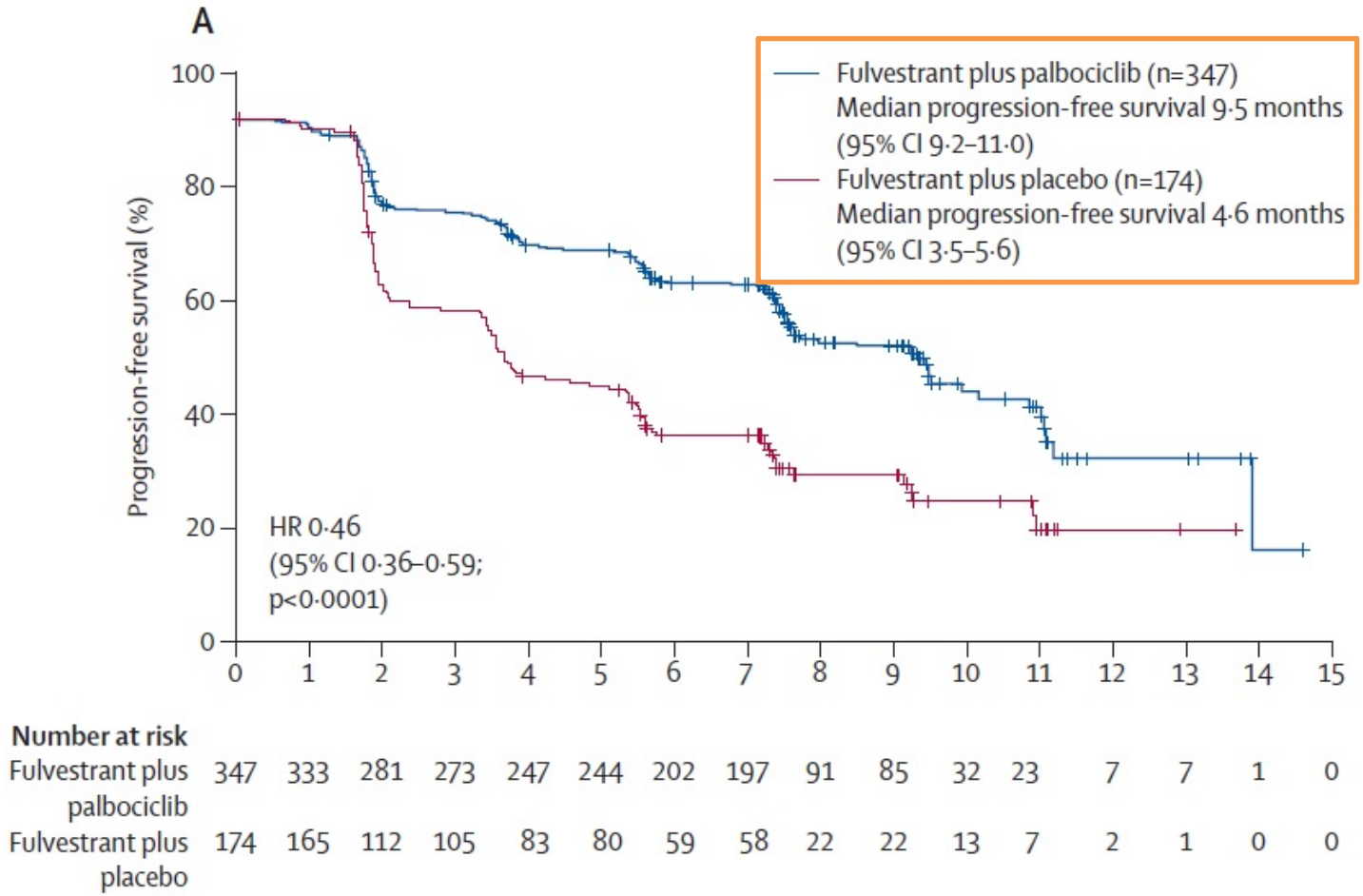
Everolimus Plus Exemestane in Postmenopausal HR+ Advanced BC



Yardley DA, et al. Adv Ther. 2013;30(10):870-84.

PALOMA-3 trial

Fulvestrant plus palbociclib in HR+ HER2- MBC that progressed on previous endocrine therapy

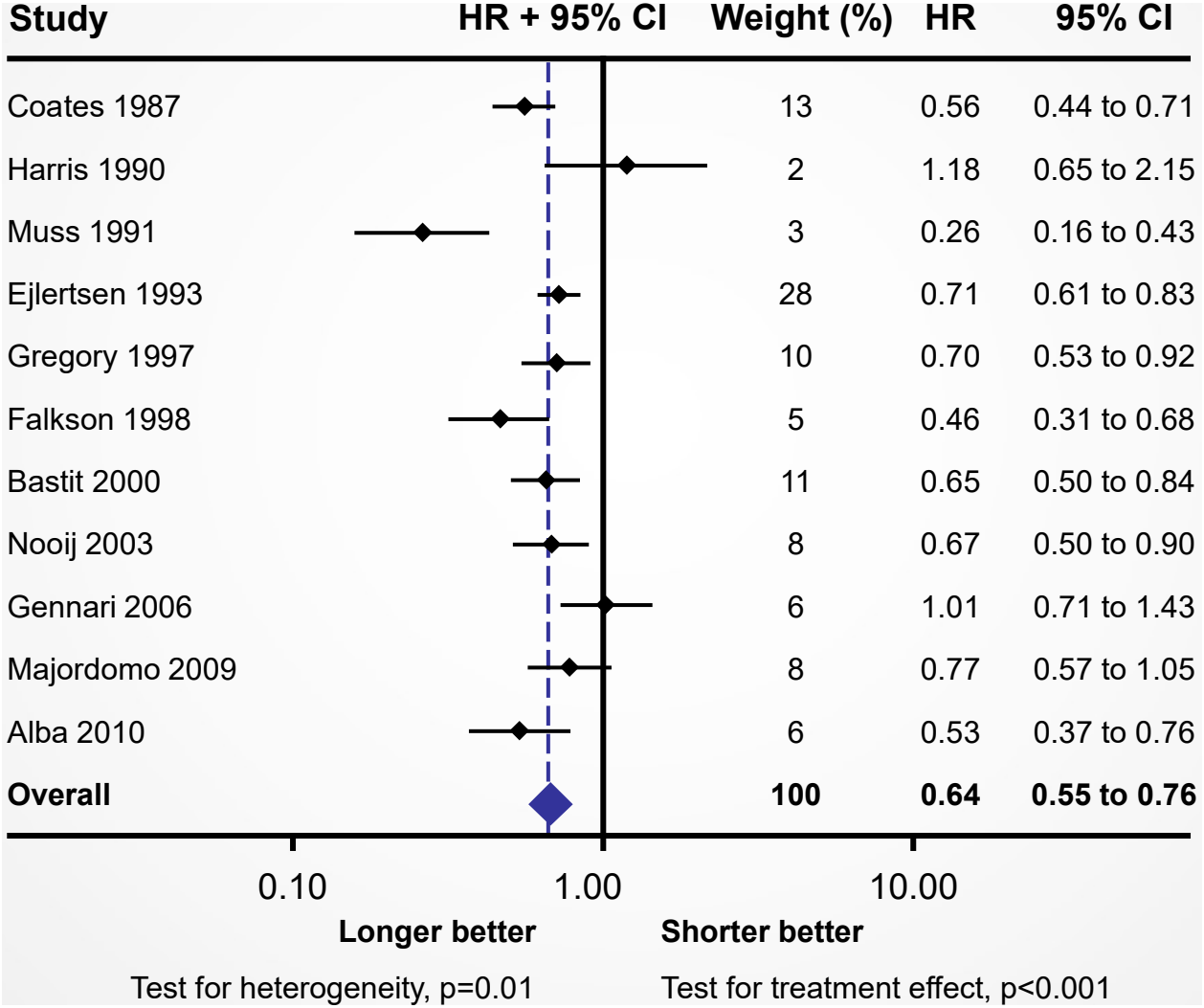


Cristofanilli M, et al. Lancet Oncol. 2016;17(4):425-39.

FIXED PERIOD OF TREATMENT

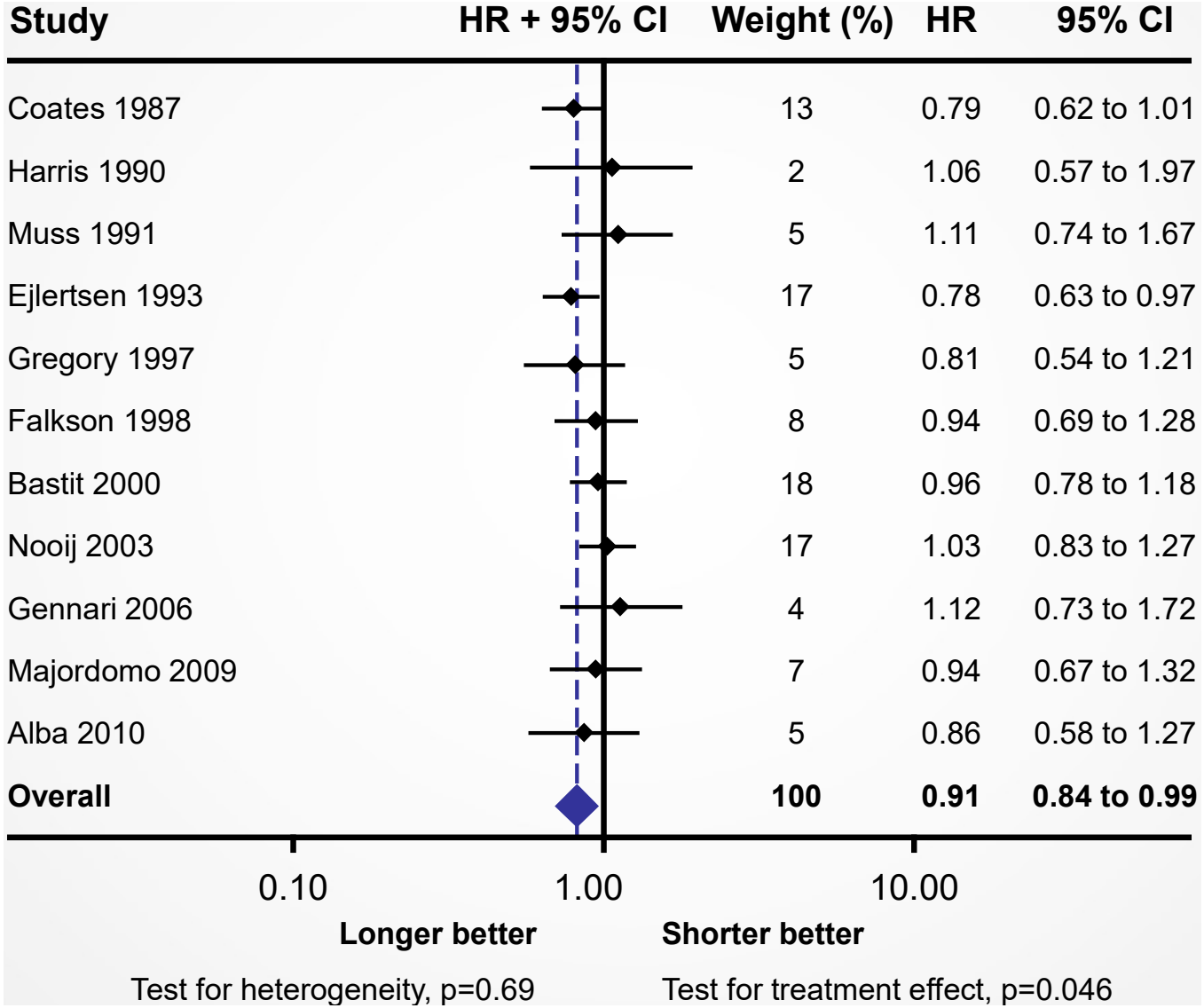
PROLONGED TREATMENT (MAINTENANCE STRATEGY)

Duration of treatment and PFS



Gennari A et al. J Clin Oncol 2011;29:2144–9

Duration of treatment and OS

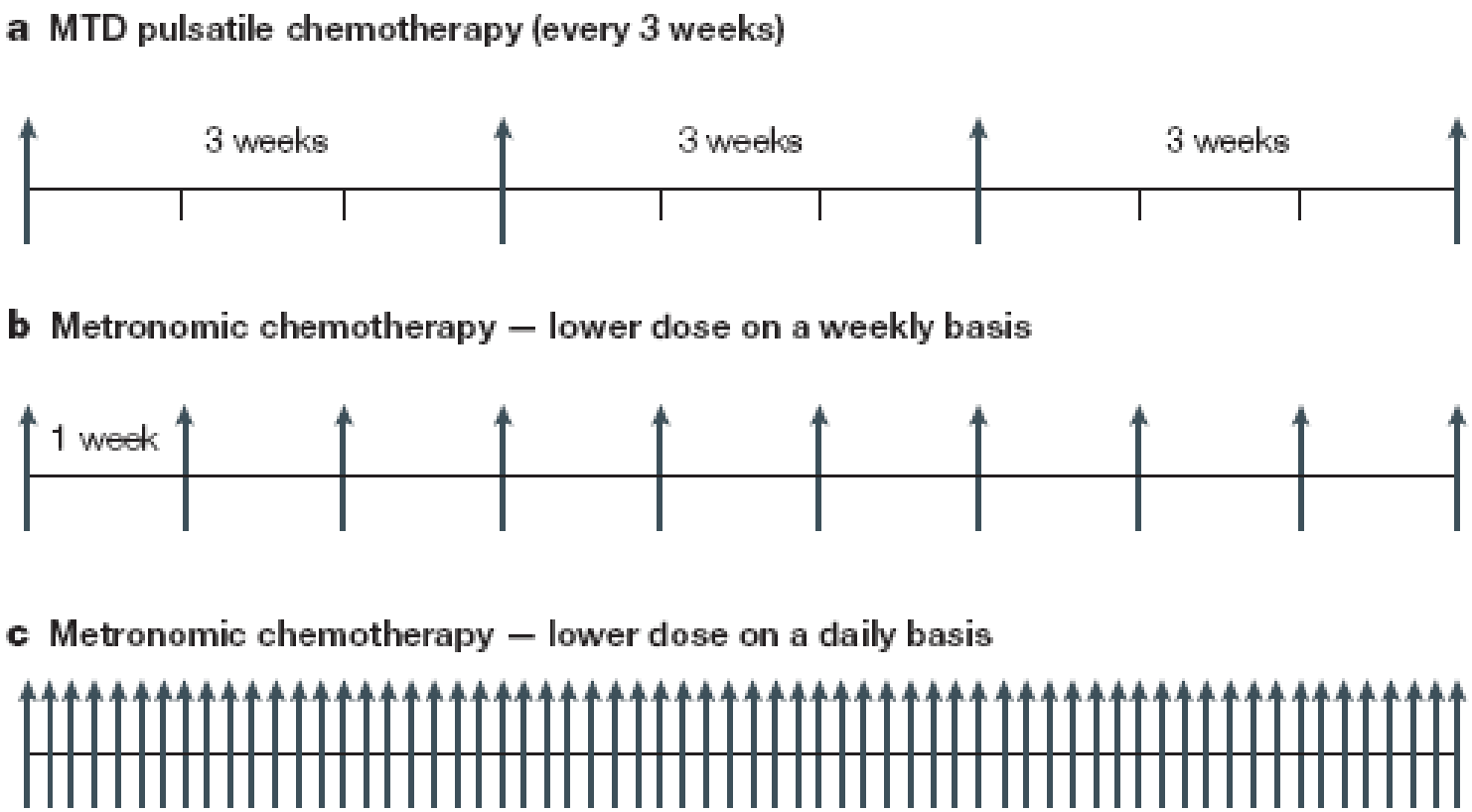


Gennari A et al. J Clin Oncol 2011;29:2144–9

Ideal Drugs for Prolonged Treatment

- Effective single agents
- Proven clinical activity in MBC setting
- Preferably oral, to avoid prolonged hospitalization
- Preferably no cumulative side effects
- Allows QOL preservation

Metronomic Approach

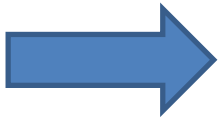


Metronomic chemotherapy with oral vinorelbine (mVNR) and capecitabine (mCAPE) in advanced HER2-negative breast cancer patients: is it a way to optimize disease control? Final results of the VICTOR-2 study

M. E. Cazzaniga[1] · L. Cortesi[2] · A. Ferzi[3] · L. Scaltriti[4] · F. Cicchiello[1] · M. Ciccarese[5] · S. Della Torre[6] · F. Villa[7] · M. Giordano[8] · C. Verusio[9] · M. Nicolini[10] · A. R. Gambaro[11] · L. Zanlorenzi[12] · E. Biraghi[13] · L. Legramandi[14] · E. Rulli[14] · On behalf of VICTOR Study Group

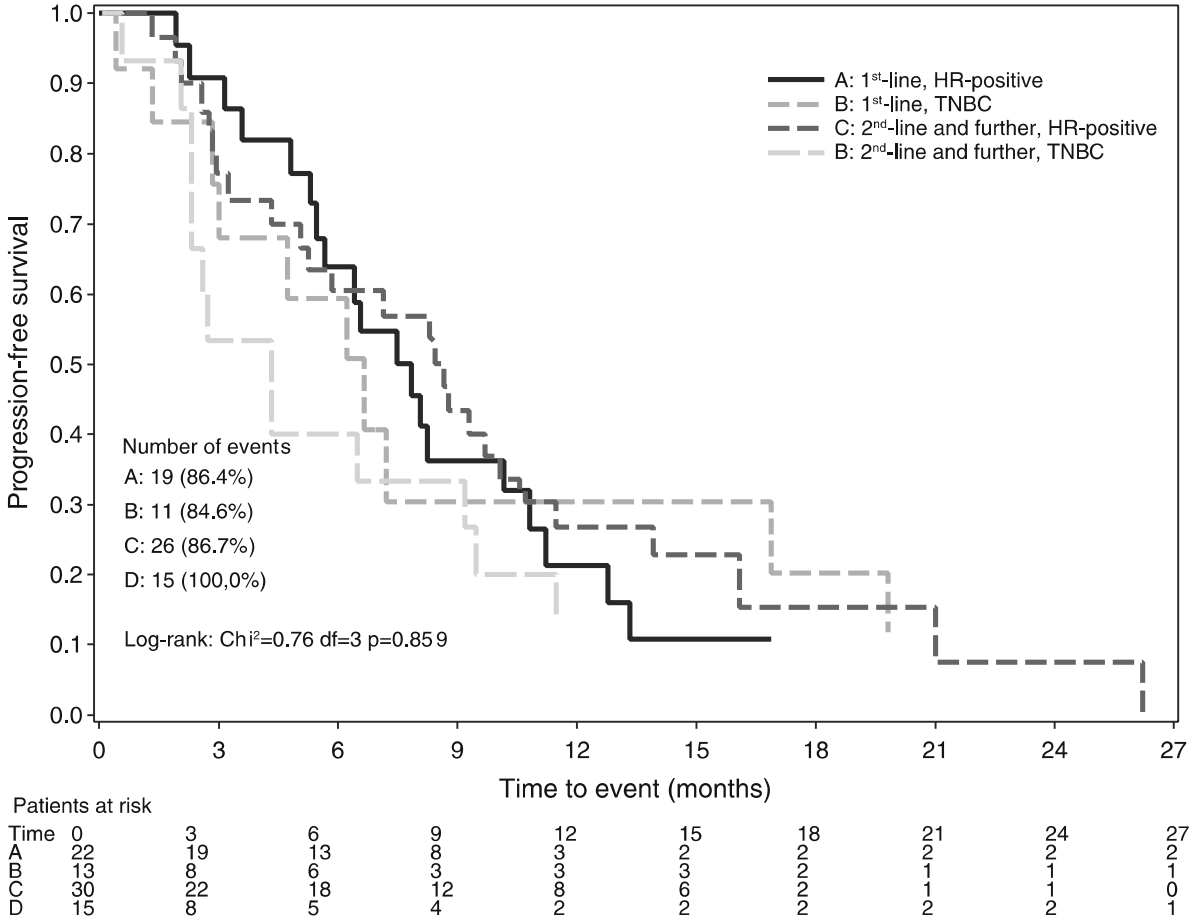
Breast Cancer Res Treat (2016) 160:501–509

Oral VNR 40 mg on Days 1, 3 and 5 per week
+
Capecitabine 500 mg tid continuously



Disease progression

Objective response rate (ORR)	First-line (Group 1) $N = 31$	Second-line (Group 2) $N = 43$	Overall $N = 74$
Responders (CR + PR): *n* (%)	11 (35.5)	11 (25.6)	22 (29.7)
[95 % CI]	[19.2–54.6]	[13.5–41.2]	[19.7–41.5]
Disease control rate (DCR)			
Responders (CR + PR + SD): *n* (%)	23 (74.2)	29 (67.4)	52 (70.3)
[95 % CI]	[55.4–88.1]	[51.5–80.9]	[58.5–80.3]



Other possible agents

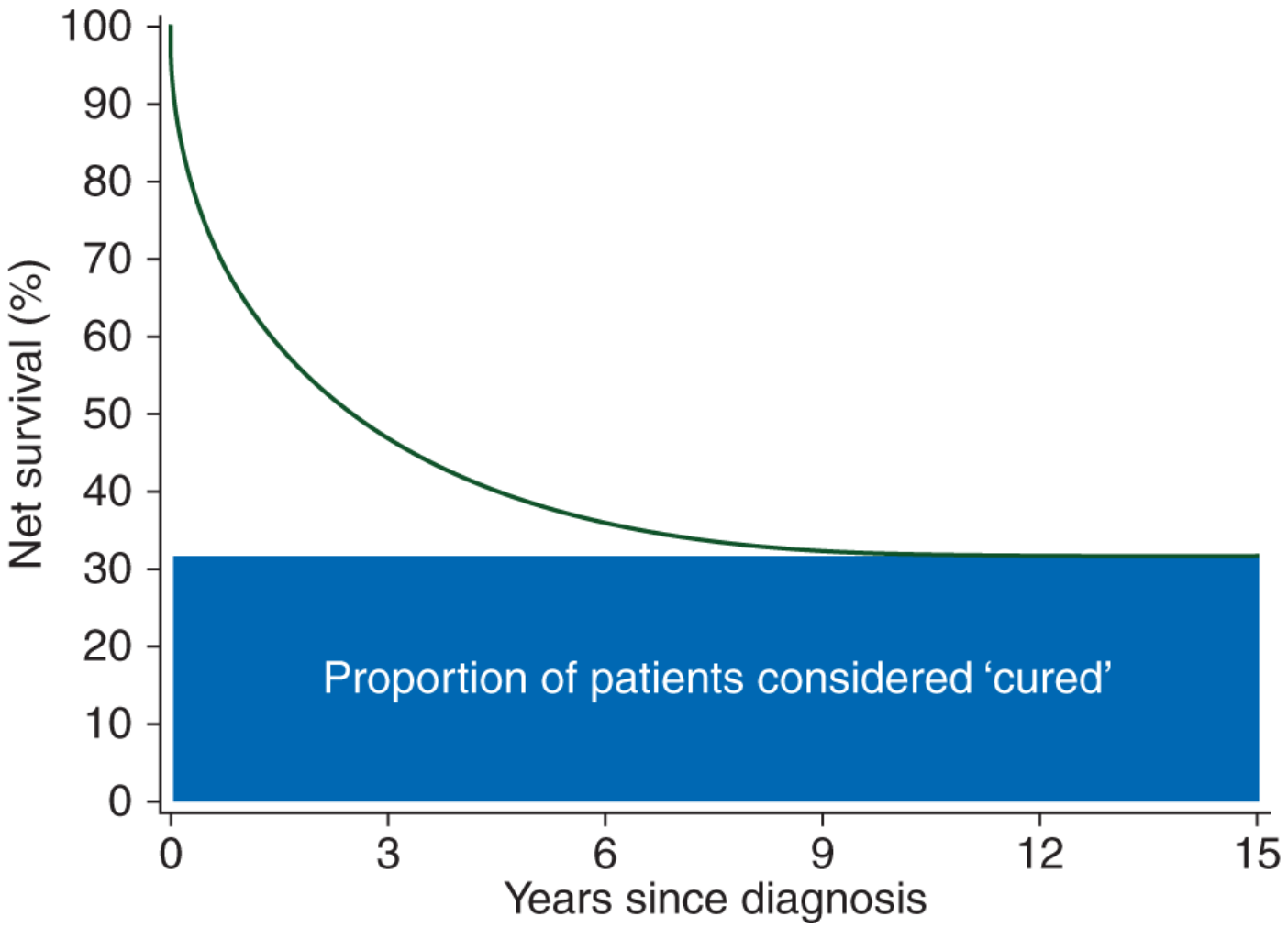
- Oral chemotherapy +/- hormonal treatment
  - Tegafur
  - YS1
  - Cyclophosphamide
  - Methotrexate …
- Oral small TKI
  - Lapatinib
  - Neratinib
  - Afatinib
  - Sutent …

Is Cure Possible?

We've come a long way to improve MBC survival

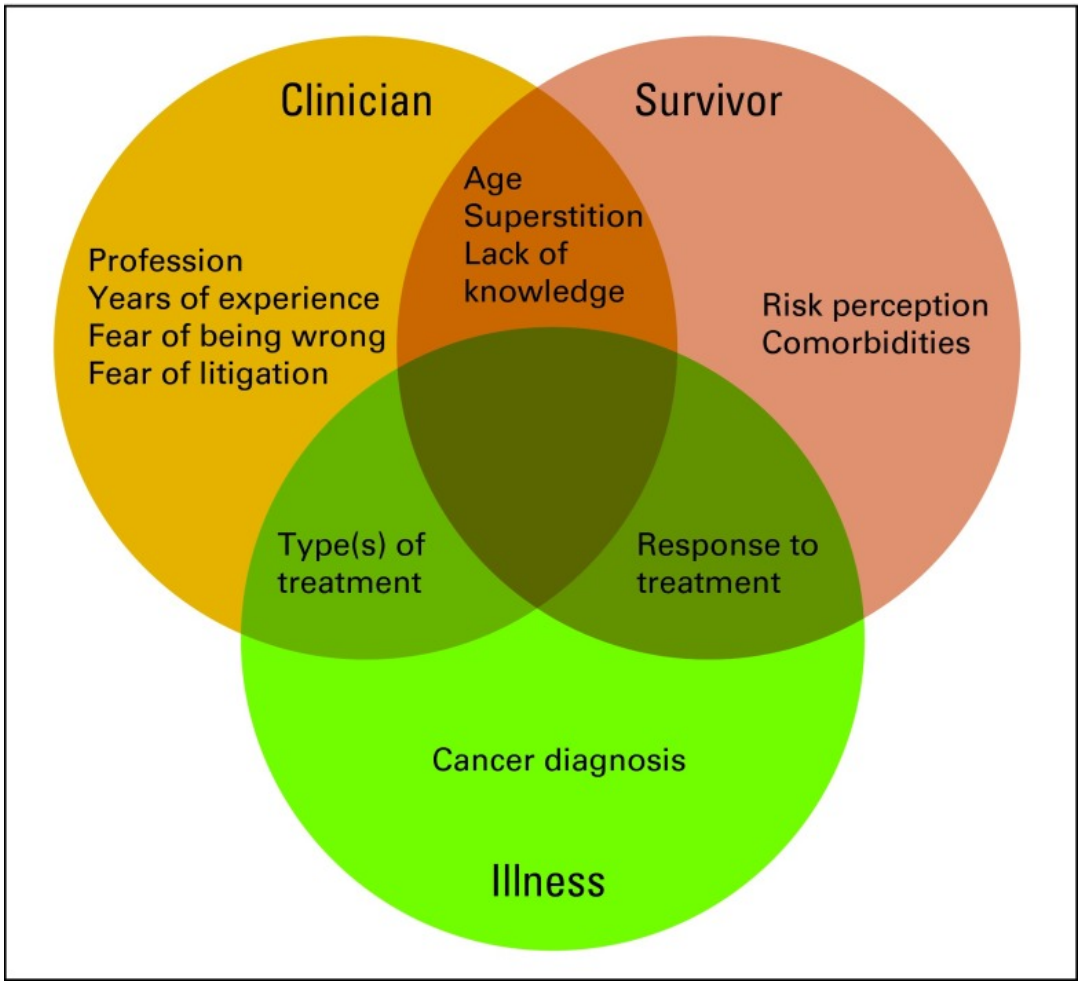
But, is it possible to "cure"?
Or maybe we are overtreating them?

Cure: disease free survival for 10-20 years or more?



Woods LM, et al. Ann Oncol. 2016; 27(11): 2025–2031; Miller K, et al. J Oncol Pract. 2013; 9(4): e136–e140.

Why is it so hard to define cure?



Miller K, et al. J Oncol Pract. 2013; 9(4): e136–e140.

Table 1. Lines of Evidence Suggesting Metastatic Breast Cancer Is Curable

Adjuvant therapy cures micrometastasis
Adjuvant therapy after isolated local-regional recurrence improves survival
Chemotherapy for overt metastatic disease produces long-term survivors
Exceptional responders with novel agents
Treatment of low-volume metastatic disease with surgery and radiation produces long-term survivors

Sledge GW Jr. J Oncol Pract. 2016;12(1):6-10.

International Guidelines for Management of Metastatic Breast Cancer: Can Metastatic Breast Cancer Be Cured?

Olivia Pagani, Elżbieta Senkus, William Wood, Marco Colleoni, Tanja Cufer, Stella Kyriakides, Alberto Costa, Eric P. Winer, Fatima Cardoso, on behalf of the ESO–MBC Task Force

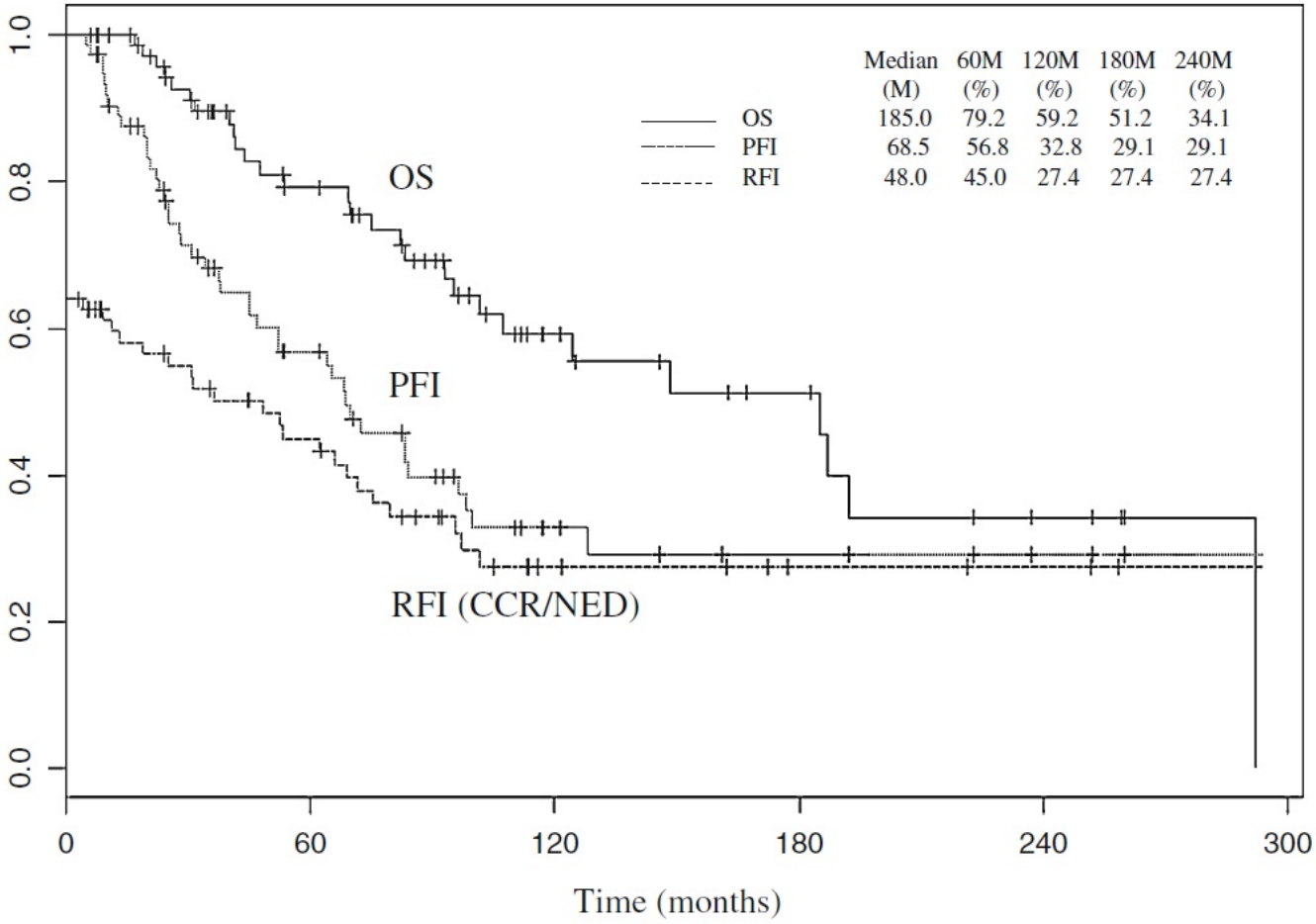
Pagani O, et al. J Natl Cancer Inst. 2010; 102(7): 456–463.

Possible clinical cure of metastatic breast cancer: lessons from our 30-year experience with oligometastatic breast cancer patients and literature review

Tadashi Kobayashi · Tamotsu Ichiba · Toshikazu Sakuyama · Yasuhiro Arakawa · Eijiroh Nagasaki · Keisuke Aiba · Hiroko Nogi · Kazumi Kawase · Hiroshi Takeyama · Yasuo Toriumi · Ken Uchida · Masao Kobayashi · Chihiro Kanehira · Masafumi Suzuki · Naomi Ando · Kazuhiko Natori · Yasunobu Kuraishi

Kobayashi T, et al. Breast Cancer. 2012;19(3):218-37.

OMBC as a distinct subgroup with possible clinical cure



CCR: continuing complete response, M: months, NED: no evidence of clinical disease, OMBC: oligometastatic breast cancer, OS: overall survival, PFI: progression-free interval, RFI: relapse-free interval, y: year

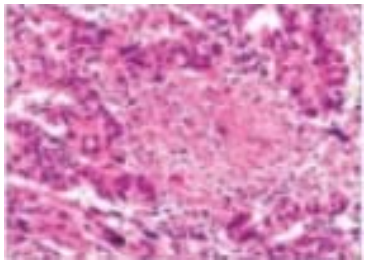
Kobayashi T, et al. Breast Cancer. 2012;19(3):218-37.

Heterogeneity of breast cancer

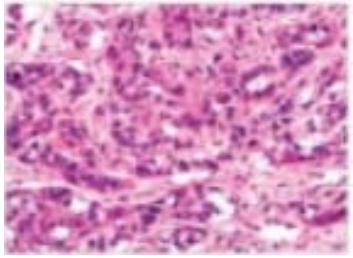
Shift to molecular methods

Intrinsic subtype by IHC

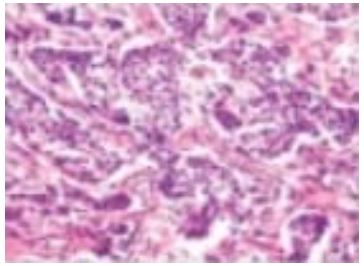
TNBC



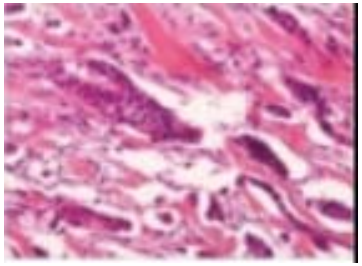
HER2



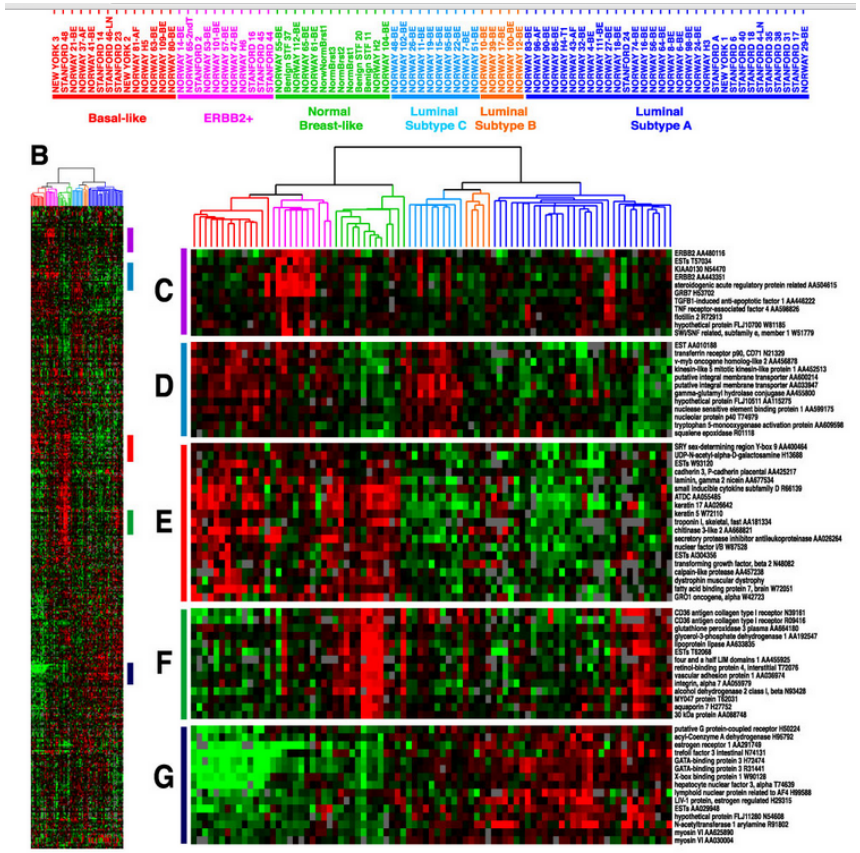
Luminal A



Luminal B



Intrinsic subtype – molecular level



Perou C, Sorlie T, Eisen M et al. Nature 2000

Perou C, Parker J, Prat A, Ellis M, Bernard P. Lancet Oncol 2010

Targeted therapy for HER2+ breast cancer

- 20-25% of breast cancers are classified as HER2+
- HER2 overexpression or amplification impacts significantly the prognosis of patients with T1a-bN0M0

Table 3. Multivariate analyses in 714 patients with pT1a-bN0M0 breast cancer.

	Variables	HR (95% CI)[1]	*P*-value
OS	HER2 status[2]	3.891 (1.583–9.565)	0.0031
	HR status[2]		0.3453
	SBR		0.2392
DFS	HER2 status[2]	3.571 (1.802–7.080)	0.0003
	SBR		0.2773
LRFS	HER2 status[2]	5.851 (1.551–22.077)	0.0091
MFS	HER2 status[2]	4.105 (1.886–8.936)	0.0004
	Age (>50 vs. ≤50)	3.420 (1.053–11.110)	0.0408
	SBR		0.0510

1. Li SG, et al. Biomed Rep. 2013 ;1(4): 499–505. 2. Rouanet P, et al. Cancer Med. 2014;3(1):134–142.

HER2 status of circulating tumor cells (CTCs)

- A total of 254 patients with metastatic breast cancer from nine German university breast cancer centers were enrolled in this prospective study
- HER2-positive CTCs can be detected in a relevant number of patients with HER2 negative primary tumors

Table 5 HER2 status of CTCs determined by the assays and correlation with primary tumor HER2 status

	No. (%)				*P* value
	Total	HER2 status of the primary tumor			
		Negative	Positive	Unknown	
CellSearch assay					
CTC positive[a]	122	76	31	15	0.02[c] ($\kappa = 0.226$)
HER2 negative	72 (59)	51 (67)	13 (42)	8 (53)	
HER2 positive	50 (41)	25 (33)	18 (58)	7 (47)	
AdnaTest BreastCancer					
CTC positive[b]	90	57	22	11	
HER2 negative	48 (53)	29 (51)	13 (59)	6 (54)	0.51[c] ($\kappa = -0.068$)
HER2 positive	42 (47)	28 (49)	9 (41)	5 (45)	

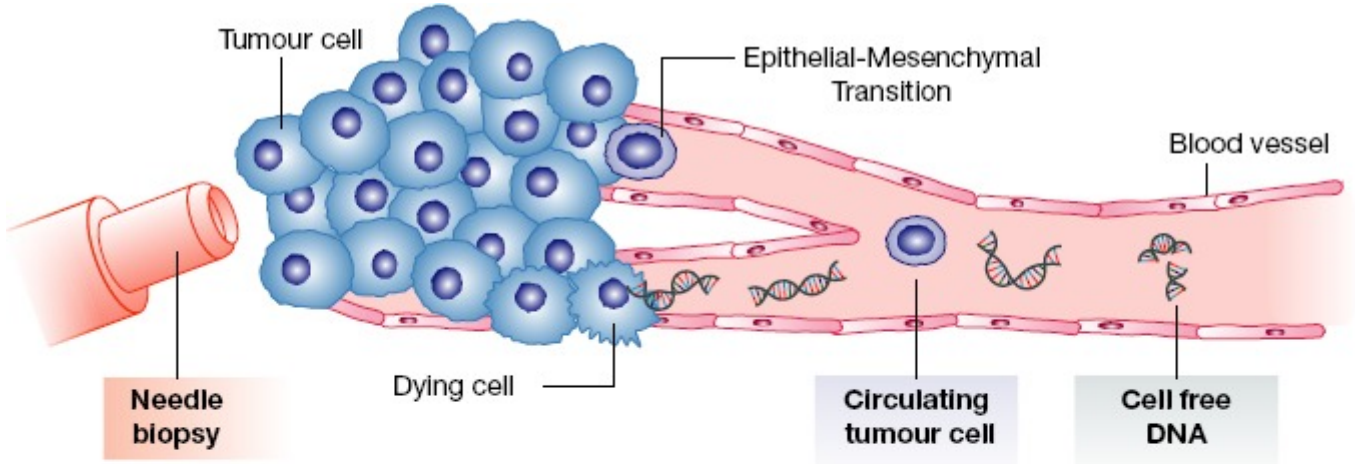
[a] HER2 positive if at least one cell is strongly stained for HER2 (3+)

[b] HER2 positive if a HER2 transcript has been detected

[c] Excluding those with unknown HER2 status

Fehm T, et al. Breast Cancer Res Treat. 2010;124(2):403-12.

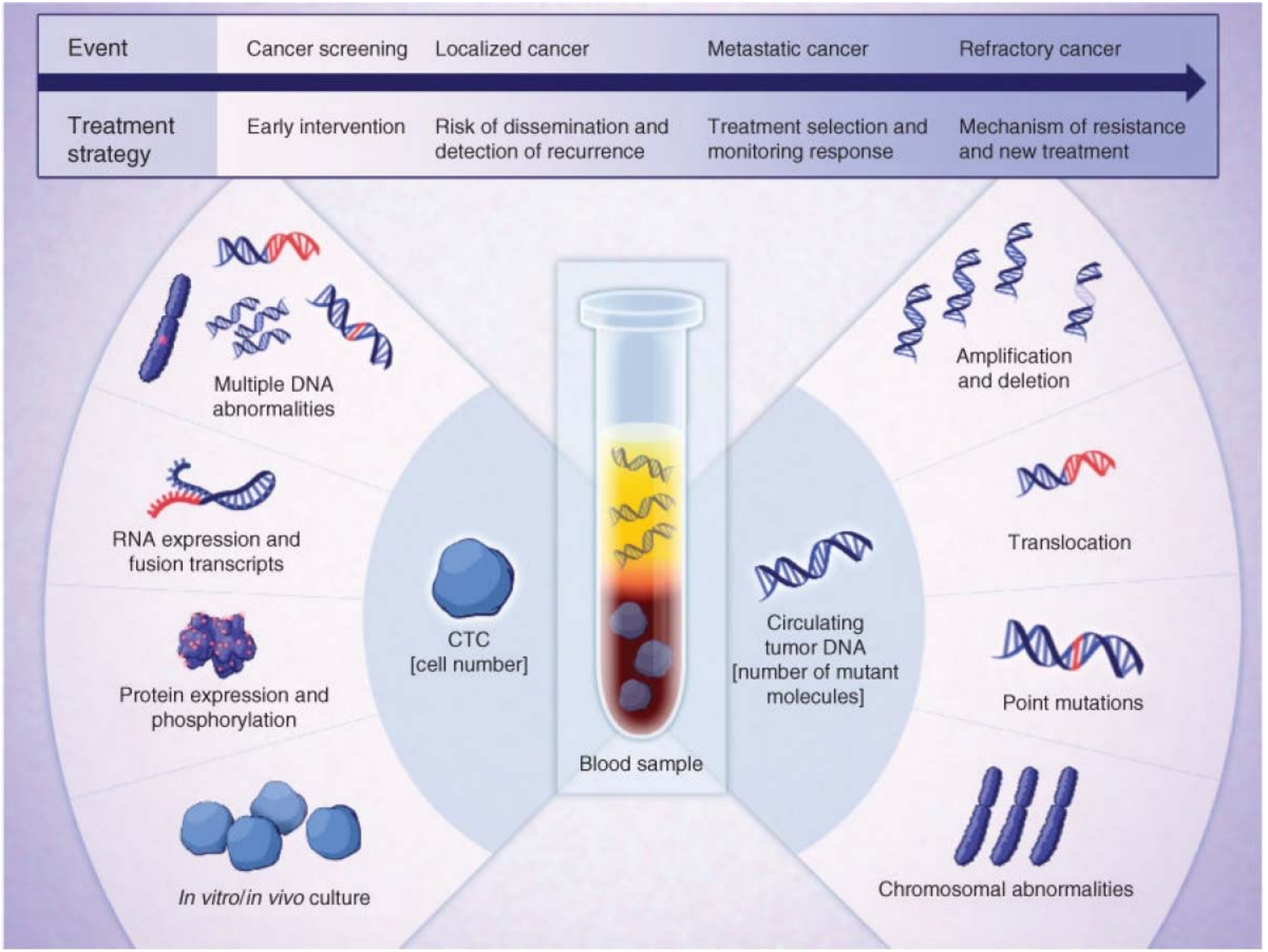
Tumour tissue biopsy, circulating tumour cell analysis, and cell-free DNA



	Biopsy	CTC	cfDNA
Invasive	+	–	–
All patients eligible	–	+	+
Instrumentation required	+	+	–
WGA required	–	+	+/–
RNA profiling	+	+	–
Research applicability	+++	++	+
Biomarker applicability	–	++	+++

WGA, whole-genome amplification

Wyatt AW, Gleave ME. EMBO Mol Med. 2015;7(7):878-94.



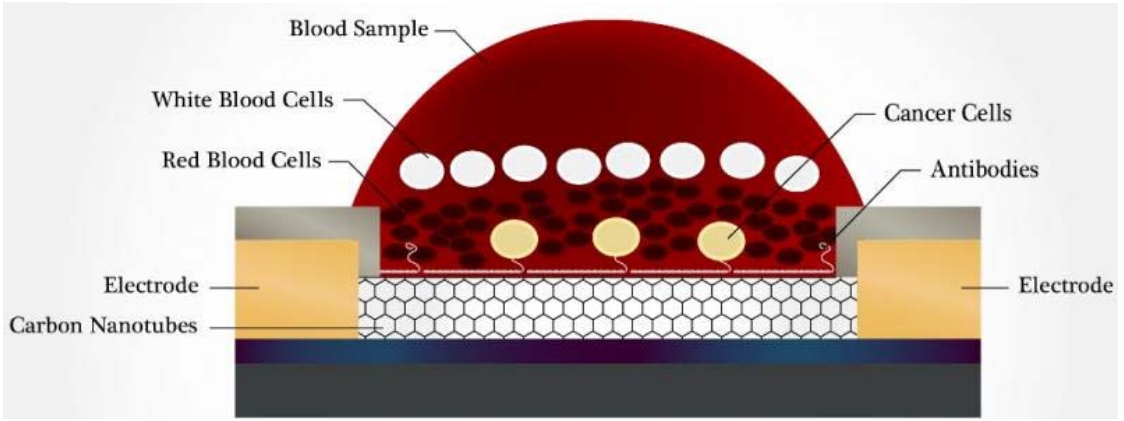
Haber DA, Velculescu VE. Cancer Discov. 2014;4(6):650-61.

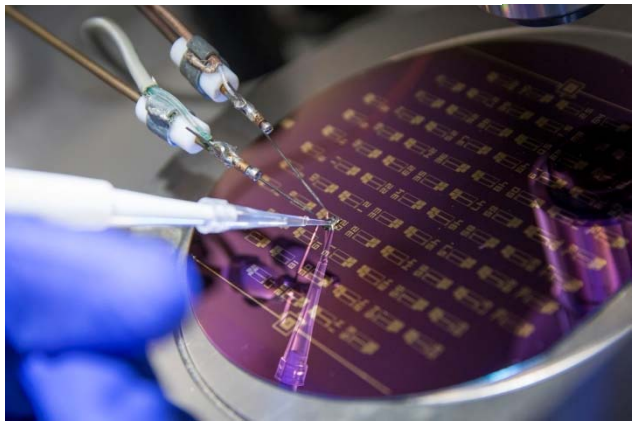
WPI Researchers Build "Liquid Biopsy" Chip that Detects Metastatic Cancer Cells in a Drop of Blood

More effective than existing microfluidic devices, the breakthrough technology paves the way for clinical development of simple blood tests for many cancers.

December 15, 2016

A chip developed by mechanical engineers at Worcester Polytechnic Institute (WPI) can trap and identify metastatic cancer cells in a small amount of blood drawn from a cancer patient. The breakthrough technology uses a simple mechanical method that has been shown to be more effective in trapping cancer cells than the microfluidic approach employed in many existing devices.





https://www.wpi.edu/news/wpi-researchers-build-liquid-biopsy-chip-detects-metastatic-cancer-cells-drop-blood

BioWheel for Visualizing the Big Data of Breast Cancer

- BioWheel showed connections between proteins and their expression levels as represented in colors that changed from light green (for no expression) to dark red (for high expression) as time progressed from the inside to the outside of the wheel.
- Shifting connections between proteins on the inner ring are seen in the center.

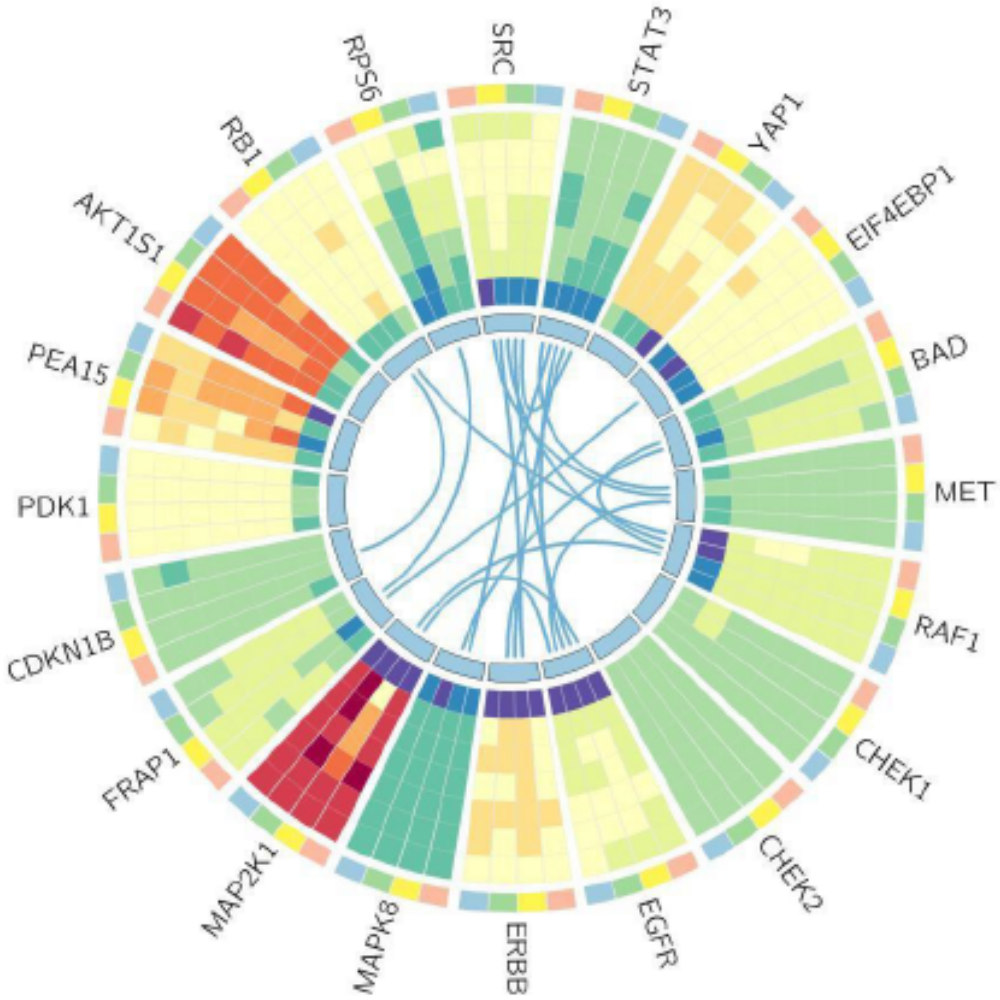
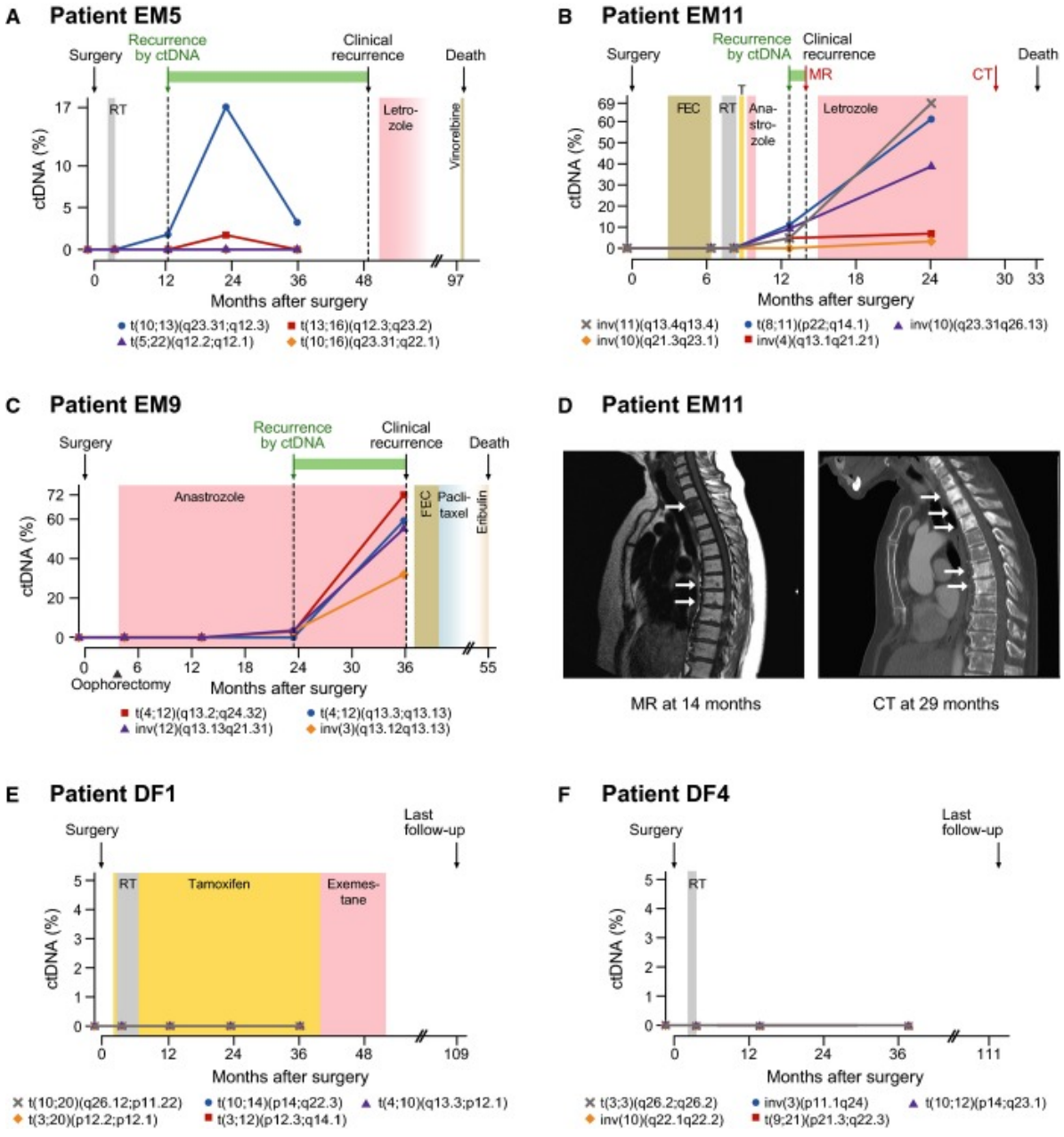


Figure 3. Mock-up of BioWheel visualization tool.

Hill SM, et al. Nat Methods. 2016;13(4):310-8.



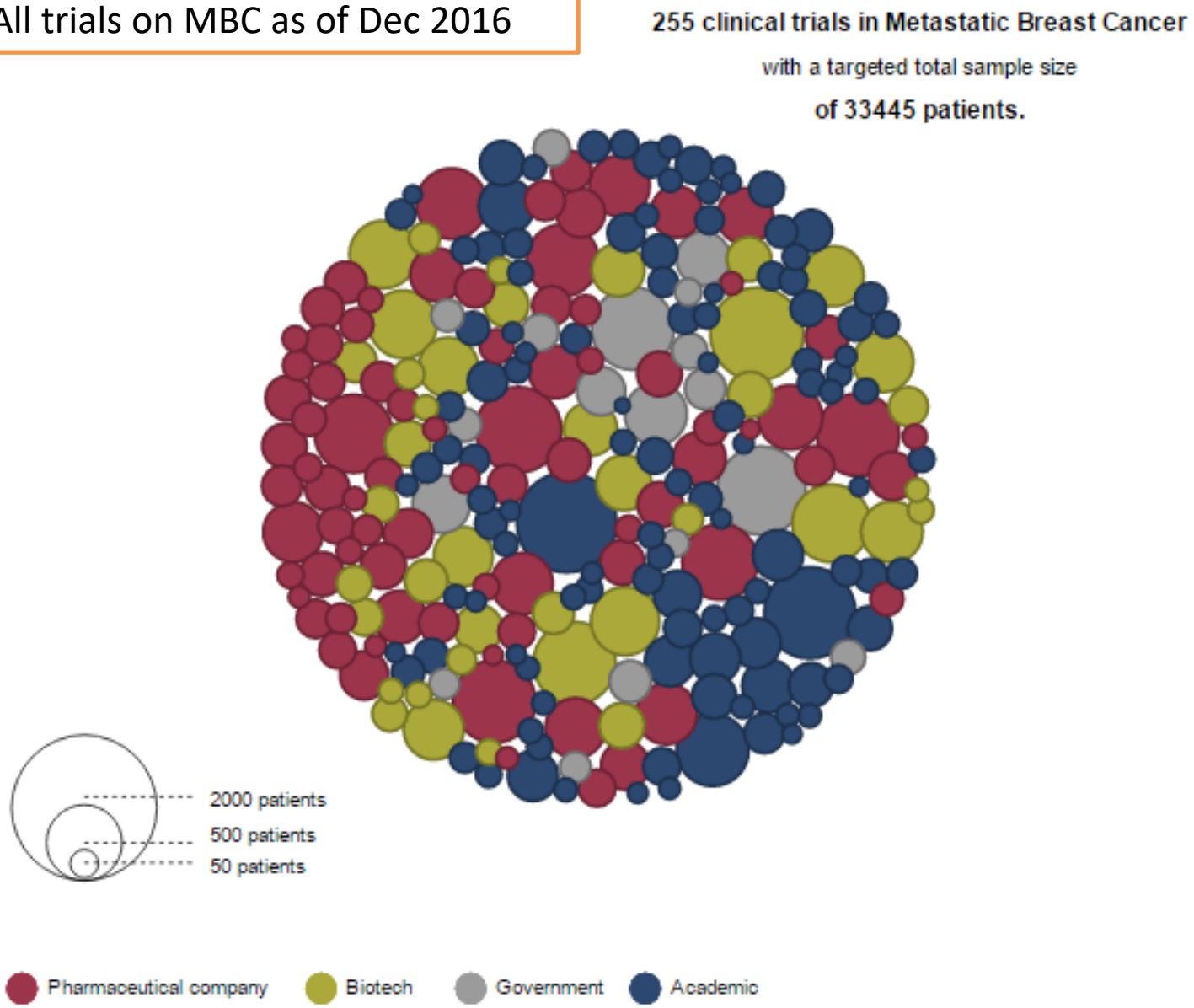
Serial monitoring of circulating tumor DNA in patients with primary breast cancer for detection of occult metastatic disease

Olsson E, et al. EMBO Mol Med. 2015;7(8):1034-47.

Gene	Alteration	Frequency (%)	Candidate drug
ERBB2	Amplification/Mutations	>10	Her2 inhibitor
FGF3	Amplification	5-10	FGFR inhibitor
FGFR1	Amplification	5-10	FGFR inhibitor
FGFR2	Amplification	1-5	FGFR inhibitor
IGF1R	Amplification	1-5	IGFR inhibitor
EGFR	Amplification	1-5	EGFR inhibitor
PIK3CA	Amplification/Mutations	>10	PI3K inhibitor
PIK3R1	Mutations	1-5	Not Known
PTEN	Mutations/Deletions	5-10	AKT inhibitor
AKT1	Amplification/Mutations	1-5	AKT inhibitor
AKT2	Amplification	1-5	AKT inhibitor
AKT3	Amplification	1-5	AKT inhibitor
INPP4B	Deletions	1-5	AKT inhibitor
CCND1	Amplifications	>10	CDK4 inhibitor
Rb	Mutations/Deletions	5-10	Resistance to CDK4 inhibitor

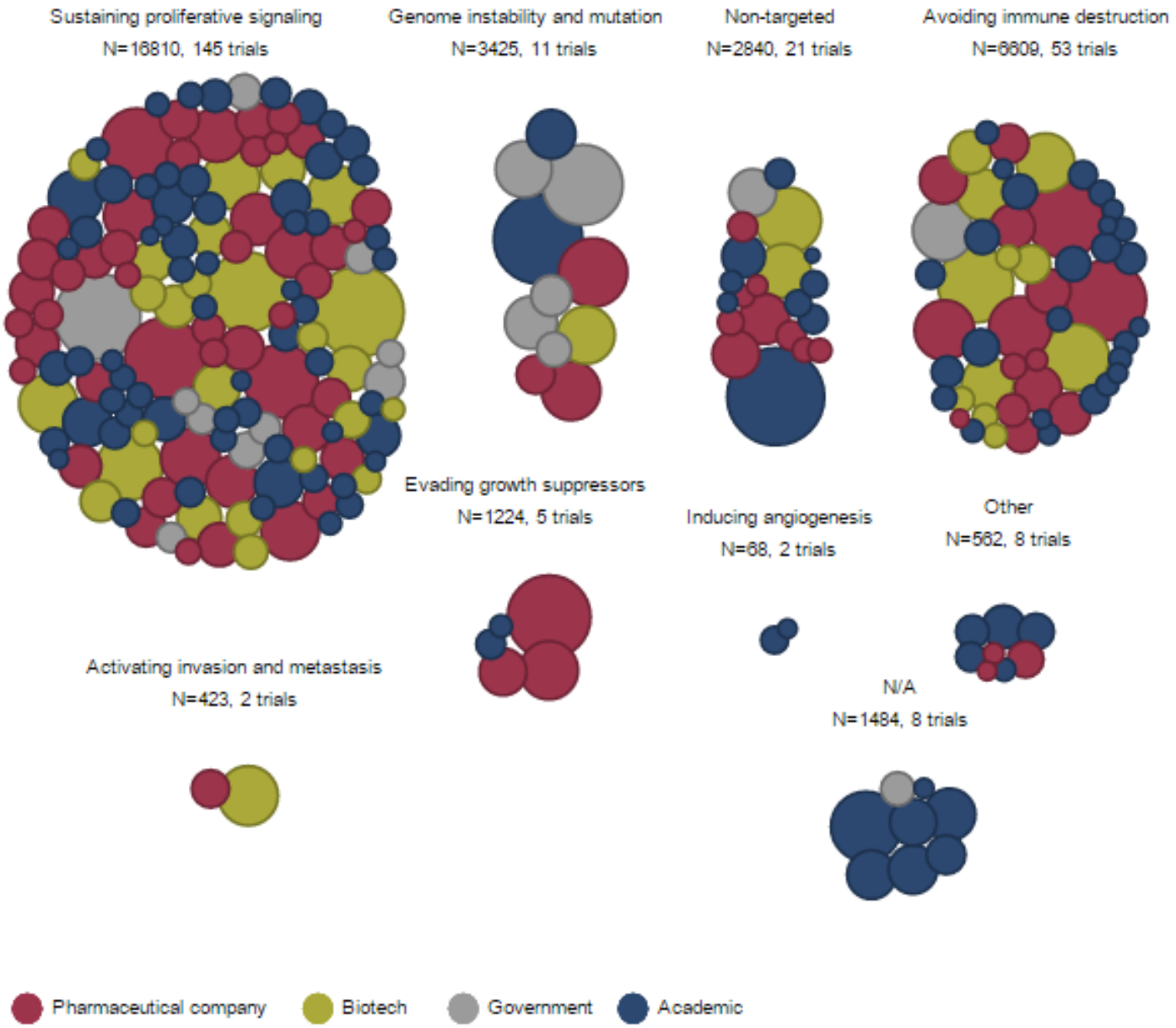
Targetable genomic alterations in breast cancer

All trials on MBC as of Dec 2016



http://www.mbcalliance.org/clinical-trials-in-metastatic-breast-cancer

Trials categorized by hallmarks of cancer



http://www.mbcalliance.org/clinical-trials-in-metastatic-breast-cancer

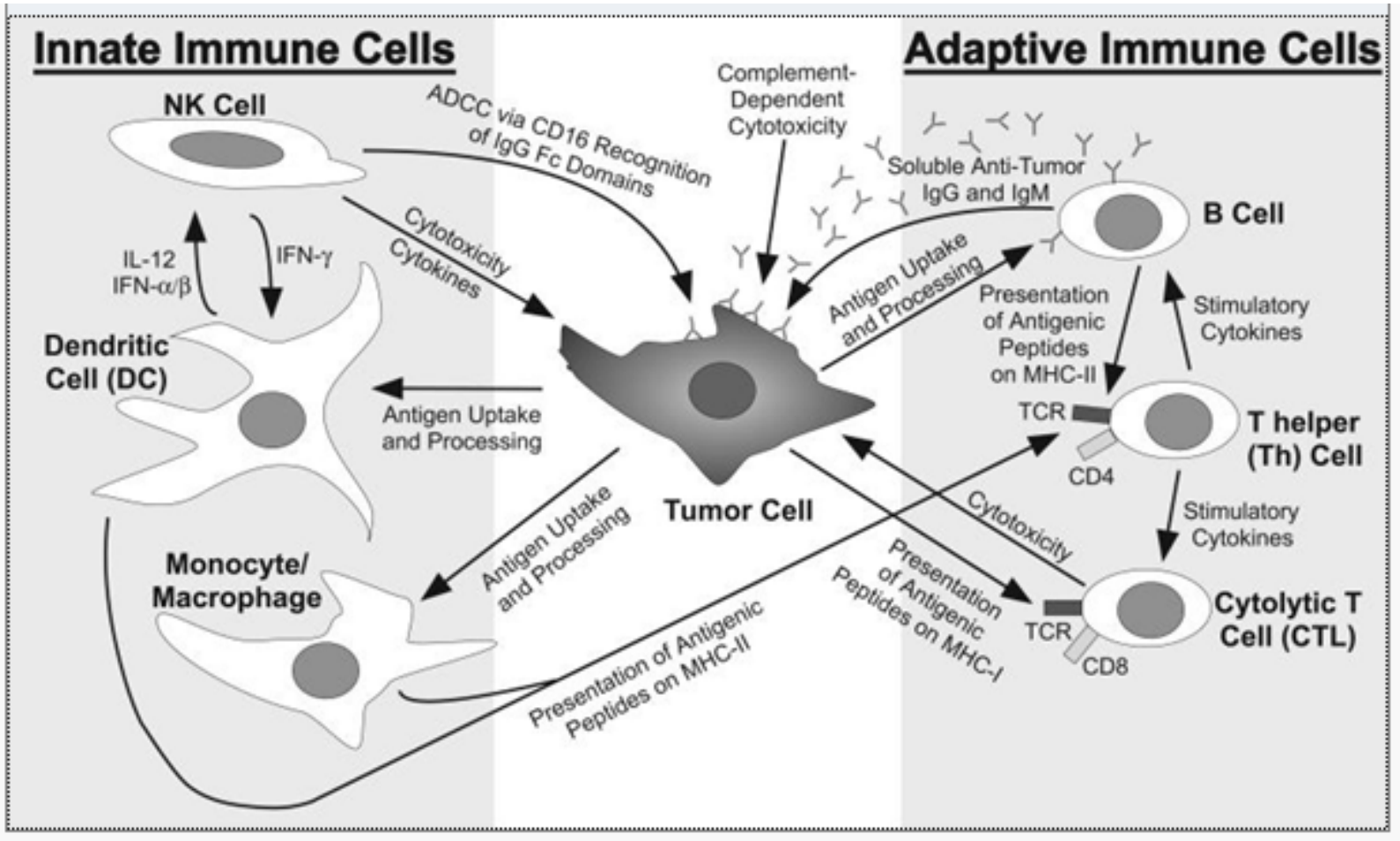
Examples of genotype-driven clinical trials in breast cancer

Trial	Phase (*N*)	Compound	Molecular mechanism of action of experimental drug	Type of disease	Molecular aberration
NCT01219699	I (200)	BYL719	PI3Kα Inhibitor	Solid tumours and ER-positive MBC	*PIK3CA* mutations
NCT01589861 (PIKHER2)	I/II (106)	BKM120	Pan-PI3K Inhibitor	HER2-amplified MBC	PTEN loss and/or *PIK3CA* mutations
NCT01277757	II (40)	MK2206	AKT Inhibitor	MBC	*AKT* mutations and/or *PIK3CA* mutations and/or PTEN loss
NCT01202591 (GLOW)	I/II (900)	AZD4547	FGFR Inhibitor	ER-positive MBC	*FGFR1* amplification
NCT02053636 (FINESSE)	II (123)	Lucitanib	FGFR Inhibitor	ER-positive MBC	*FGFR1* amplification
NCT01670877	II (29)	Neratinib	Irreversible EGFR/HER2 inhibitor	HER2 non-amplified MBC	*ERBB2* mutations

EGFR, epidermal growth factor receptor 2; ER, oestrogen receptor; FGFR, fibroblast growth factor receptor; HER2, human epidermal growth factor receptor 2; MBC, metastatic breast cancer; PI3K, phosphatidylinositide 3-kinase; PTEN, phosphatase and tensin homologue.

Zardavas D, et al. Br J Cancer. 2014 Nov 11;111(10):1881-7.

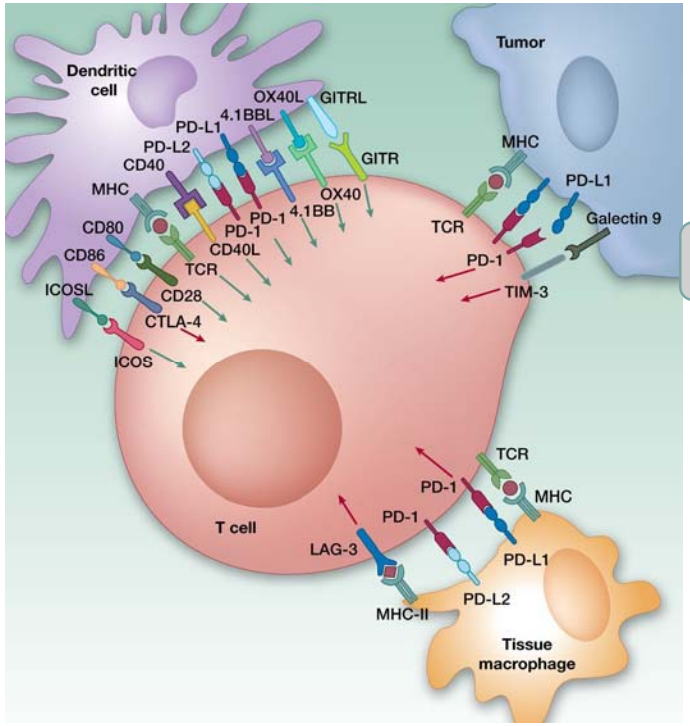
Immune Response to Tumors

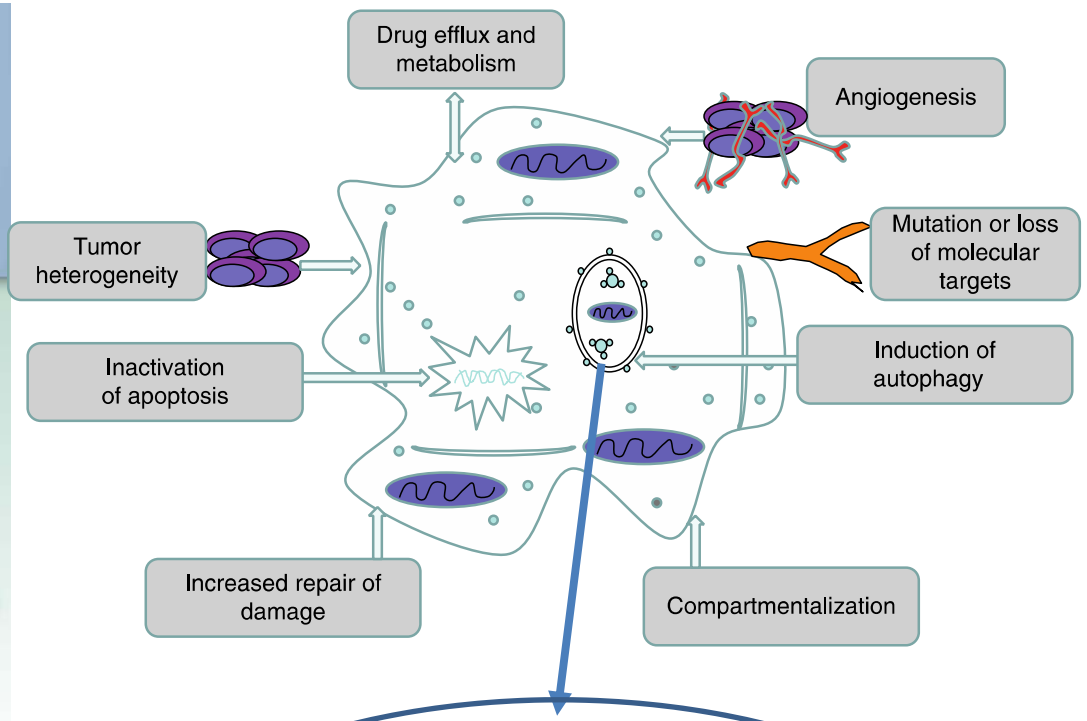


Major cells of the innate and adaptive immune systems and their functions in response to a tumor cell.

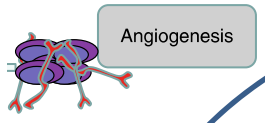
Types of immunotherapy

- Passive immunotherapy:
  - Administration of monoclonal antibodies which target either tumor-specific or over-expressed antigens
- Active immunotherapies:
  - Cytokines- IL-2 / IFNs / TNFα
  - Cancer vaccines
  - Cell-based therapies
  - Tumor-specific CTL
  - Tumor-derived APC
  - DC priming


Tumor
Dendritic cell
OX40L
GITRL
4.1BBL
PD-L1
PD-L2
CD40
GITR
MHC
OX40
4.1BB
PD-1
CD80
CD40L
CD86
TCR
ICOSL
CD28
CTLA-4
ICOS
MHC
PD-L1
Galectin 9
TCR
PD-1
TIM-3
T cell
TCR
PD-1
MHC
PD-1
LAG-3
PD-L1
PD-L2
MHC-II
Tissue macrophage


Drug efflux and metabolism
Angiogenesis
Tumor heterogeneity
Mutation or loss of molecular targets
Inactivation of apoptosis
Induction of autophagy
Increased repair of damage
Compartmentalization

TME


Angiogenesis

Autophagy activation
EGFR
PI3K/ AKT/ mTOR
p53
VEGF
MAPK14/ p38α
miRNA
ERK
AMPK/ AKT/ mTOR
K-Ras
β-catenin
Chemotherapy resistance or insensitivity
Cytotoxicity

13TH Annual Conference of the Organisation for Oncology and Translational Research (OOTR)

Theme : Clinical Trials, Tumor Microenvironment, Biomarkers and Liquid Biopsies in Cancer Research

Date : June 9-10, 2017

Venue : Charles K Kao Auditorium, Science Park, Hong Kong

FREE FOR STUDENTS

Scientific Programme

Biomarkers
Genetic markers for DCIS
Multi-gene expression assays
Liquid biopsy: transformation in cancer care
Translational research in oncology
The role of tumor microenvironment in cancer progression
Implementation in clinical trials
Innovations in biopharmaceuticals

Conference Chairmen

Louis WC Chow, Hong Kong
Executive Director, OOTR

Masakazu Toi, Japan
President, OOTR

Distinguished Guest

Yan Sun, China
Academician, Chinese Academy of Engineering

Confirmed Faculty Members (in alphabetical order)

Bao-San Han, China
Bruce Mann, Australia
Chiun-Sheng Huang, Taiwan
Er-Wei Song, China
Gary MK Tse, Hong Kong
Giuseppe Viale, Italy
Guo-Jun Zhang, China
Hironobu Sasano, Japan
Jun Jiang, China
Li Fu, China
Nadir Arber, Israel
Peter Dubsky, Austria
Seigo Nakamura, Japan
Stefan Glück, USA
Stephen Fox, Australia
Takayuki Ueno, Japan
Won-Shik Han, Korea
Xi-Chun Hu, China
Xiao-Ming Xie, China
Yong-Sheng Wang, China
Jin Zhang, China

REMINDER

Early bird registration: until **April 10, 2017**

CALL FOR ABSTRACTS

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谢谢

Thank You