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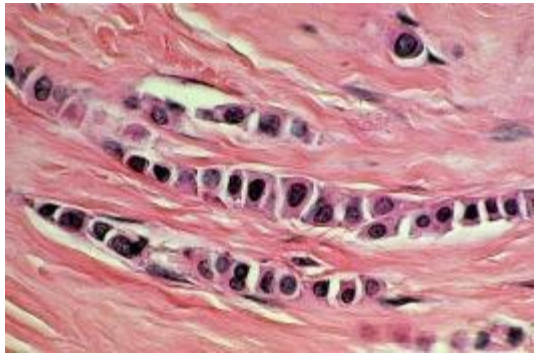
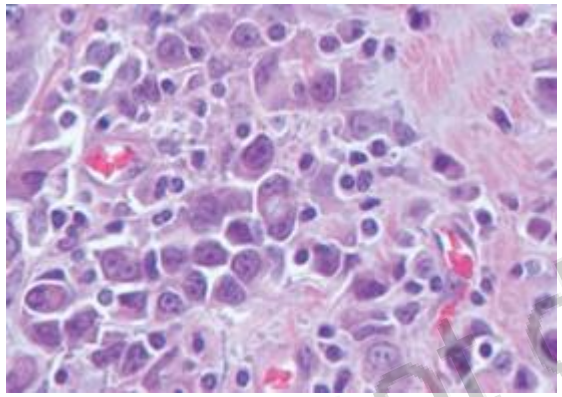
Are all invasive lobular cancers the same?

Expert: **Prof Frederique Penault-Llorca**, Jean Perrin Center, Clermont-Ferrand, France

Discussant: **Dr Simona Volovat**, Grigore T. Popa University of Medicine, Iasi, Romania

Are all invasive lobular cancers the same?





Frédérique Penault-Llorca MD, PhD

COI: Pr Frédérique Penault-Llorca

Personal:	Institutional:	Congress Invitation:
• AbbVie, AstraZeneca, Bayer, BMS, Daiichi Sankyo, Genomic Health, Gilead, GSK, Lilly, MERCK lifa, MSD, Myriad, Nanostring, Novartis, Pfizer, Pierre-Fabre, Roche, Seagen,Tesaro	• Abbvie, Agendia, AstraZeneca, Bayer, BMS, Genomic Health, MSD, Myriad, Nanostring, Roche	• AbbVie, AstraZeneca, BMS, MSD, Novartis, Pfizer, Roche

Objectives

- To understand the specificities of invasive lobular carcinoma
- To understand the heterogeneity of invasive lobular carcinoma in terms of
  - Morphology
  - Biology
  - Therapeutic opportunities

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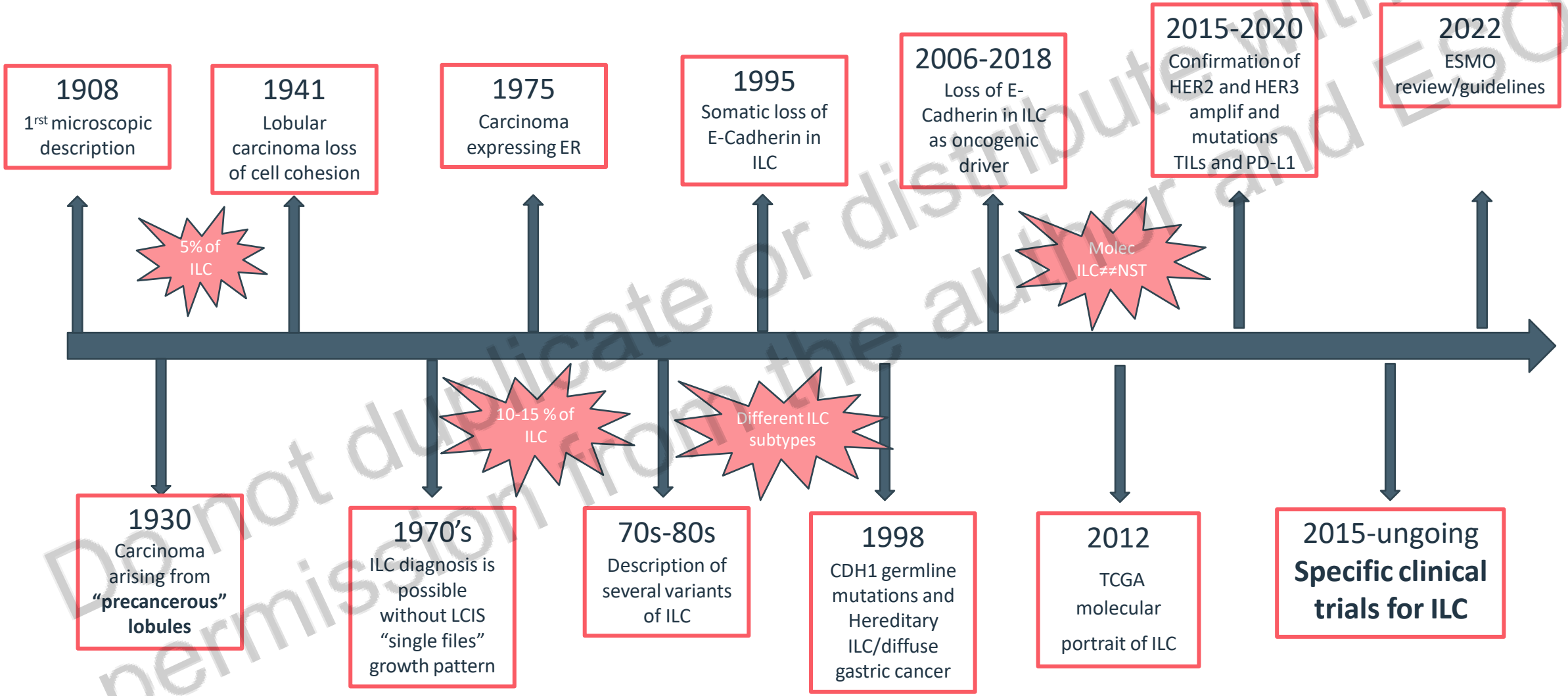
Lobular carcinoma: the most frequent special histopathological type of breast cancer

Evoluting landscape of ILC



Clinicopathological features

- ILC is the **most common special type** of BC [WHO classification of tumors of the breast (5th edition) 2019]
- **10–15%** of all BC cases, less common in Asian populations (2–6%)
- **Different clinical presentation** from BC of no special type (NST BC): associated with
  - higher patient age, higher pT stage, higher nodal stage, lower histological grade, and over-represented in bilateral and primary metastatic BC
  - Lower rates of pathological complete response to neoadjuvant chemotherapy,
  - Higher rate of positive resection margins
  - a **distinct pattern of metastatic dissemination**: metastasis to the digestive tract, ovaries, bones, leptomeninges, orbital soft tissue, and skin less CNS, lung metastasis

  - Higher rate of multiple metastases compared to other BCs

Allison, K.H.; Brogi, E.; Ellis, I.O.; Fox, S.B.; Morris, E.A.; Sahin, A.; Salgado, R.; Sapino, A.; Sasano, H.; Schnitt, S.; et al. WHO Classification of Tumours Editorial Board. Breast Tumours; International Agency for Research on Cancer: Lyon, France, 2019.

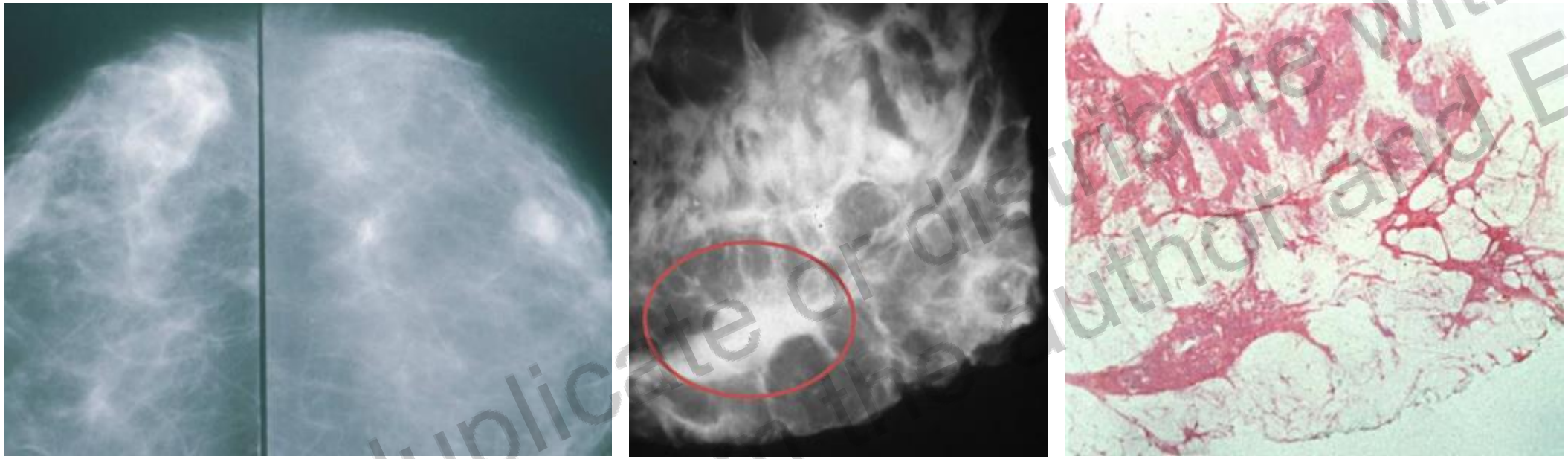
Clinicopathological features

- **Different biology/NST BC**: different mutational characteristics including **E-Cadherin mutations**, different DNA copy numbers, different gene expression profiles, and tumor microenvironment
- **Different subtypes of ILC** with different morphology, prognosis, molecular alterations possibly impacting therapy

Allison, K.H.; Brogi, E.; Ellis, I.O.; Fox, S.B.; Morris, E.A.; Sahin, A.; Salgado, R.; Sapino, A.; Sasano, H.; Schnitt, S.; et al. WHO Classification of Tumours Editorial Board. Breast Tumours; International Agency for Research on Cancer: Lyon, France, 2019.

Clinical features
Histopathology

Clinical presentation



- ILCs are generally palpable, a **high false-negative mammography rate** is possible
- More often **larger tumour, multifocal and bilateral** and with nodal involvement
- **More frequent late relapses**, frequently occurring >10 years after diagnosis

https://doi.org/10.1016/j.annonc.2022.05.006

Risk factors

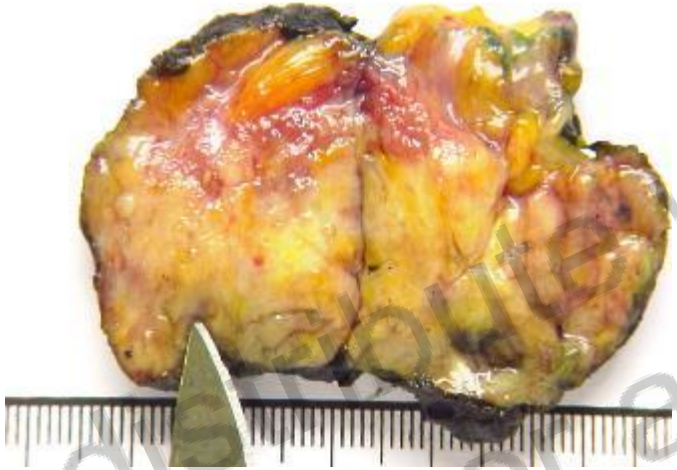
- **Estrogen related**: Early menarche, use of progesterone-based HRT, late age at first live birth and alcohol consumption are more strongly associated with the risk of developing ILC as compared to NST.
- **Obesity** in postmenopausal women does not seem to affect the risk of developing ILC over NST
- **High risk germline mutations: BRCA2 and CDH1** (the gene coding for E-cadherin)
- Moderate risk germline mutations: ATM, CHEK2 and PALB2
- No clinically relevant risk germline BRCA1 mutations
- **Classically absent in males** (exceptional cases in BRCA2 and CDH1 mutation carriers)

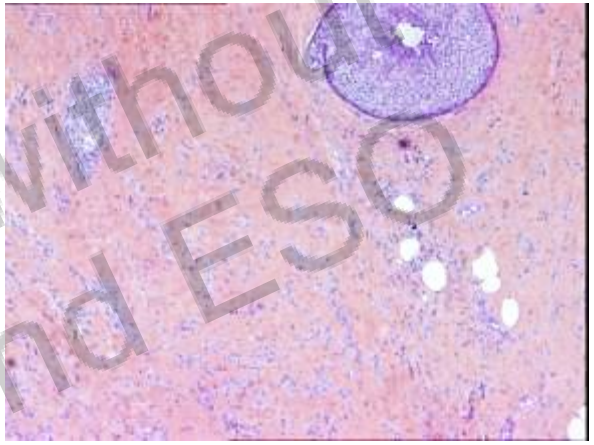
https://doi.org/10.1016/j.annonc.2022.05.006 ; DOI: 10.3389/fonc.2022.891426

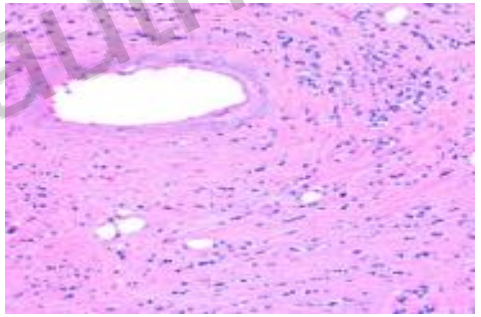
Classical ILC

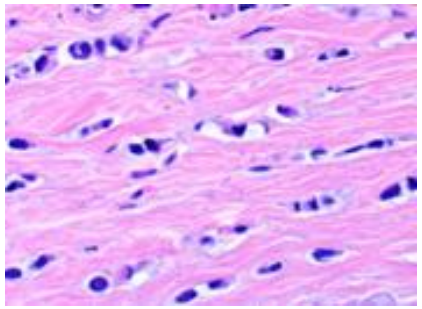
Classical ILC (70%)

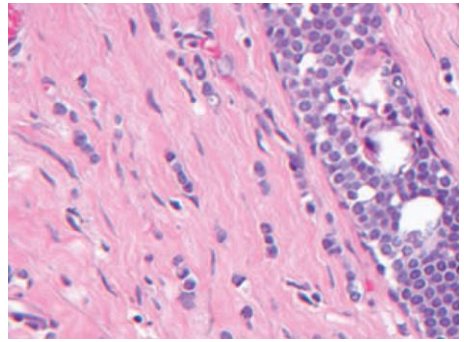
- **Single cell infiltration** and a characteristic targetoid pattern of growth with minimal associated stromal response.
- This pattern of subtle invasion is such that **the size of the tumour often exceeds the imaging findings** and **obtaining clear surgical margins may be challenging**.







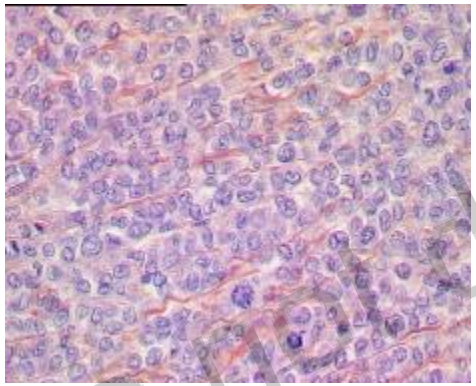




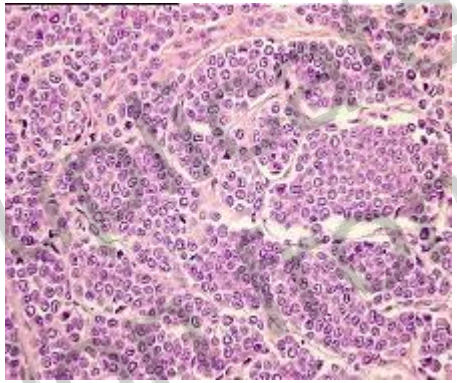
Images courtesy F Penault-LLorca

ILC variants

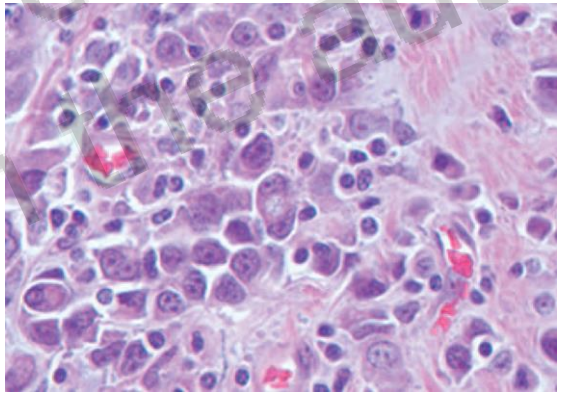
- ILC variants account for up to 70% (~30-40%) of ILC cases
- Common variants: The **WHO** classification of tumors of the breast (5th edition) mentions **four different ILC variants** (solid, alveolar, pleomorphic, tubulolobular)



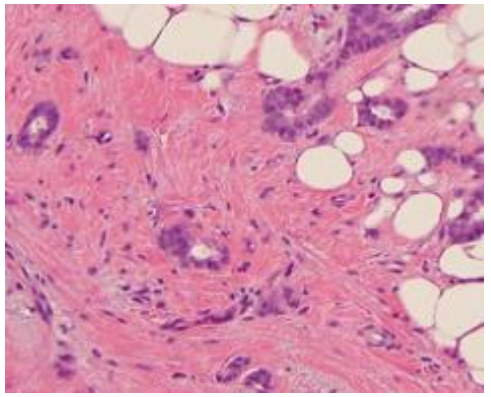
Solid



Alveolar



Pleiomorphic



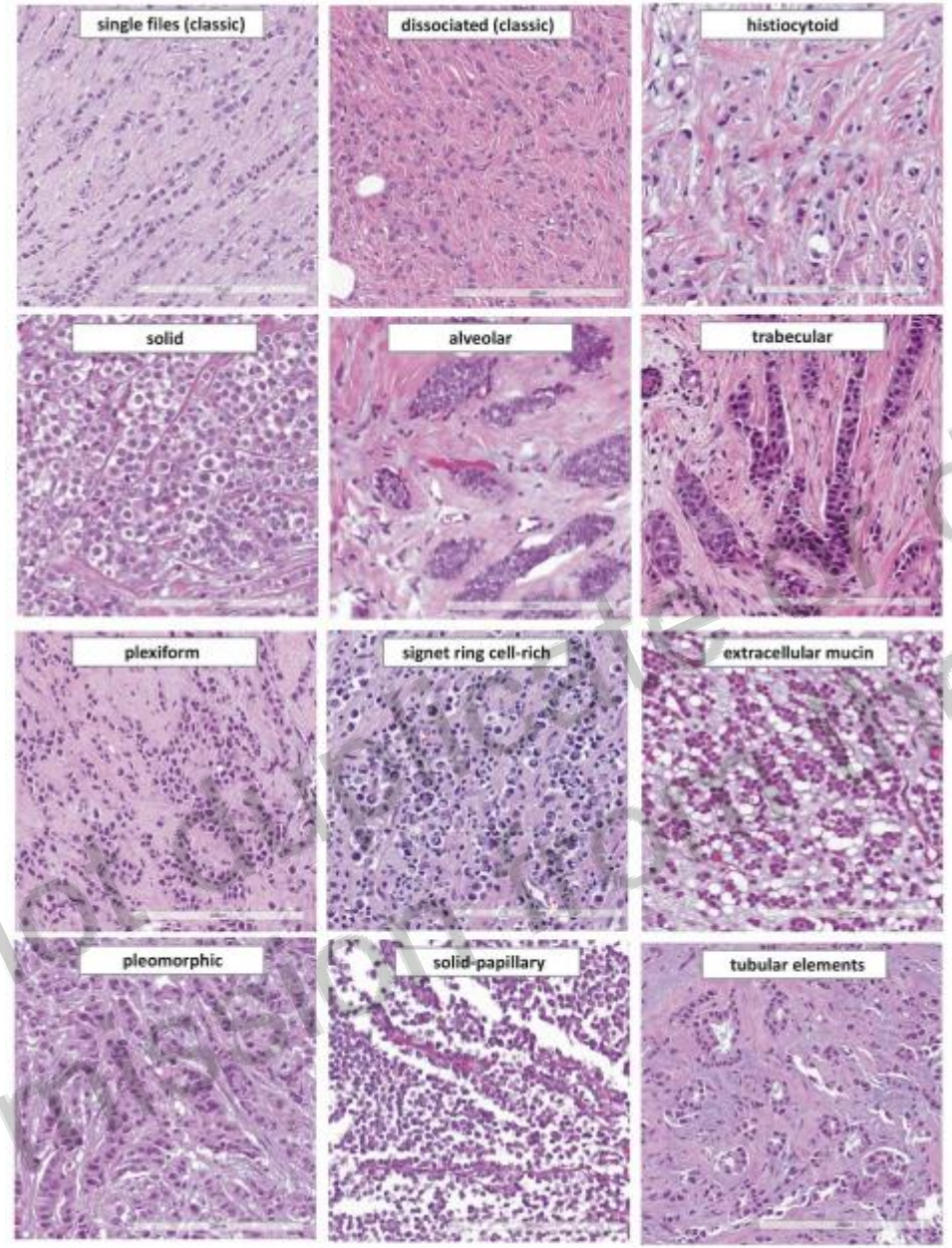
Tubulolobular

Images courtesy F Penault-LLorca

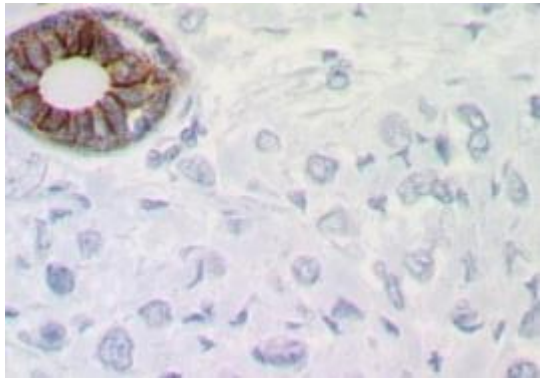
Allison, K.H.; Brogi, E.; Ellis, I.O.; Fox, S.B.; Morris, E.A.; Sahin, A.; Salgado, R.; Sapino, A.; Sasano, H.; Schnitt, S.; et al. WHO Classification of Tumours Editorial Board. Breast Tumours; International Agency for Research on Cancer: Lyon, France, 2019.

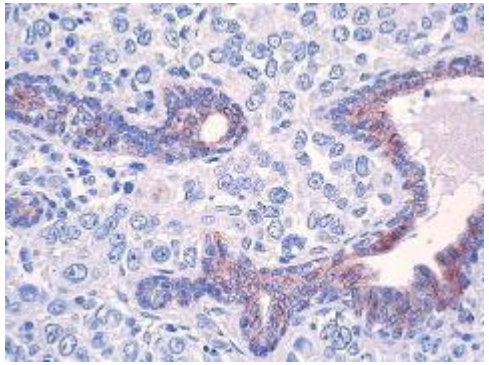
But more ILC variants have been described in the literature

- Some variants are named for their **growth pattern**, such as solid ILC.
- Other variants are named for **cytologic features**, such as pleomorphic ILC.
- Some variants are associated
  - With **distinct molecular alterations** (therapeutic targets)
  - With **different clinical outcome**
- **Common feature: loss or low expression of E-Cadherin**



Images from Cancers 2021, 13, 3695



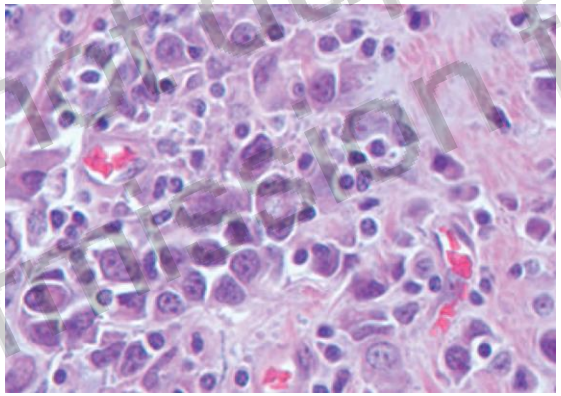


Images courtesy F Penault-LLorca

Some featured subtypes

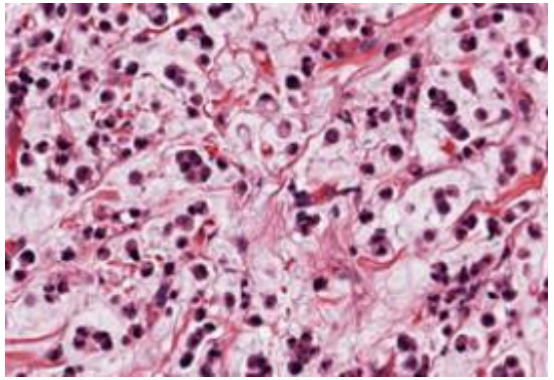
Pleiomorphic ILC (5%)

- **De-differentiated variant**
- **high nuclear grade and SBR3**
- E-cadherin-negative
- **RS frequently>25, N+**
- **11–42%** TP53m**, HER2 amplification, and mutations**
- Survival similar to ILC if corrected for HER2



ILC with Extracellular Mucin

- 80% classic ILC and 20% signet ring cells floating in pools of extracellular mucin (MUC2).
- Both tumor components ➔ E-cadherin-negative
- **N+ ~Over 50% of the cases**
- **12–40%, HER2 amplification**
- **high nuclear grade** is also frequently reported TP53 and PIK3CA mutations associated with relapses

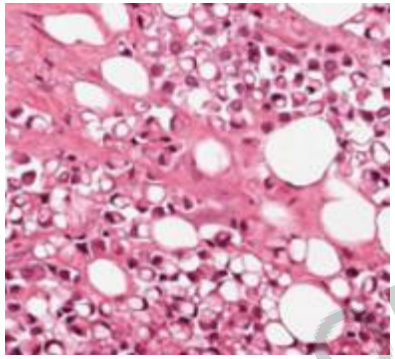


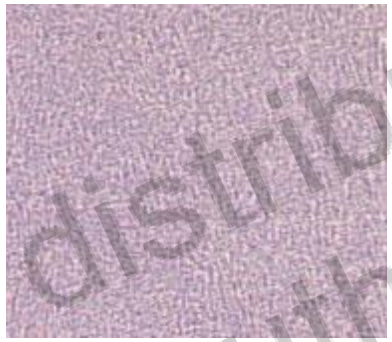
Images courtesy F Penault-LLorca

Some featured subtypes

Histiocytoïd ILC

- Rare
- Frequently **TN, AR+,apocrine like** HER2amp
- Metastasis to the eyelid



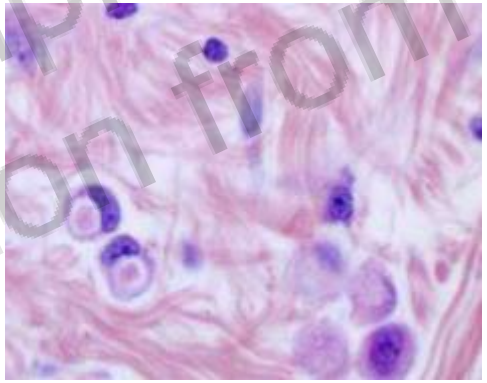


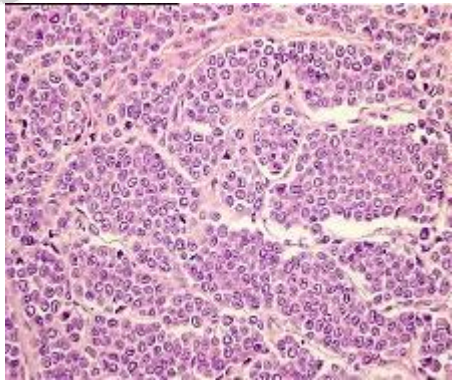
Solid ILC and solid papillary ILC

- Rare poorer prognosis
- Differential diagnosis with lymphoma
- Mutation od ARID1A, TP53 CN gain of ESR1

Signet Ring Cell-Rich ILC

- >50 high power field
- Close to classical ILC



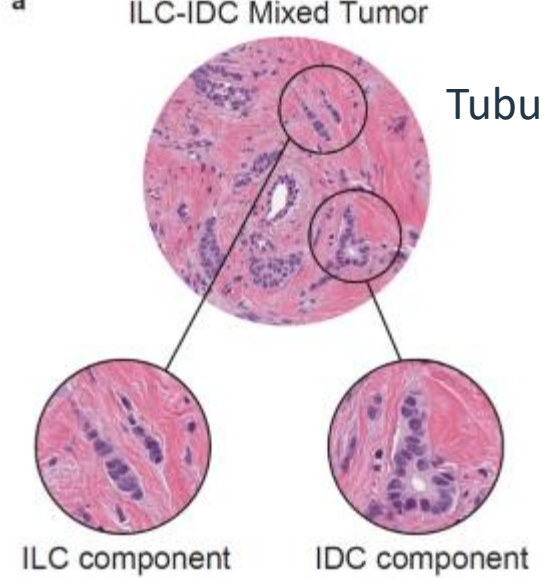


Alveolar ILC

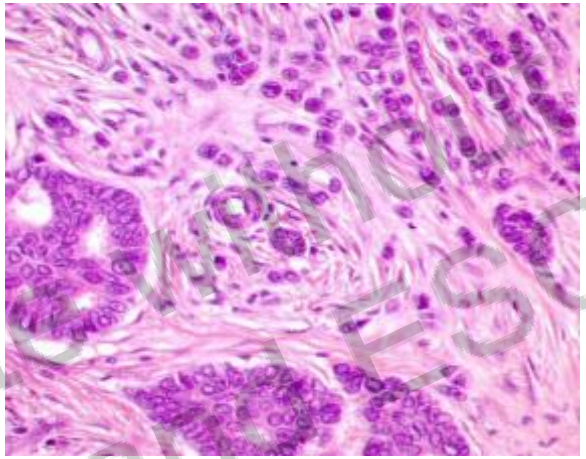
- CN gain on chromosome 11q13.3 (CCND1) and 11q14 (PAK1)

Images courtesy F Penault-LLorca

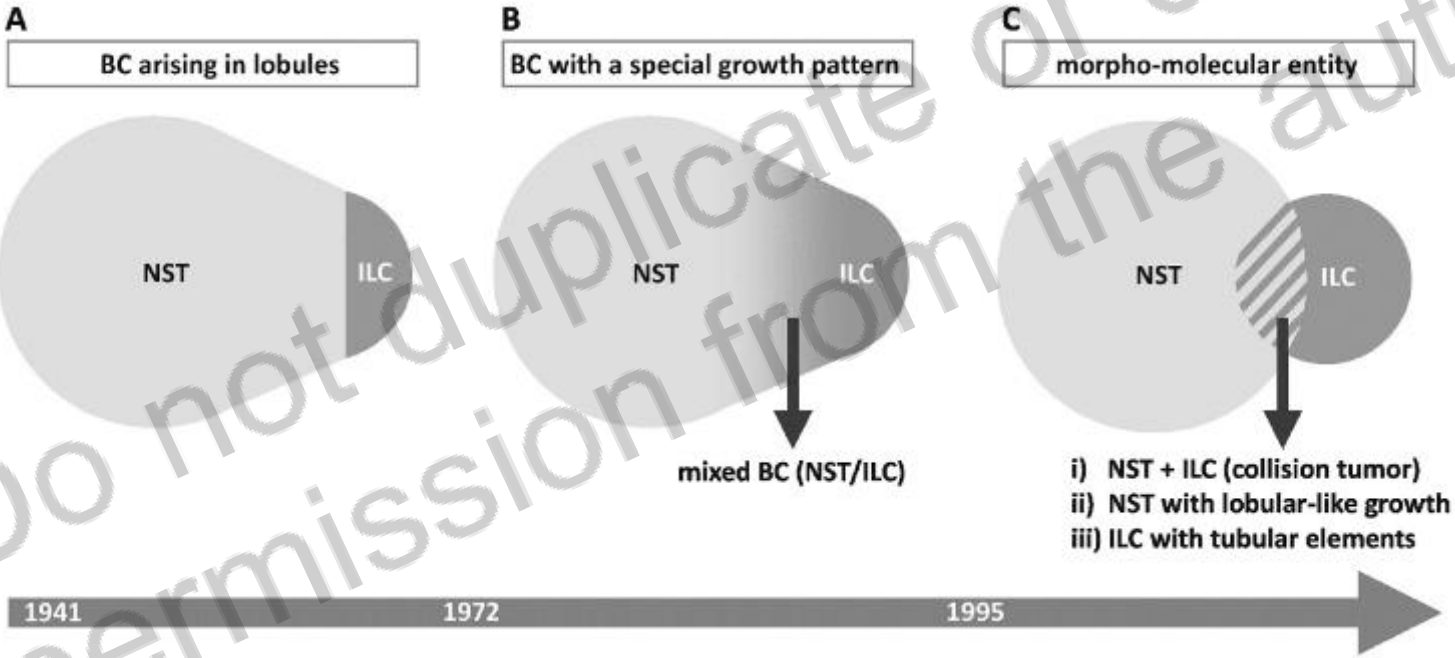
Some featured subtypes

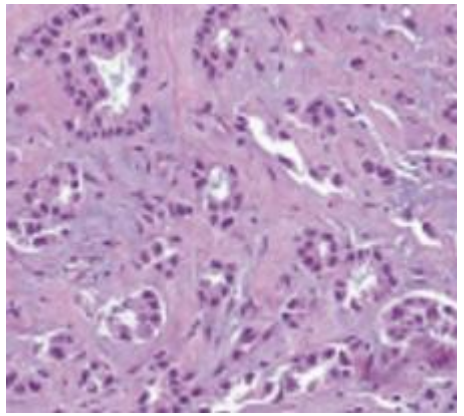


Tubulo lobular (collision tumor?)



E-Cad neg in ILC, +
in Tubular/NST





ILC with tubular elements
(both components are E-Cad neg)

Christgen M et al Cancers 2021, 13, 3695; Ciriello G,et al. Cell 2015;163:506–519.

Images courtesy F Penault-LLorca

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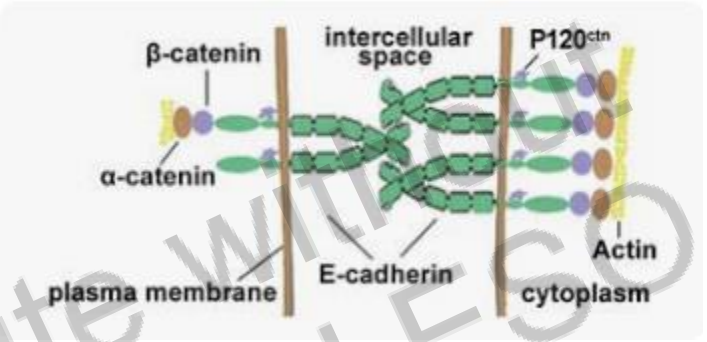
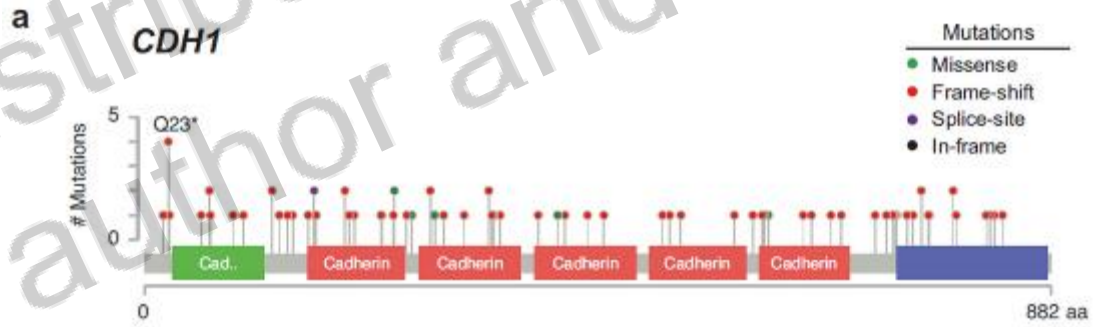
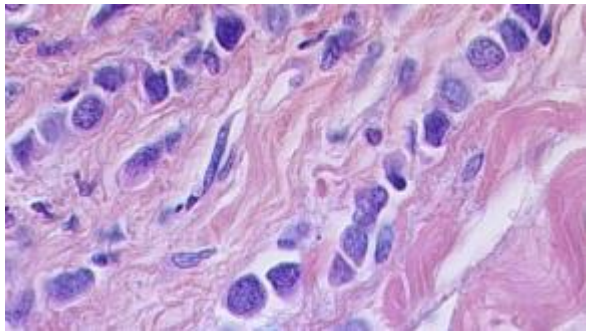
What is shared?

E cadherin down regulation

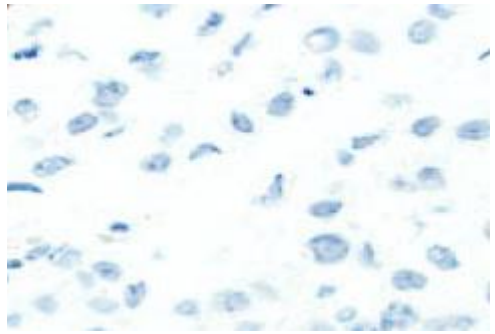


- **Loss or aberrant expression of the cell-to-cell adhesion molecule E-cadherin supports the diagnosis of ILC.**
- Observed in the majority of ILC (>85% of the cases)
- Predominantly caused by **somatic mutations** and LOH, in *CDH1* gene gene mapping in 16q22.1 accompanied by allelic loss of the remaining allele
- In rare cases: **promoter methylation** of CDH1
- Mutational loss of E-cadherin causes **cytosolic translocation** of p120-catenin (p120), multifaceted protein that plays crucial roles in the pathobiology of ILC.
- When the E-cadherin stain is difficult to interpret➔ use of IHC for **beta-catenin** loss of membranous staining and cytoplasmic accumulation of **p120** = lobular phenotype





Classical ILC



E-Cadherin

Cell. 2015 October 8; 163(2): 506–519. doi:10.1016/j.cell.2015.09.033Wang, T., Li, X. (2018). E-Cadherin. In: Choi, S. (eds) Encyclopedia of Signaling Molecules. Springer, Cham. https://doi.org/10.1007/978-3-319-67199-4_101598

Images courtesy F Penault-LLorca

Molecular signatures➔ need for specific signatures

Test	Ref.	Cohort		Results	Study conclusion
GGI/ MapQuantDx™	[89]	166 ILC		Test outperformed grade	Prognostic value in ILC
MammaPrint	[90]	217 ILC		Independent value of MammaPrint, specifically in lymph node-negative ILC	
	[91]	487 ILC (255 CILC)		10.2% CILC and 22.8% of ILC variants were high risk	Prognostic value in ILC
OncotypeDx	[55]	353 ILC		20% low-, 72% intermediate-, and 8% high-risk score	ILC more likely low/int score but 5-year DMFS equivalent to non-ILC
	[92]	30 ILC		All ILC low or int risk	Questions utility in ILC; more data required
	[93]	97 ILC		1% of ILC (non-pleomorphic) record high-risk RS	Questions utility in ILC; more data required
	[94]	102 ILC		Different RS distribution in ILC v IBC-NST	More data required
	[95]	59 ILC		50% ILC in low risk	More data required
	[96]	9037 ILC	SEER data	38.1% ILC intermediate risk; 2.4% high risk	More data required
	[97]	7316 ILC	SEER data	72% ILC in intermediate-risk group; 8% high risk	Adjuvant Ctx did not confer survival benefit to int or high risk; note LN+ cases included
	[98]	49,819 ILC	Genomic Health clinical lab 2004–2017	63.9% ILC in low risk, 33.6 in intermediate, 2.5% in high risk	Classic ILCs have lower average RS (16.3) compared to IDC (18.4) and ILC variants (18.2), and lower rate of tumours with high scores (2.5% vs. 10.7% vs. 8.4%, respectively)
Prosigna	[99]	341 ILC	Danish Breast Cancer Group	ILC had poorer 10-year DR rates than ROR matched IDC	Prognostic value in ILC
EndoPredict/ EPClin	[100]	470 ILC	TransATAC and ABCSG-6/8	63.4% were low EPClin risk group (a 10-year DR risk of 4.8%) compared to 172 (36.6%) women in the high-risk group (110-year DR risk of 26.6%)	Significant prognostic value; Ctx in low-risk group not indicated

GGI, Mammaprint OK

OncotypeDx utility or ILC ?

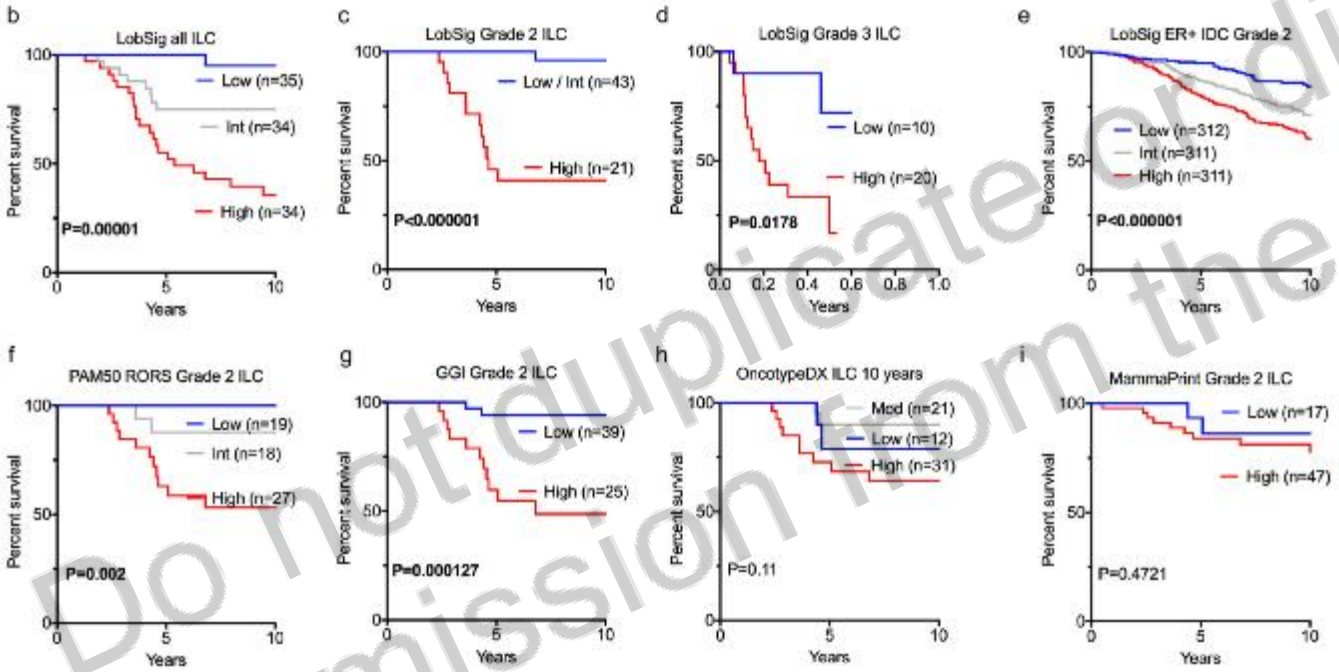
Prosigna Endopredict OK

McCart Reed et al. Breast Cancer Research (2021) 23:6

ARTICLE OPEN

LobSig is a multigene predictor of outcome in invasive lobular carcinoma

Amy E. McCart Reed[1], Samir Lal[1,5], Jamie R. Kutasovic[1], Leesa Wockner[2], Alan Robertson[3], Xavier M. de Luca[1], Priyakshi Kalita-de Croft[1], Andrew J. Dalley[1], Craig P. Coorey[1], Luyu Kuo[1], Kaltin Ferguson[1], Colleen Niland[1], Gregory Miller[1,4], Julie Johnson[1], Lynne E. Reid[1], Renique Males[1], Jodi M. Saunus[1], Georgia Chenevix-Trench[2], Lachlan Coin[3], Sunil R. Lakhani[1,4] and Peter T. Simpson[1]



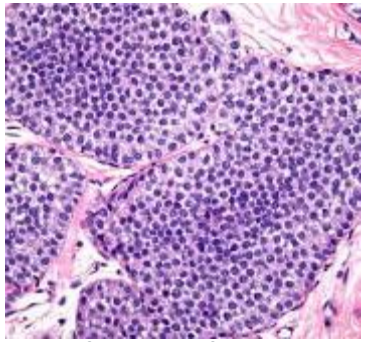
- **LobSig high tumors** were enriched for <u>mutations</u> in **ERBB3** (P = 0.00007), **ERBB2** (P =0.0002), **BIRC6** (P =0.005), **AKT1** mutations (P=0.02), **ROS1** (P < 0.01); <u>amplifications</u> of **PRMT2** (P= 7.329e–08), **S100B** (P= 7.33e–08) and **DIP2A** (P =7.99e–07; 21q22.3); and for <u>deletions</u> of CTCF (16q22.1; P= 8.41e–11), C17ORF39 (17p11.2; P= 4.597e–09) and **ARID1A** (1p36.11; P =8.045e–06).
- **The LobSig low** tumors showed a relatively quiet genome.

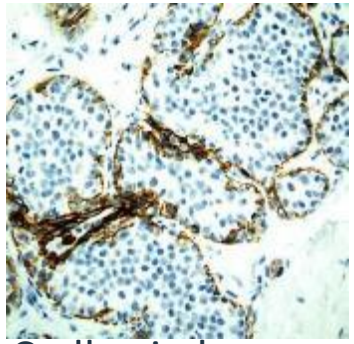
npj Breast Cancer (2019) 18

What differs?

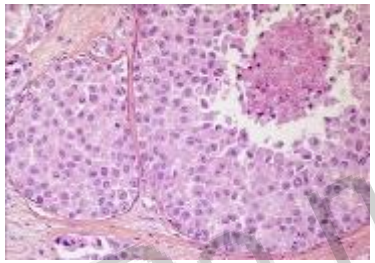
Carcinogenesis: 3 subtypes of LCIS with ≠ molecular alterations

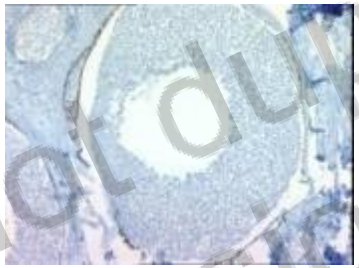
WHO 2019



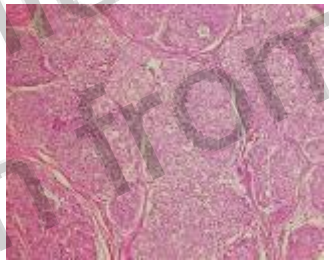


Classical-CLCIS E-Cadherin loss





Pleomorphic (PLCIS),E-Cadherin loss



Florid (FLCIS)

Both **a risk factor** for and a **non-obligate precursor** of IBC.

PLCIS may be RE, PR- and HER2+

PCLIS and FLCIS on biopsy ➔ surgical verification

McCart Reed et al. Breast Cancer Research (2021) 23:6 https://doi.org/10.1186/s13058-020-01384-6; Vincent-Salomon, Lakhani. Modern Pathology (2021) 34:8–14 https://doi.org/10.1038/s41379-020-00689-3

Histopathological differences between classical ILC and variants

Classical ILC

- ER+, PR+, HER2 non amplified – lumA
- Grade 2 tumors
- HER2 amplifications ~0%
- HER2 mutations ~6%
- Absent or low TILS

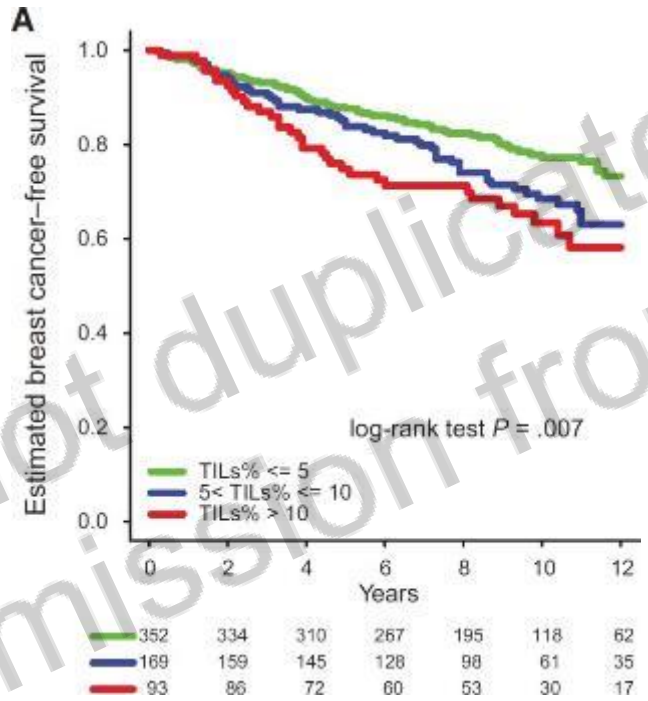
Rare types - Pleiomorphic

- 2-9% of TN-ILC (express AR, *HER2* & *HER3* mutations and *ESRRA*m)
- Grade 3 tumors in pleiomorphic or other non classical (~12%)
- HER2 amplifications up to 25% (incl Apocrine variant)
- HER2 mutations ~15%
- TILs high (>15%) younger age poor Prognostic

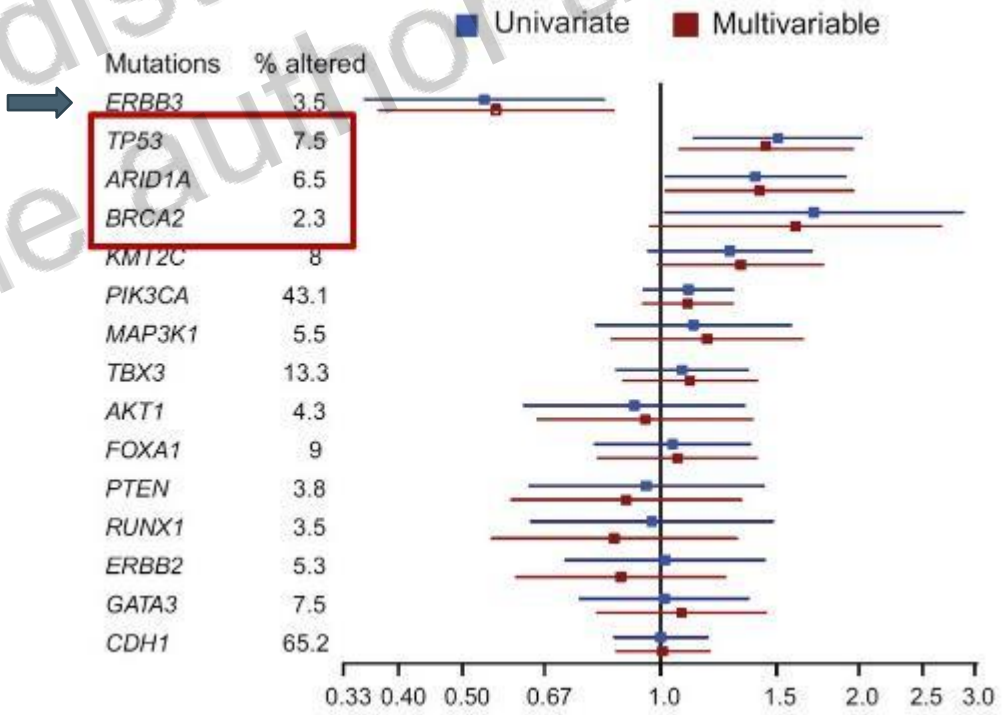
Cancers 2021, 13, 3695

TILs in ILC

TILs in ILC= poor prognosis



Fold change in TILS and mutation status



Desmedt et al doi: 10.1093/jnci/djx268

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Focus on molecular targets

Specific/frequent molecular alterations

Lobular carcinomas (10-15%) and their precursors (lobular neoplasia): *CDH1* mutation (located on 16q), ➔the pathognomonic **loss of E-Cadherin** expression (adhesion protein):aspect of non cohesive cells

➔ **PI3K alterations >50%** (with Akt/mTOR in 45%)

➔ **AKT1**, FOXA1, **HER2, HER3**, PTEN and TBX3 mutations in ILC>IDC

➔HER2 and AKT1 mutations associated with increased risk of early relapse

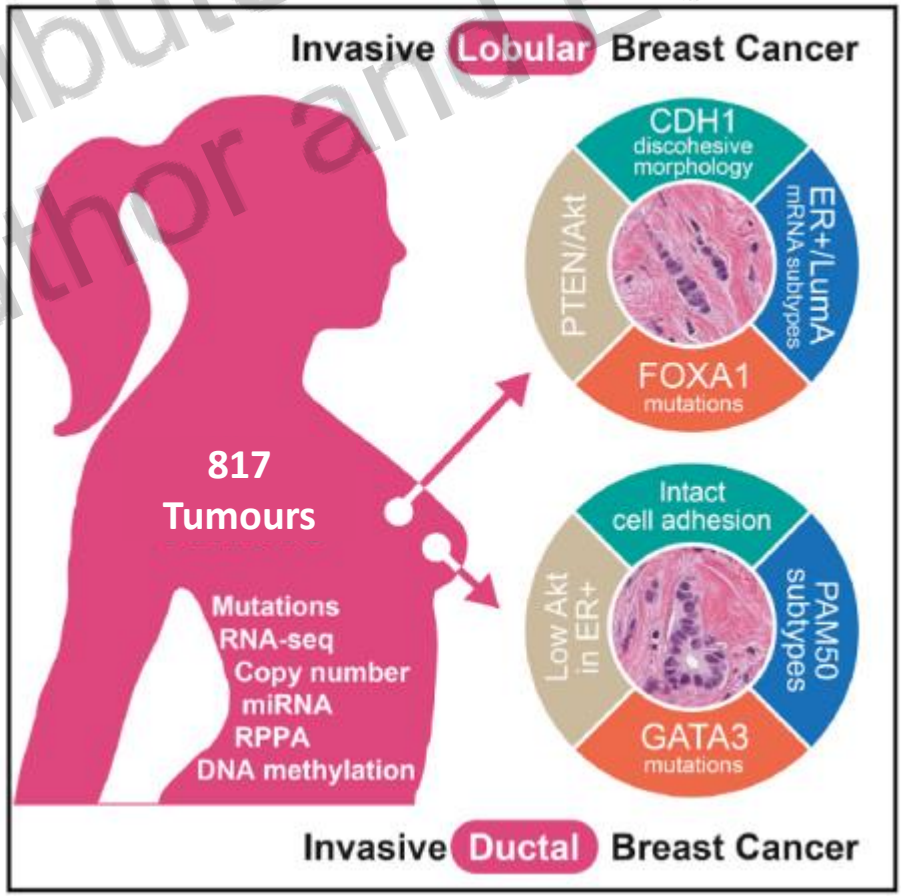
➔Histologic subtype–specific associations: **ESR1 gains in solid subtype**, **HER2 mutations in mixed non classic**, and TP53 mutations in both.

Ciriello G, et al. Cell. 2015;163:506-19.

Comprehensive molecular portraits of invasive lobular BC

Giovanni Ciriello,[1,2,23] Michael L. Gatza,[3,4,23] Andrew H. Beck,[5] Matthew D. Wilkerson,[6] Suhn K. Rhie,[7] Alessandro Pastore,[2] Hailei Zhang,[8] Michael McLellan,[9] Christina Yau,[10] Cyriac Kandoth,[11] Reanne Bowlby,[12] Hui Shen,[13] Sikander Hayat,[2] Robert Fieldhouse,[2] Susan C. Lester,[5] Gary M.K. Tse,[14] Rachel E. Factor,[15] Laura C. Collins,[5] Kimberly H. Allison,[16] Yunn-Yi Chen,[18] Kristin Jensen,[16,17] Nicole B. Johnson,[5] Steffi Oesterreich,[19] Gordon B. Mills,[20] Andrew D. Cherniack,[8] Gordon Robertson,[12] Christopher Benz,[10] Chris Sander,[2] Peter W. Laird,[13] Katherine A. Hoadley,[3] Tari A. King,[21] TCGA Research Network,[22] and Charles M. Perou[3,*]

- Invasive lobular carcinoma (ILC) is a **clinically** and **molecularly** distinct disease
- ILCs show **CDH1 and PTEN** loss, AKT activation, and mutations in *TBX3* and *FOXA1*
- Proliferation and **immune-related** gene expression signatures define **3 ILC subtypes**
- Genetic features classify mixed tumours into lobular-like and ductal-like subgroups



Ciriello G, et al. Cell. 2015;163:506-19.

ESCAT alterations in primary and metastatic ILC

Table 1. Actionability of the genomic alterations in primary and metastatic ILC as per ESCAT[53,206]

	Readiness of use in clinical practice	ESCAT alterations in breast cancer	% Primary ILC (% primary NST)	% Metastatic ILC (% metastatic NST)
Tier I	Targets ready for implementation in routine clinical decisions	*ERBB2/HER2* amplification	7.4 (20.6)[25]	6.8 (11.4)[55]
		Germline *BRCA1* mutations	0.3 (2.3)[25,55]	
		Germline *BRCA2* mutations	2.2 (2.4)[25]	
		NTRK1-3 fusions	0 (0)	0.6 (0)[33]
		PIK3CA mutations	43-48 (33.5)[31]	38-47.2 (33.1)[42,55]
		MSI	NA	NA
		High TMB (>10 mutations/Mb)	4.7 (NA)[54]	16 (5)[58]
Tier II	Investigational targets likely to define patients who benefit from a targeted drug, but additional data needed	*AKT1* mutations	1.6-4 (3.1)[31,33]	7.5-10 (6.4)[42,55]
		ERBB2/HER2 mutations	3.9-5 (1.4)[31,33]	14.3-15 (4.6)[42,55]
		ESR1 mutations	0-0.8 (0.8)[31,33]	15.5-18 (15.3)[42,55]
		PTEN loss	13.4 (11.2)[33]	14.3-15 (8.4)[42]
Tier III	Clinical benefit previously demonstrated in other tumour type or for similar molecular targets	Somatic *BRCA1* mutations	0 (2.9)[31,33]	1.2 (1.9)[55]
		Somatic *BRCA2* mutations	0.8-2 (2.5)[31,33]	6.2 (3.5)[55]
		MDM2 amplifications	2-2.4 (4.7)[31,33]	2-6.2 (4.2)[42,55]
		NF1 mutations	1-3.9 (2.9)[31,33]	7-7.5 (5.8)[42,55]
		ERBB3/HER3 mutations	0.8-4 (2.3)[31]	0-2.5 (1.9)[42,55]

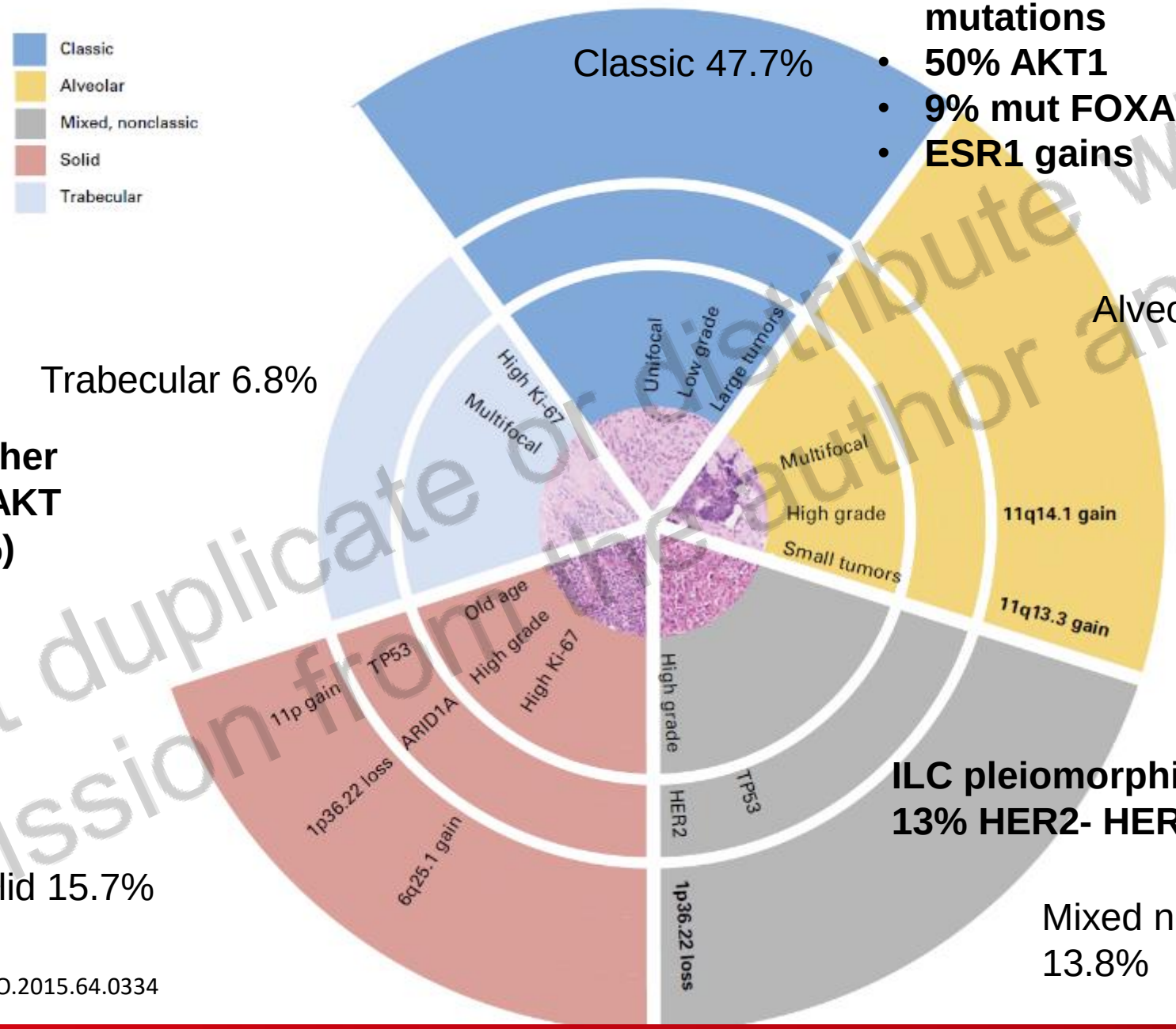
https://doi.org/10.1016/j.annonc.2022.05.006

ESCAT alterations in primary and metastatic ILC

Tier IV	Preclinical evidence of actionability	*ARID1A* mutations	5.5-6.3 (3.7)[31]	8.7-15 (6.1)[42,55]
		ARID1B mutations	0.2-0.8 (2.1)[31,33]	2.5 (2.8)[55]
		Germline *ATR* mutations		NA
		Germline *ATM* mutations		1.03 (1.1)[25]
		Germline *PALB2* mutations		0.37 (1.5)[25]
		CDH1 mutations	63-65 (2)[31,33]	69-75.8 (2.1)[42,55]
		Germline *CDH1* mutations		0.54 (0.04)[25]
		IGF1R mutations	0-1.6 (0.8)[31,33]	0-13 (2.1)[42,55]
		INPP4B loss	0.8 (0.8)[33]	0.6 (0.3)
		MAP3K1 mutations	5.3-5.5 (8.2)[31,33]	7.5-17 (7)[42,55]
		MAP2K4 mutations	1.2-1.4 (4.9)[31,33]	2-4.3 (3.7)[42,55]
		MT4 mutations		NA
		MYC amplifications	4.7-6.3 (26.8)[31,33]	3.7-15 (11.6)[42,55]
		PIK3R1 mutations	0-1 (1.8)[33]	1.9 (2.7)[55]
		RUNX1 mutations/deletions	4-10.2 (2.5)[33]	5 (2.7)[55]
		CBFB mutations/deletions	3.2 (3.3)[33]	4.3 (3.5)[55]
		SF3B1 mutations	1.5-3.1 (2.5)[33]	3.1 (1.7)[55]
		TP53 mutations	7.3-7.9 (44)[31,33]	12-19.9 (42.9)[42,55]
Tier X	Lack of evidence of actionability	*FGFR1* amplification	9.4-25 (13.9)[31,33]	14.3-15 (14.6)[42,55]
		CCND1 amplification	17.3-38 (16.2)[31,33]	19.9-24 (22.7)[42,55]
		ESR1 amplification	0.8-10 (3.5)[33,207]	1.0 (3.2)[55]
		FGFR4 mutations	0 (0.2)[33]	2.5 (1.0)[55]

https://doi.org/10.1016/j.annonc.2022.05.006

- **ERBB2 & ERBB3 8.5% mutations**
- **50% AKT1**
- **9% mut FOXA1**
- **ESR1 gains**



ILC lumA: higher activation of AKT pathway (45%)

ILC pleiomorphic 15% HER2 amplif 13% HER2- HER3 mutations

Desmedt et al 2015 DOI: 10.1200/JCO.2015.64.0334

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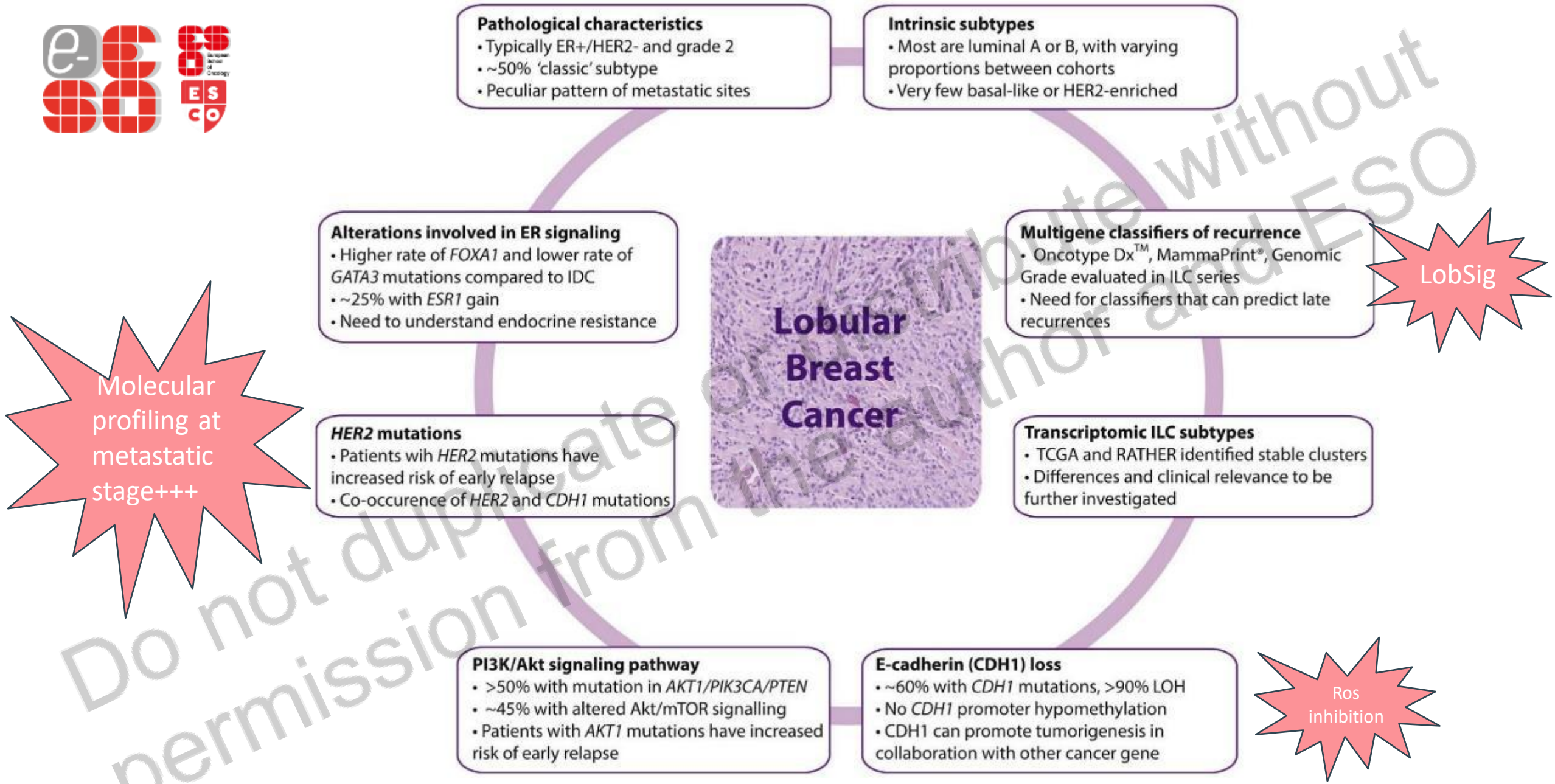
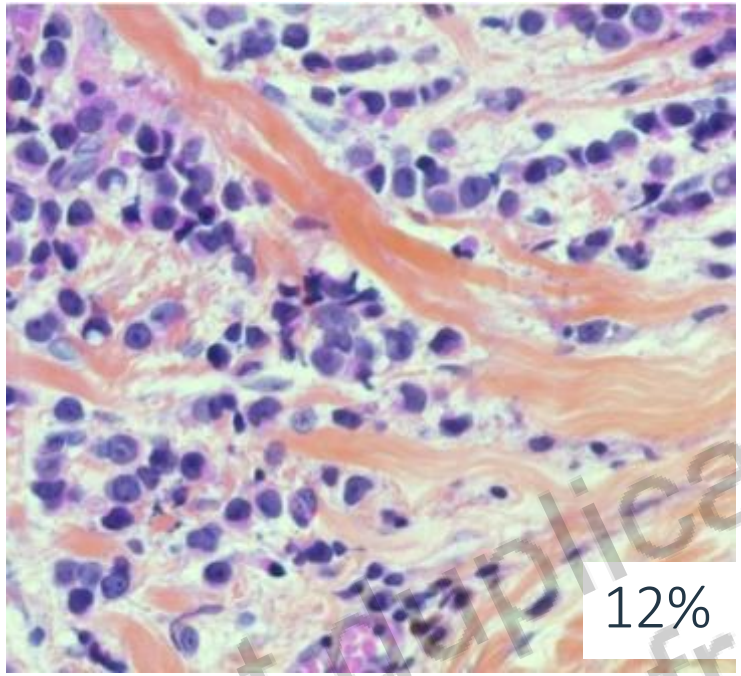
In conclusion



Desmedt et al https://doi.org/10.1016/j.semcancer.2017.03.007

What is relevant for clinical practice?

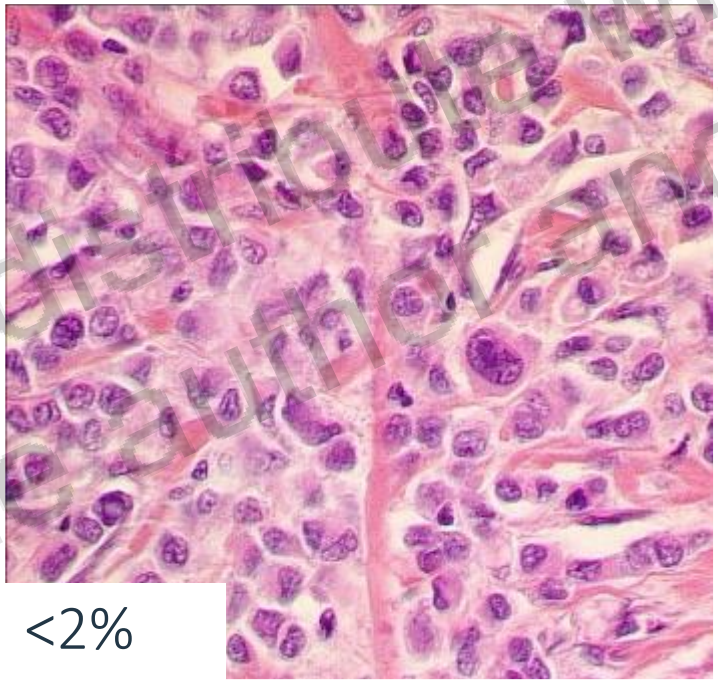
Classical



12%

HER2 amplification ~0%

Pleiomorphic and non classical high grade



<2%

HER2 amplification ~20-25%
POOR PROGNOSIS

- *CDH1* mutations ~85%
- *HER2, HER3* mutations ~8.5%
- *PIK3CA, AKT1, PTEN* mutations ~50%

https://doi.org/10.1016/j.annonc.2022.05.006

Lobular carcinoma in summary

- E-cadherin Inactivation in 95% of cases
- ER+ > 90% of cases
- Low proliferation

→ Low chemosensitivity
ET, CDK4/6i sensitivity

- HER2 score 3+ < 5% of cases
- *HER2* Mutations :
  - 6% classical ILC
  - 15% ILC high grade

→ Targeted therapies such as anti HER2 TKIs?

- ***PIK3CA* Mutations in 48% of the cases**

→ mTOR inhibitor
PIK3CA inhibitor

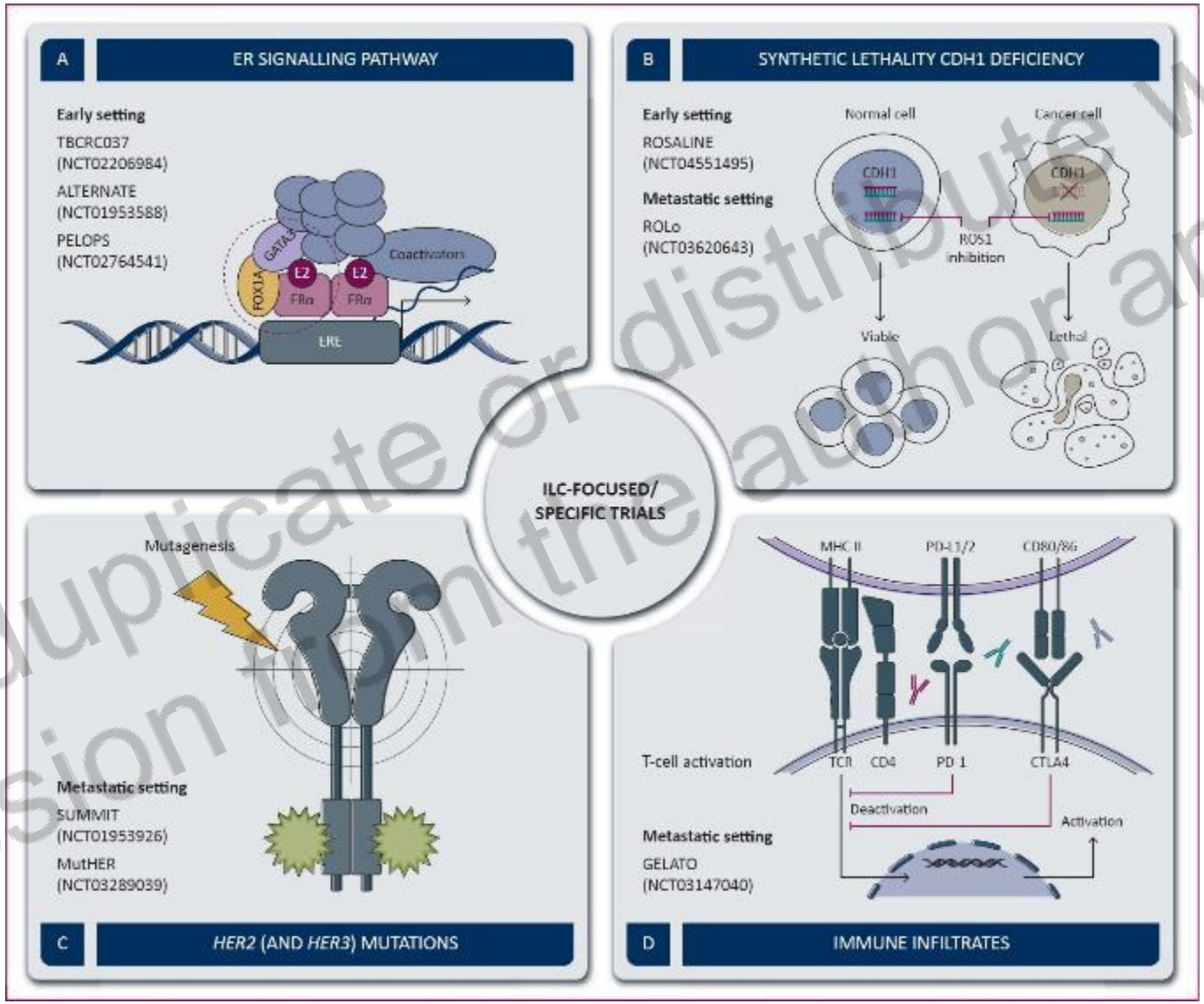
- Mutations *TP53*, *GATA3*, *FOXA1*, *RUNX1* ~ 5 -10% of the cases PTEN/AKT pathway activation mutually exclusive with *PIK3CA* mutations.
- **3 to 5 transcriptomic groups have been identified**
  - « reactive-like » (good prognostic), « Immune-related » &« proliferative »

  or « immune-related » & « hormone – related »

Michaut et al Scientific report 2016
Ciriello *et al* Cell 2015
Deniziaut *et al* Oncotarget 2016

TCGA. Nature 2012.
Huang-Chun *et al*. Breast Cancer Res Treat 2015

ILC-focused clinical trials



https://doi.org/10.1016/j.annonc.2022.05.006

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REVIEW

Current and future diagnostic and treatment strategies for patients with invasive lobular breast cancer

K. Van Baelen[1,2], T. Geukens[1,2], M. Maetens[1], V. Tjan-Heijnen[4], C. J. Lord[5], S. Linn[6,7,8], F.-C. Bidard[9], F. Richard[1], W. W. Yang[5], R. E. Steele[5], S. J. Pettitt[5], C. Van Ongeval[10], M. De Schepper[1,11], E. Isnaldi[1], I. Nevelsteen[12], A. Smeets[12], K. Punie[5], L. Voorwerk[1,13,14], H. Wildiers[3], G. Floris[11], A. Vincent-Salomon[15], P. W. B. Derksen[6], P. Neven[2], E. Senkus[16†], E. Sawyer[17†], M. Kok[18,19‡] & C. Desmedt[1*]

Seminars in Cancer Biology 44 (2017) 98–105
Contents lists available at ScienceDirect
Seminars in Cancer Biology
journal homepage: www.elsevier.com/locate/semcancer
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Review

Transcriptomic and genomic features of invasive lobular breast cancer

Christine Desmedt[a,*], Gabriele Zoppoli[b], Christos Sotiriou[a], Roberto Salgado[a,c]
[a] Breast Cancer Translational Research Laboratory J. C. Heuson, Université Libre de Bruxelles, Institut Jules Bordet, 1000 Brussels, Belgium
[b] Department of Internal Medicine (DIMI), University of Genoa and Istituto di Ricovero e Cura a Carattere Scientifico San Martino-National Cancer Institute, Genoa, Italy
[c] Department of Pathology/TCRU, Ziekenhuis Netwerk Antwerpen, Antwerp, Belgium

cancers
MDPI

Review

Lobular Breast Cancer: Histomorphology and Different Concepts of a Special Spectrum of Tumors

Matthias Christgen [1,*], Gábor Cserni [2,3], Giuseppe Floris [4], Caterina Marchio [5,6], Lounes Djerroudi [7], Hans Kreipe [1], Patrick W. B. Derksen [8] and Anne Vincent-Salomon [7,*]

The Oncologist
Breast Cancer

Comprehensive Review of Molecular Mechanisms and Clinical Features of Invasive Lobular Cancer

Nikhil Pramod,[a,†] Akanksha Nigam,[a,†] Mustafa Basree,[b] Resham Mawalkar,[a] Saba Mehra,[c] Neelam Shinde,[a] Gary Tozbikian,[a] Nicole Williams,[a] Sarmila Majumder,[a] Bhuvaneswari Ramaswamy[a]

McCart Reed et al. Breast Cancer Research (2021) 23:6
https://doi.org/10.1186/s13058-020-01384-6
Breast Cancer Research
REVIEW Open Access

Invasive lobular carcinoma of the breast: the increasing importance of this special subtype

Amy E. McCart Reed[1,2*], Lauren Kalinowski[1,3], Peter T. Simpson[1,2] and Sunil R. Lakhani[1,4]

diagnostics
MDPI

Review

Diagnostically Challenging Subtypes of Invasive Lobular Carcinomas: How to Avoid Potential Diagnostic Pitfalls

Nektarios Koufopoulos [1,*], Ioannis S. Pateras [1], Alina Roxana Gouloumis [1], Argyro Ioanna Ieronimaki [1], Andriani Zacharatou [1], Aris Spathis [1], Danai Leventakou [1], Panagiota Economopoulou [2], Amanda Psyrri [2], Nikolaos Arkadopoulos [3] and Ioannis G. Panayiotides [1]

Thank you!

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e-Session

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The next e-ESO Session

will take place on 12th January 2022, at the same time

Primary axillary surgery: techniques and indications:

Expert: **Prof Andreas Karakatsanis**, Uppsala University; Uppsala University Hospital, Uppsala, Sweden

Discussant: **Dr Orit Kaidar-Person**, Head of Breast Radiation Unit, Sheba Tel Hashomer, Ramat Gan, Israel

Discussant: **Dr Corrado Tinterri**, Humanitas Cancer Center, Rozzano, Italy

Thank you!

for participating in this
e-session
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