La nuova terapia immunologica per combattere il tumore al polmone

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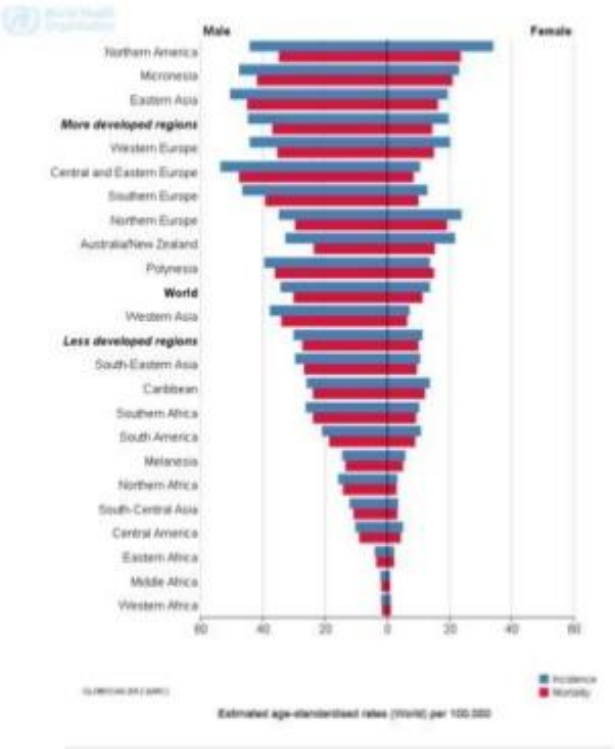
FONDAZIONE IRCCS
ISTITUTO NAZIONALE
DEI TUMORI

Global Lung Cancer Incidence and Mortality



Most common cancer worldwide

- 1.6 million deaths in 2012

Fifty eight percent of new cases in underdeveloped regions

Highest incidence and mortality in men

- Central and Eastern Europe
- Eastern Asia

Women have lower incidence and mortality

- Highest in North America – cultural differences in smoking prevalence
- Lag in when women started smoking

 http://globocan.iarc.fr/Pages/fact_sheets_cancer.aspx

In Italia circa 42000 nuovi casi all' anno

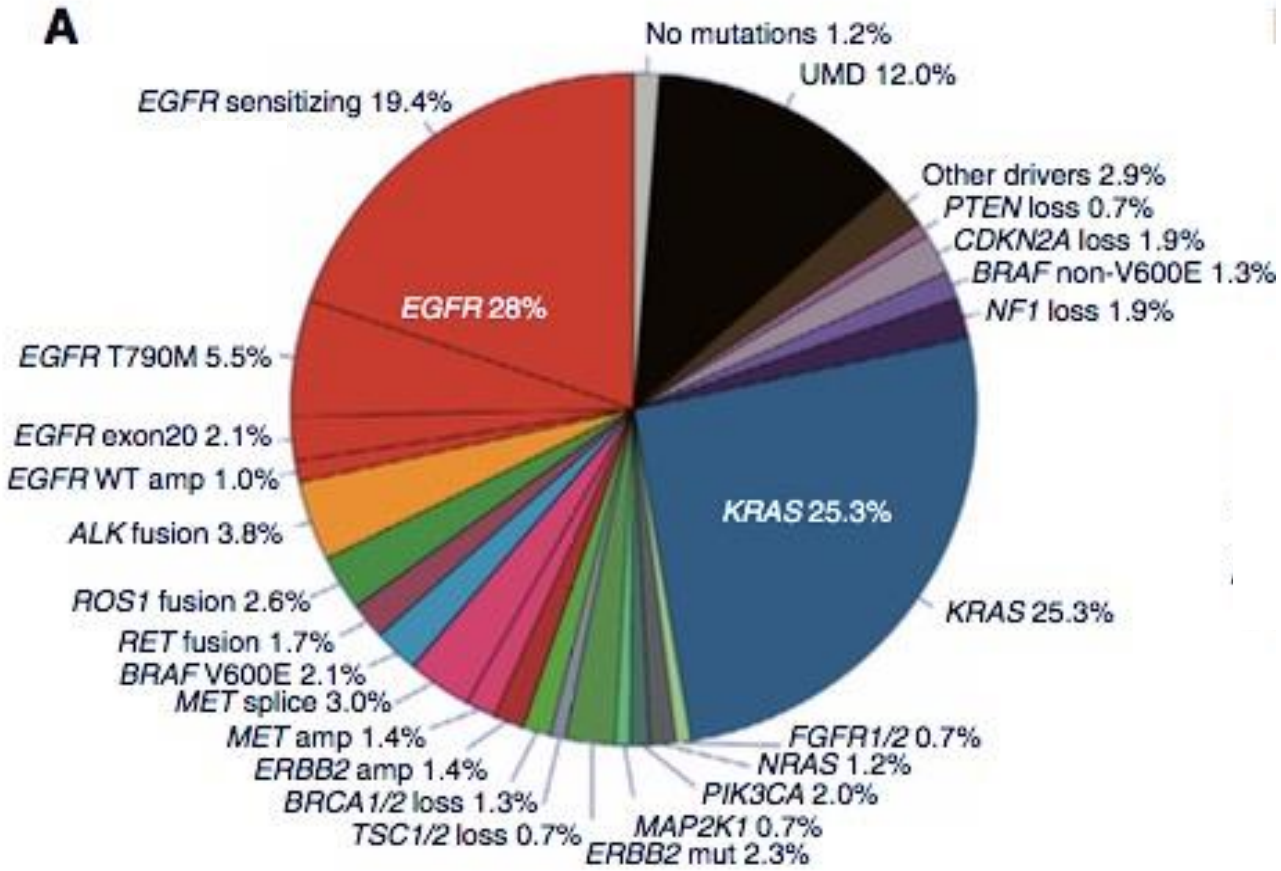
“MUTATED”MAINLY NEVER SMOKERS

TARGET THERAPIES
TARGET THERAPIES
CHEMOTHERAPY

“WILD TYPE” MAINLY SMOKERS

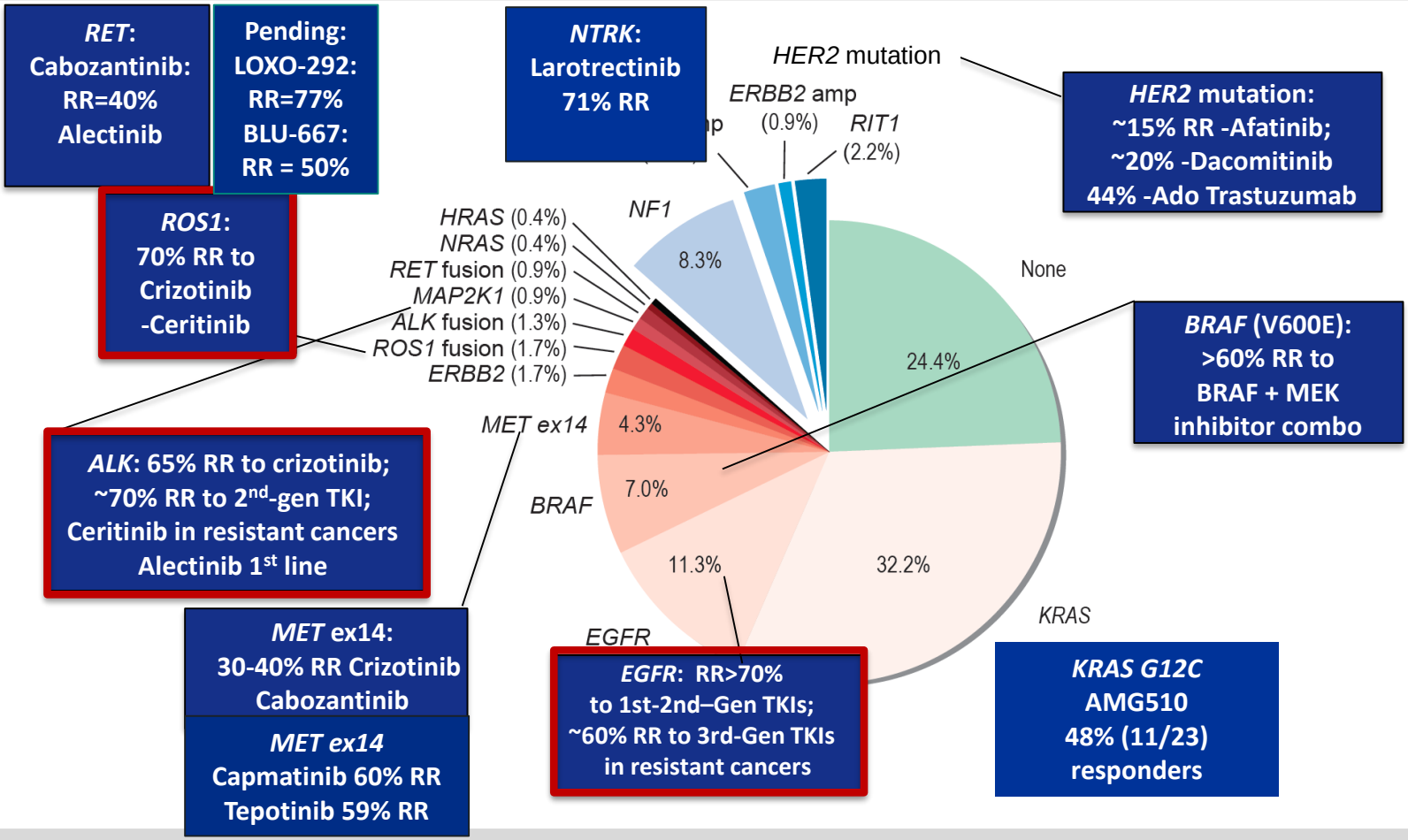
IMMUNOTHERAPY
CHEMOTHERAPY

Molecular alterations in lung adenocarcinoma



Jordan EJ, et al. Cancer Discovery 2017

Growing Number of Oncogene-driven NSCLCs with Active Targeted Therapies



STORIA MOLTO BELLA E LUNGA….

William Coley (1862-1936)

1891: William Coley (Memorial Sloan Kettering Cancer Center-MSKCC, NY). Used the Coley toxin containing live or inactivated bacteria like Serratia Marcescens and Streptococcus pyogenes to treat over 1000 sarcoma patients by intratumor injections. Reproducibility was limited but some patients showed a benefit

Albert Calmette (1863-1933) and Camille Guèrin (1872-1961)

BCG is a vaccine used to prevent tuberculosis (TB). Is composed of mycobacterium Bovis that causes inflammation-dependen immunotherapy of superficial bladder cancer; it has been used for over 30 years. The most effective immunotherapy against a human tumor (ladder)

Paul Ehrlich (1854-1905)

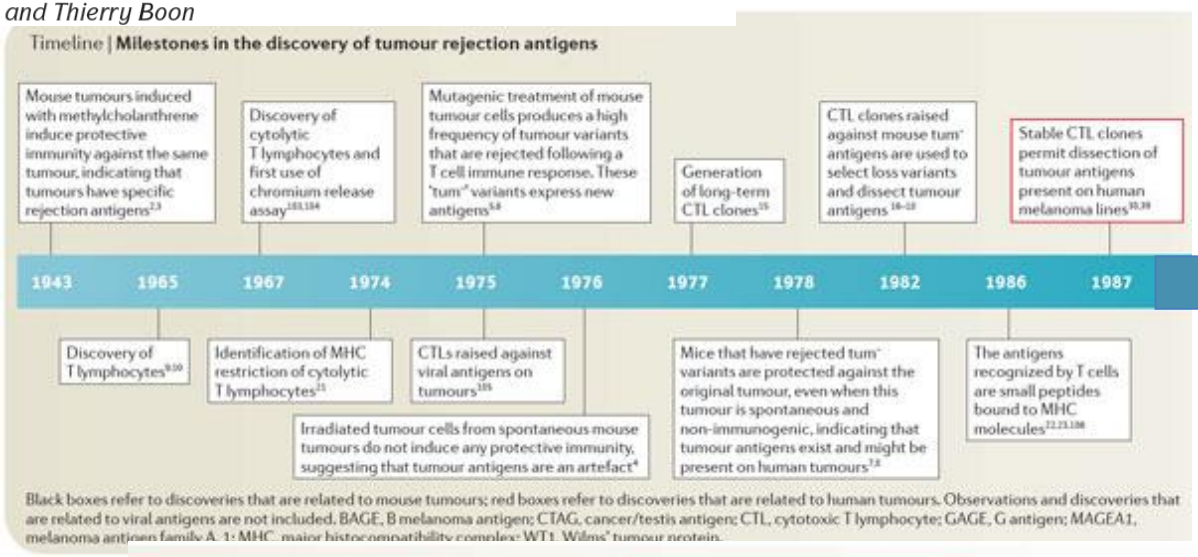
Microbiologo tedesco (fondatore della chemioterapia) 1900: suggerisce che alcune molecole all'interno dell'organismo possono essere in grado di combattere i tumori

Frank Macfarlane Burnet (1899-1989)

Suggerisce che le cellule tumorali possono causare una risposta immunitaria in grado di distruggere il tumore senza alcuna manifestazione clinica (1957: teoria dell'Immunosorveglianza)

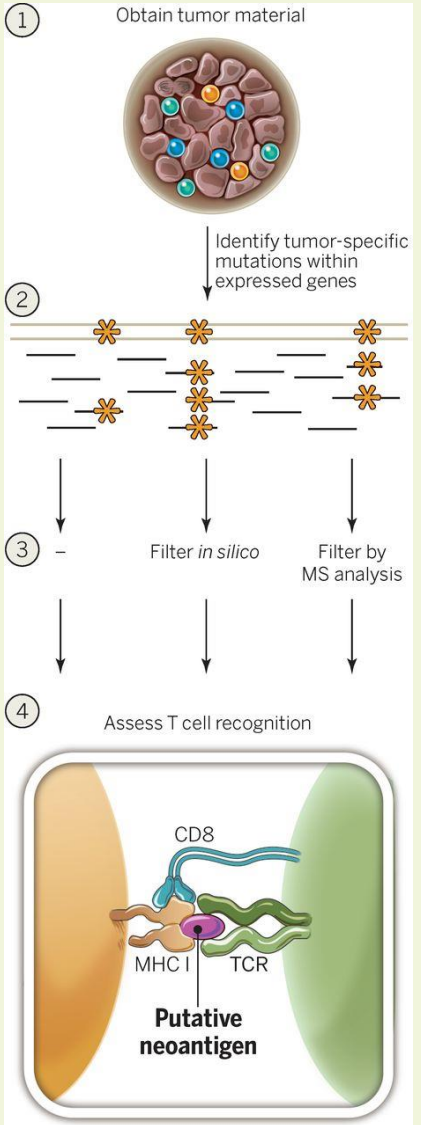
Tumour antigens recognized by T lymphocytes: at the core of cancer immunotherapy

Pierre G. Coulie, Benoît J. Van den Eynde, Pierre van der Bruggen and Thierry Boon



NATURE REVIEWS | CANCER 14:135,2014

Cancer exome–based identification of neoantigens



Ton N. Schumacher, and Robert D. Schreibe
Science 2015;348:69-74

Science
AAAS

The Rapid Pace of Cancer Immunotherapy Research

From the breakthrough of year 2013 for *Nature* and *Science* to the inspiration of the moonshot project for next generation immunotherapy

Escape from immune control is a hallmark of cancer

Elimination

Cancer immunosurveillance

- **Effective antigen processing/presentation**
- **Effective activation and function of effector cells**
  - e.g. T cell activation without co-inhibitory signals

Equilibrium

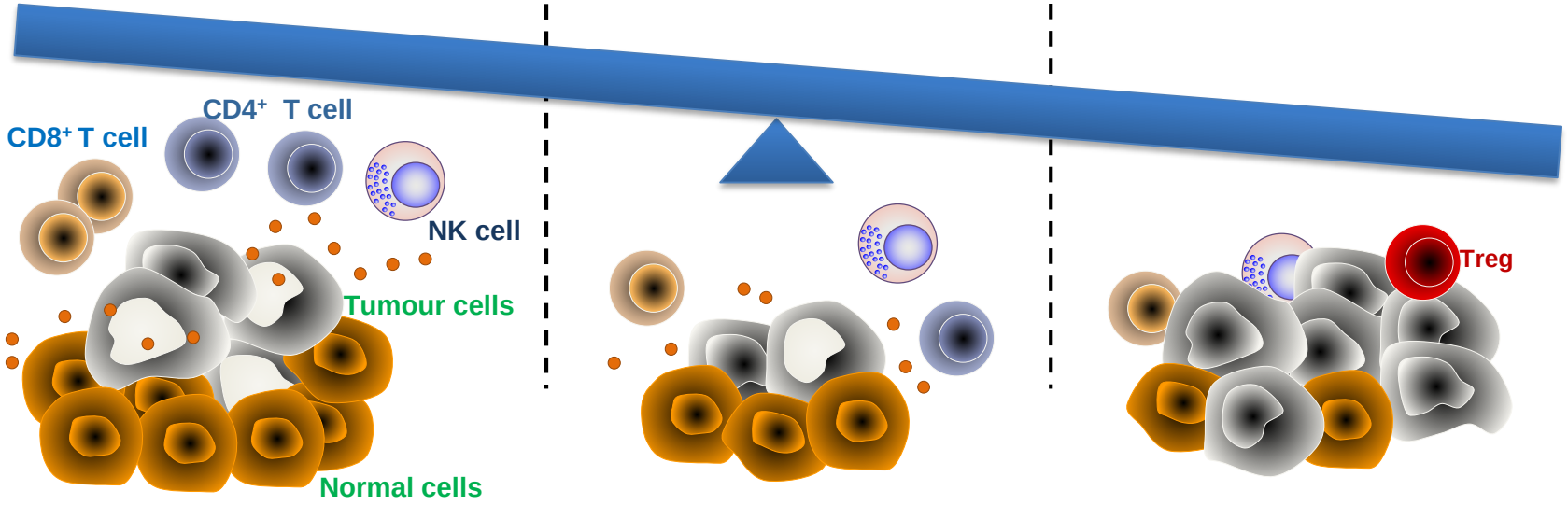
Cancer dormancy

- **Genetic instability**
- **Tumour heterogeneity**
- **Immune selection**

Escape

Cancer progression

- **Tumours avoid elimination through the outgrowth of tumour cells that can suppress, disrupt or 'escape' the immune system**
- **Reduced immunogenicity**



NK = natural killer; Treg = regulatory T cells.

Vesely M and Schreiber R. *Ann N Y Acad Sci.* 2013;1284:1–5.

Tumours use various mechanisms to escape the immune system

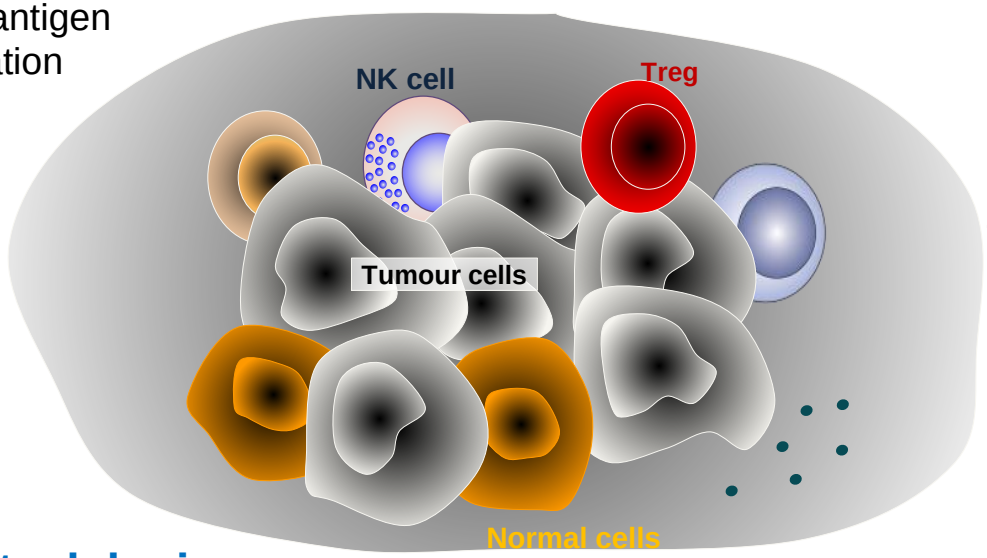
Immune escape mechanisms are complex and frequently overlapping

Ineffective presentation of tumour antigens[1]

e.g. down regulation of MHC I and DC/APC defects in antigen processing/presentation

Recruitment of immunosuppressive cell types[1,2]

e.g. Tregs, MDSC, others



Inhibition of attack by immune cells[1,2]

e.g. disruption of T cell-activating and checkpoint pathways (i.e. PD-1/PD-L1)

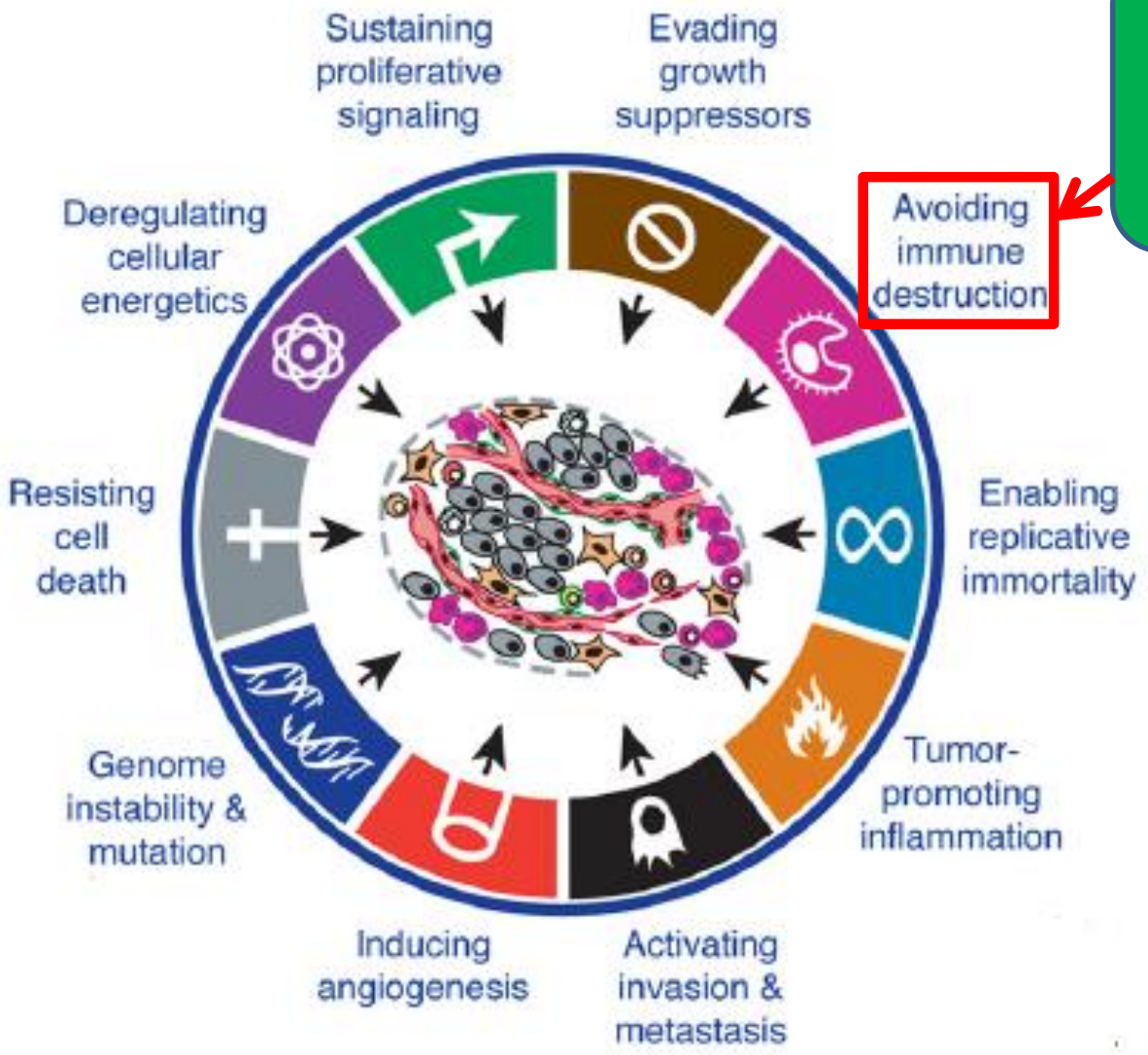
Secretion of immunosuppressive cytokines[1,2]

e.g. TGF-*B*, IDO, IL-10 inhibiting T cells directly

APC = antigen presenting cell; DC = dendritic cell; IDO = indoleamine 2,3-dioxygenase; IL-10 = Interleukin-10; MDSC = myeloid-derived suppressor cells; MHC = major histocompatibility complex; TGF-β = transforming growth factor-β.

1. Jadus M, et al. *Clin Dev Immunol*. 2012:160724; 2. Quezada S, et al. *Immunol Rev.* 2011;241:104–118.

Immune evasion is an important target for cancer treatment

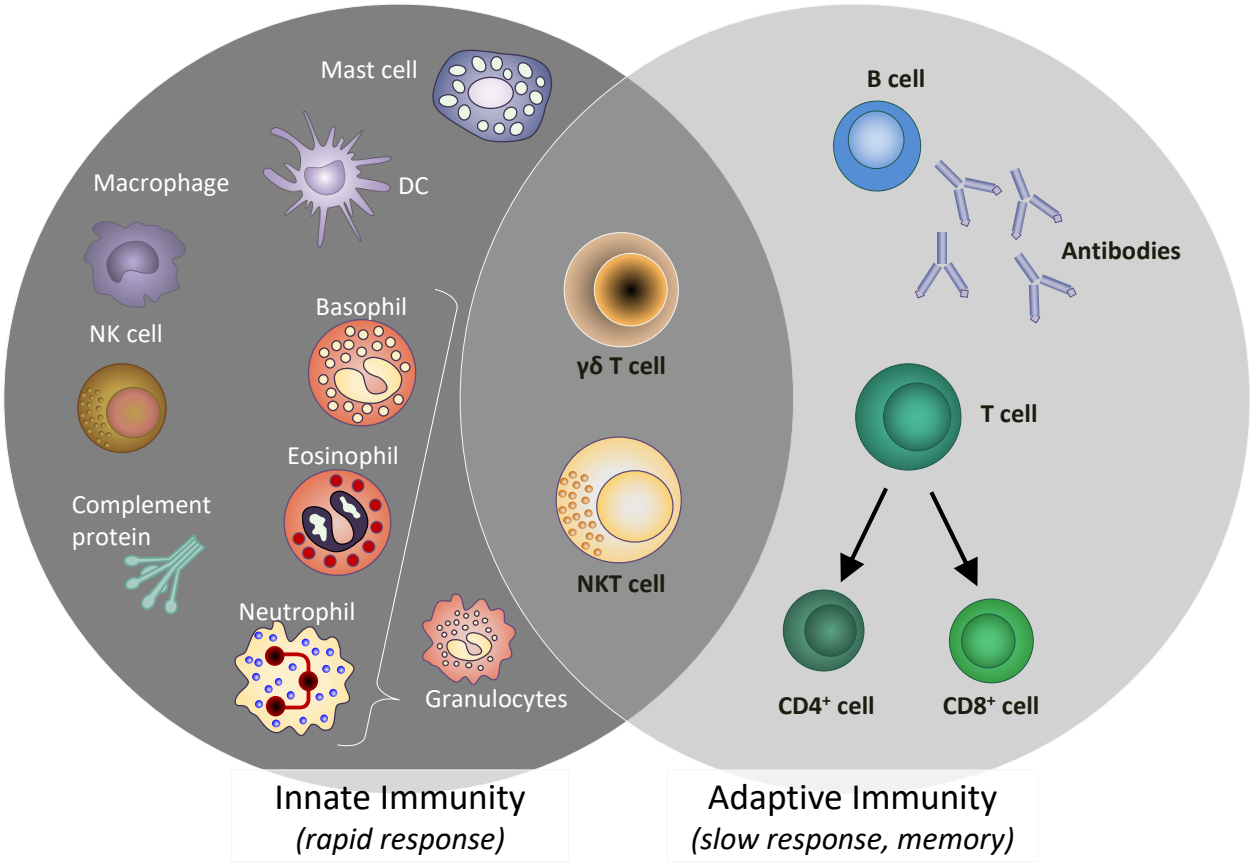


Immune activating
Anti CTLA4
Anti PD-1
Anti PD-L1

Adapted by Hanahan D and Weinberg RA. *Cell* 2011;144:646–674

Cells of the Immune System

- **Innate immune system: involving proteins (chemokines and cytokines) and cells, is considered to be the first line of immune defense and does not generate an antigen-specific response[1,2]**
- **Adaptive immune system: mediated by B and T cells is highly specific and capable of generating an antigen-specific response[1,2]**
  - Induction requires presentation of antigens by cells of the innate immune system

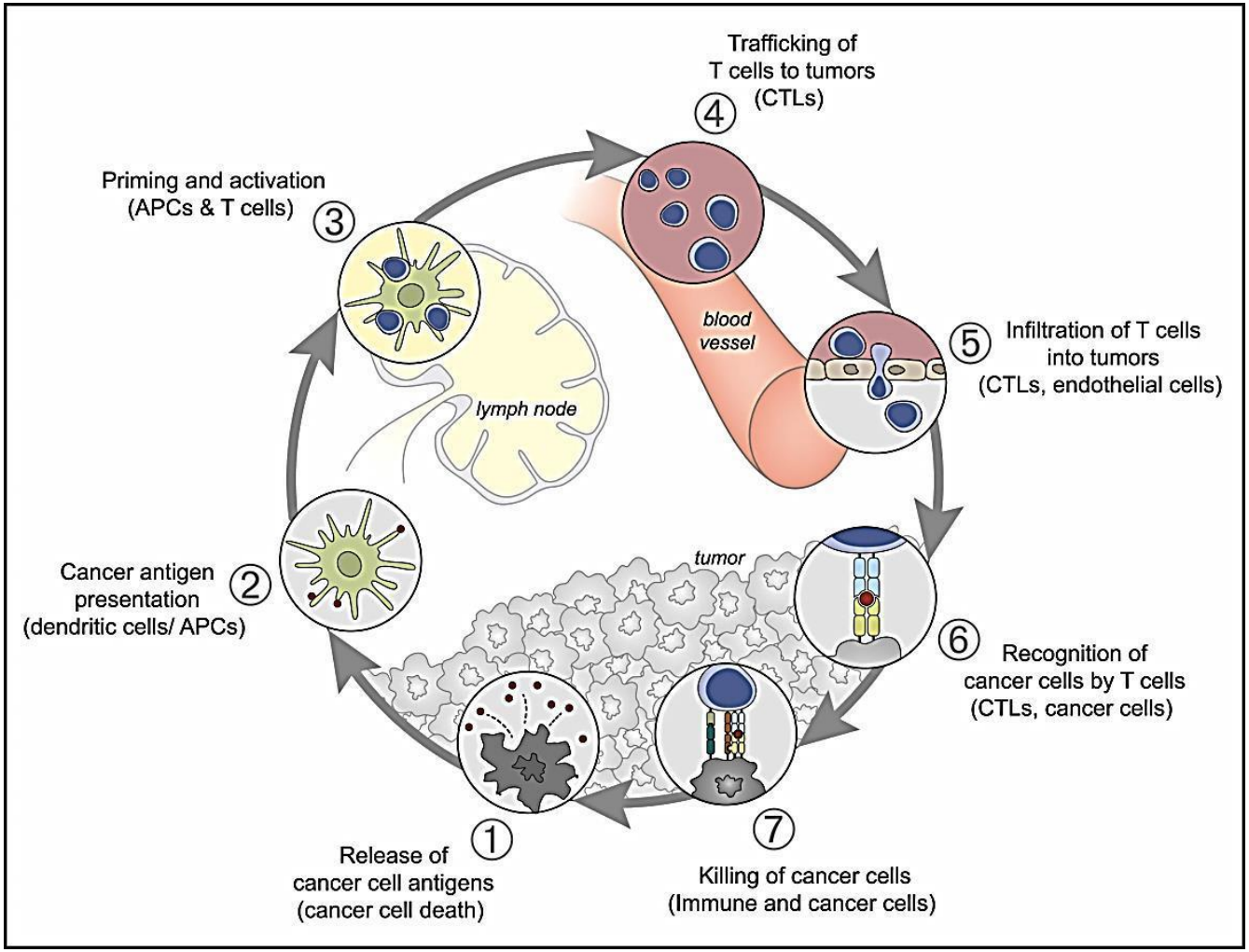


DC = dendritic cell; NK = natural killer

Adapted from Dranoff G.[1]

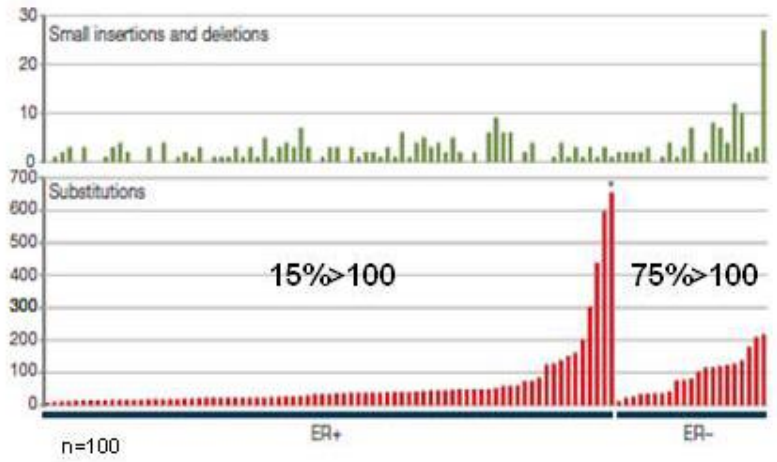
1. Dranoff G. *Nat Rev Cancer*. 2004;4:11–22; 2. Janeway CA, et al. *Immunobiology: The Immune System in Health and Disease* 6th ed. New York, NY: Garland Science; 2004

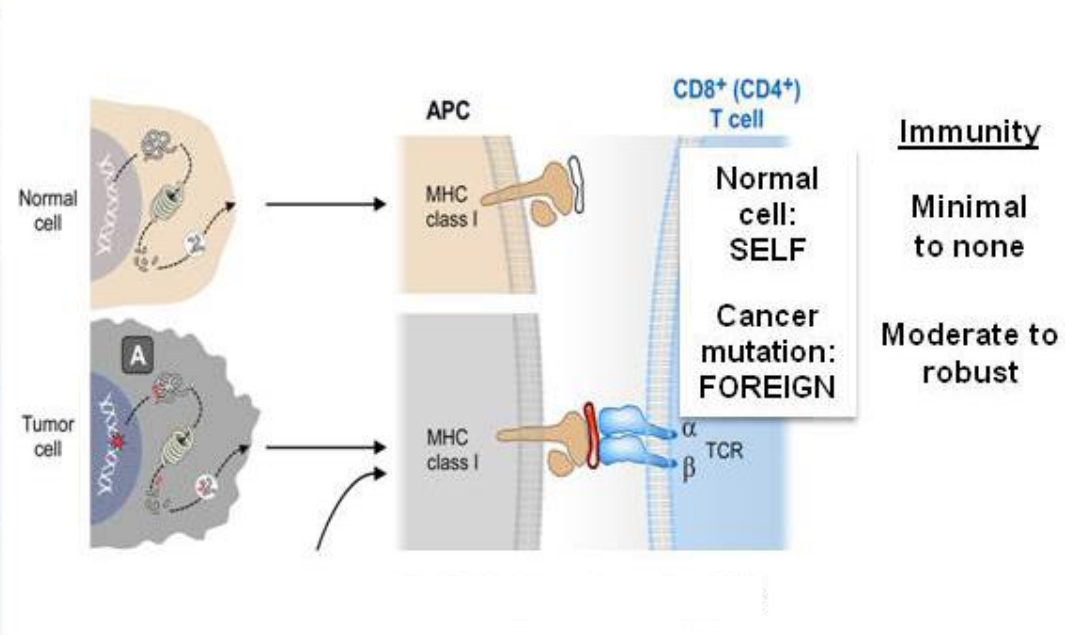
The cycle of cancer immunity



Chen D & Mellman I, Immunity 2013 39,1

Mutational Load Creates Neoantigens

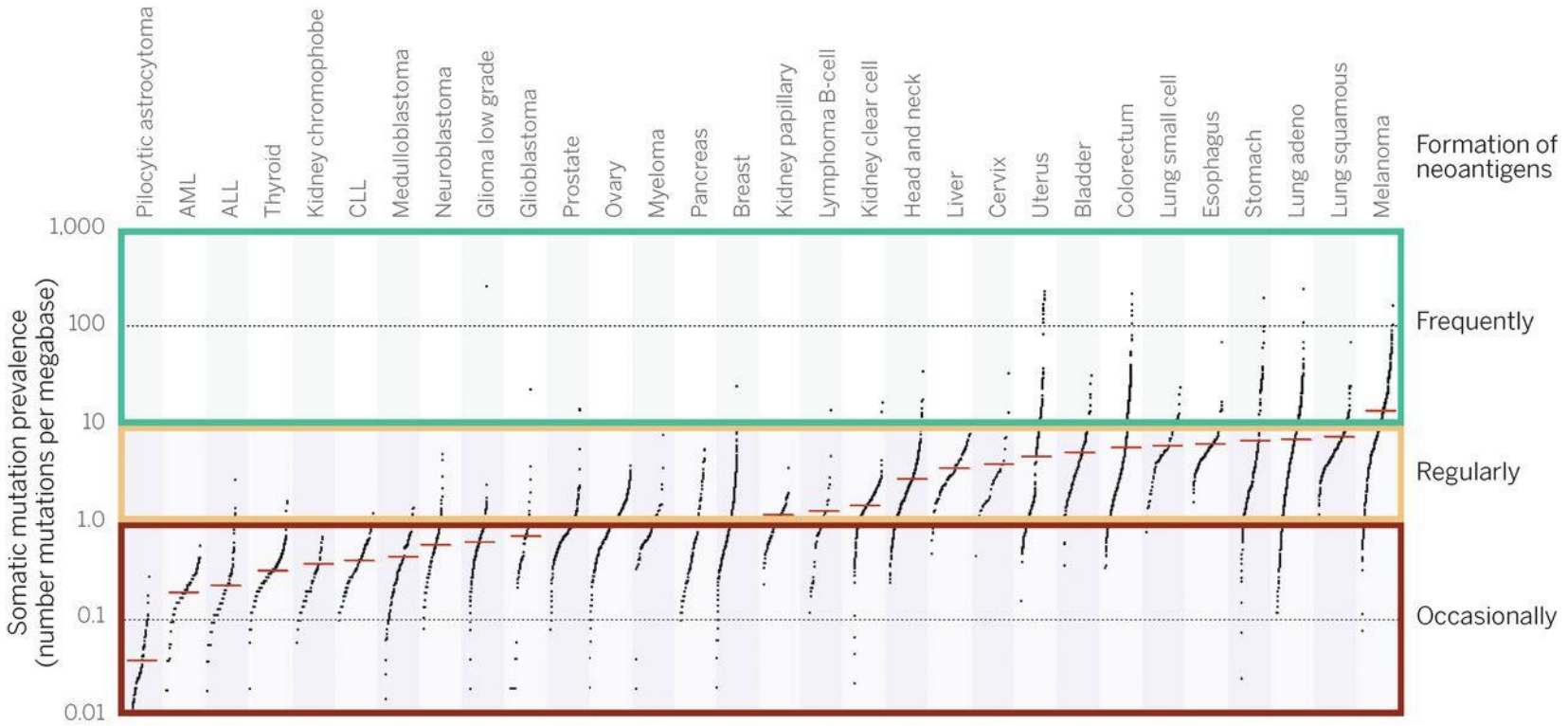




Stephens PJ, et al. *Nature*. 2012;486(7403):400-404. Fritsch EF, et al. *Oncoimmunology*. 2014;3:e29311

Mutational Heterogeneity in Cancer: Altered Proteins Contain Neo-Epitopes for Immune Recognition

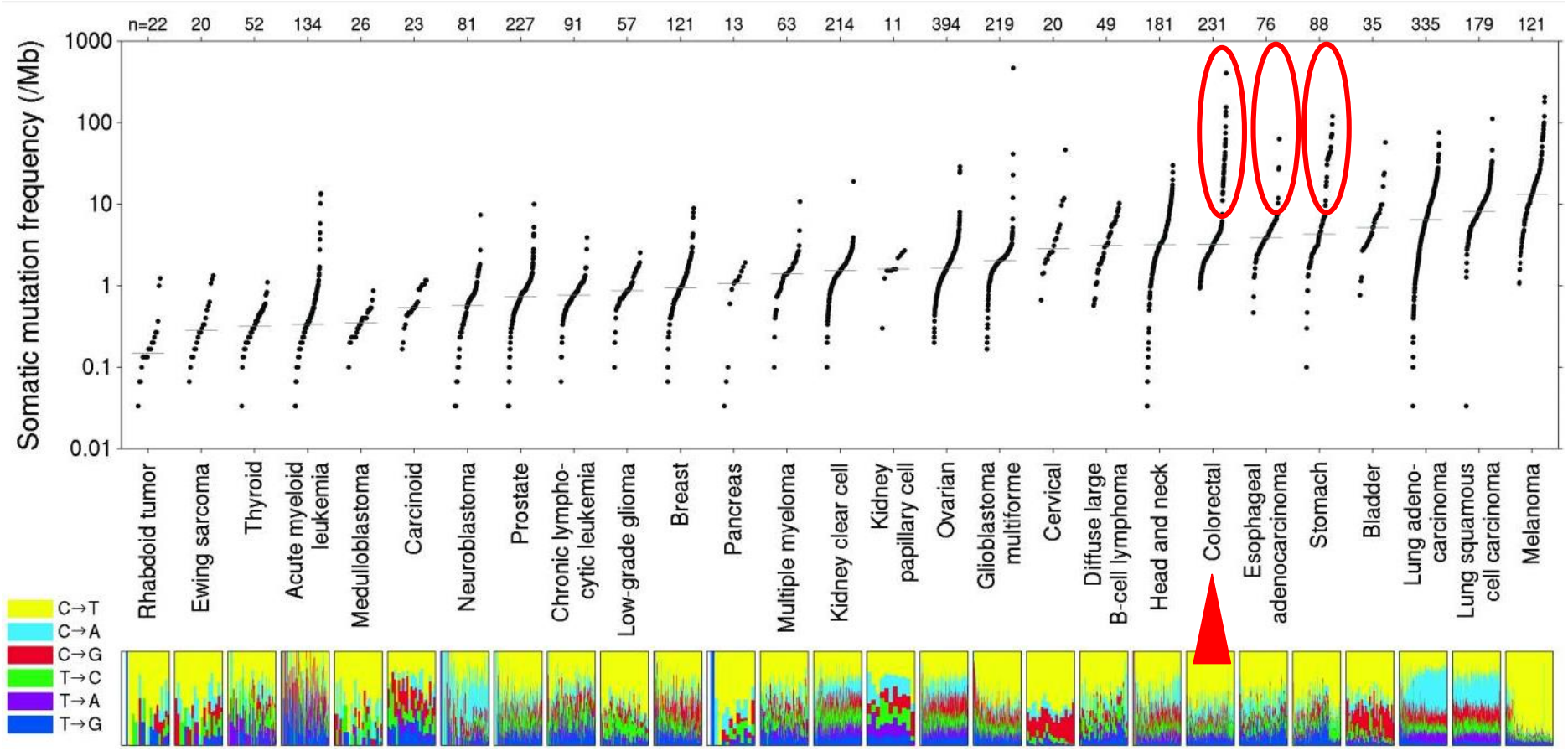
Fig. 2 Estimate of the neoantigen repertoire in human cancer.



Does mutational load correlate with response to immune checkpoint blockade?

Ton N. Schumacher, and Robert D. Schreiber Science 2015;348:69-74

Colorectal Cancers Are Generally Unresponsive to PD-1 Blockade, but the MSI-High Subset Has a High Mutational Load

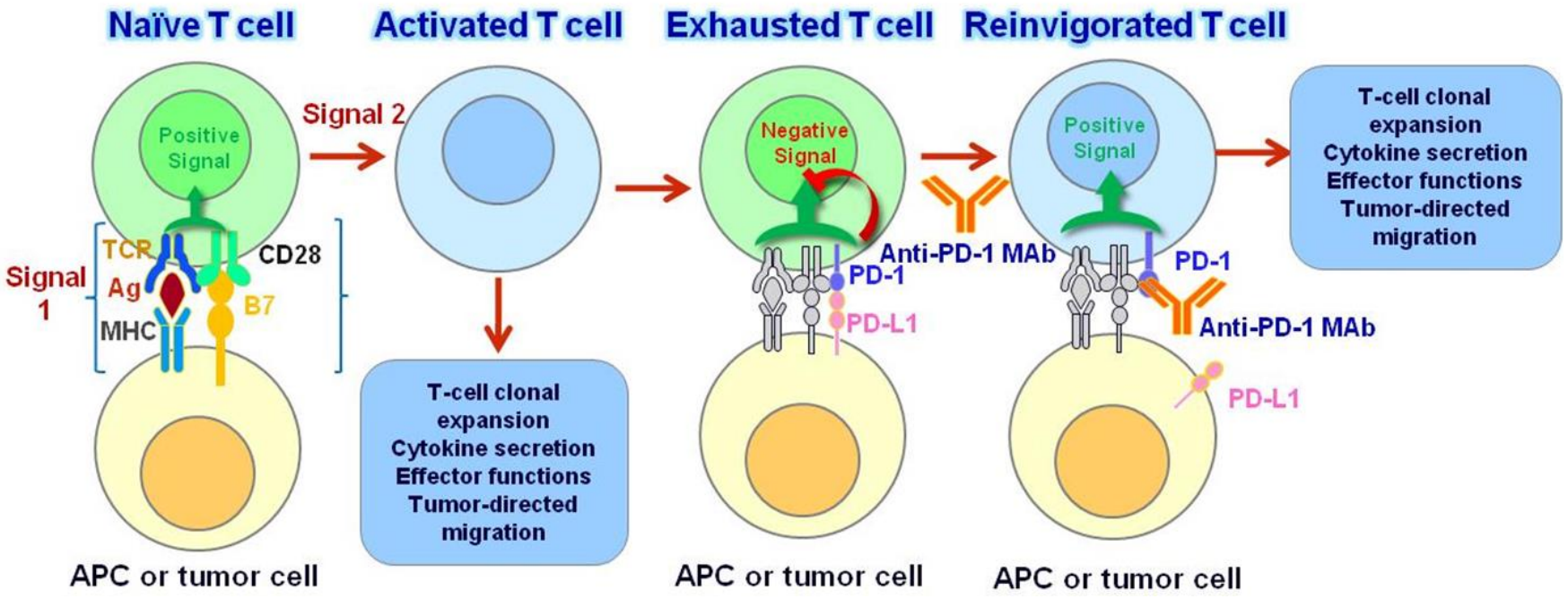


Lawrence MS, et al. *Nature*. 2013;499(7457):214-218.

Microsatellite instability (MSI): Genetic hypermutability resulting from deficient mismatch repair (dMMR), present in ~15% colon cancers and in some other tumor types

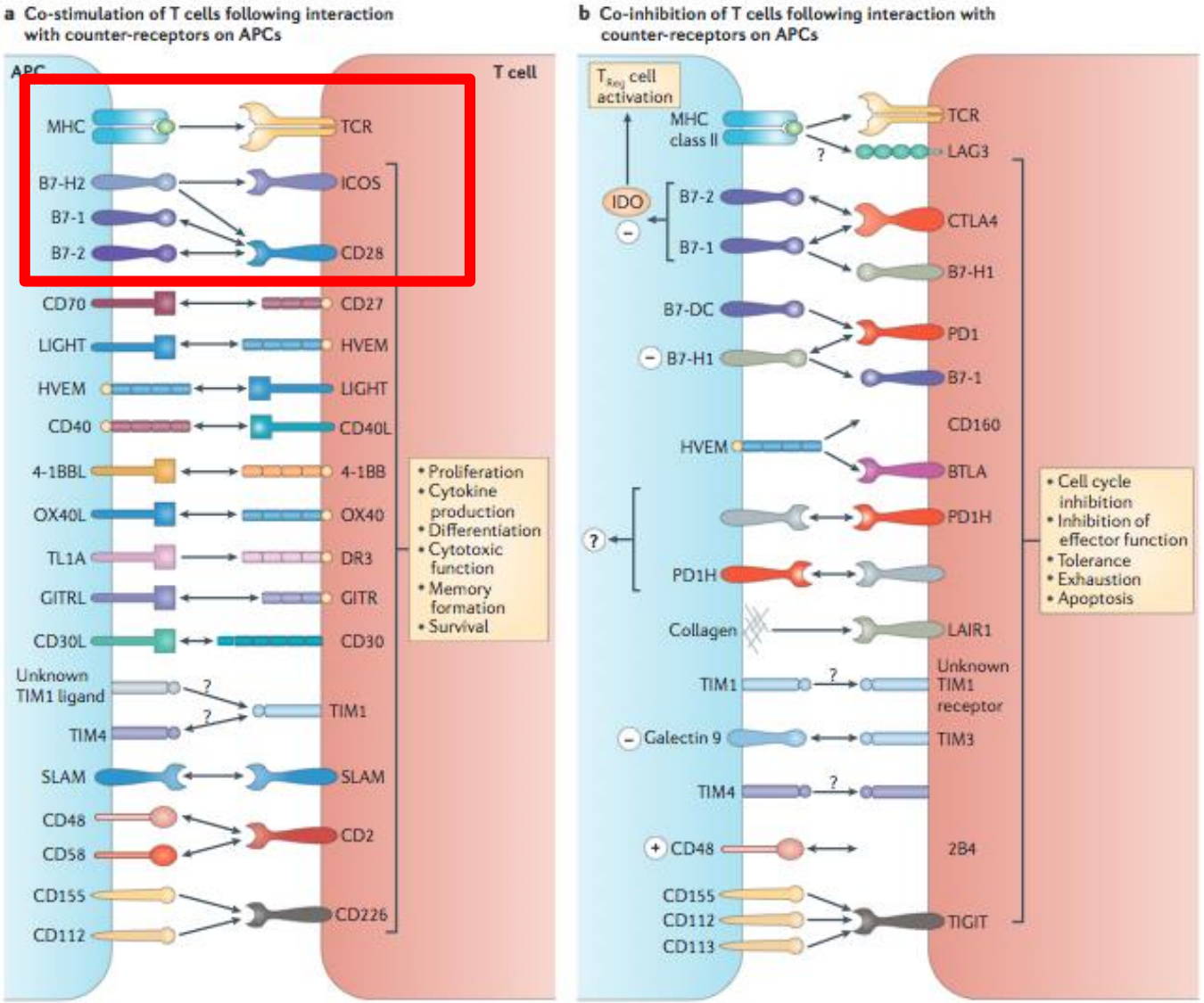
Topalian SL, et al. *J Clin Oncol.* 2013;31(suppl): Abstract 3002.

Segnali attivatori e inibitori



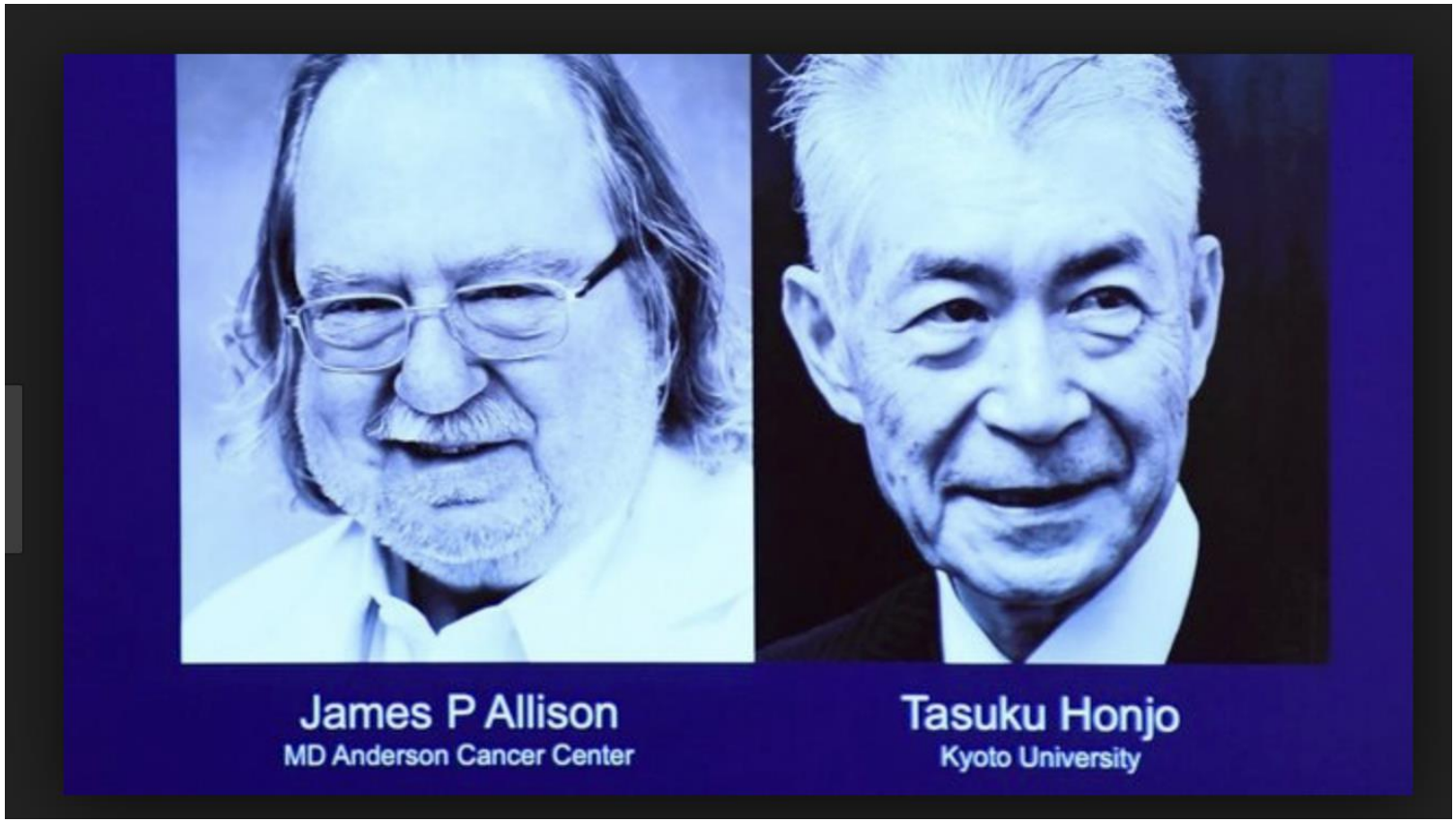
Topalian and Brahmer NEJM 2012

IMMUNOLOGIC SYNAPSIS

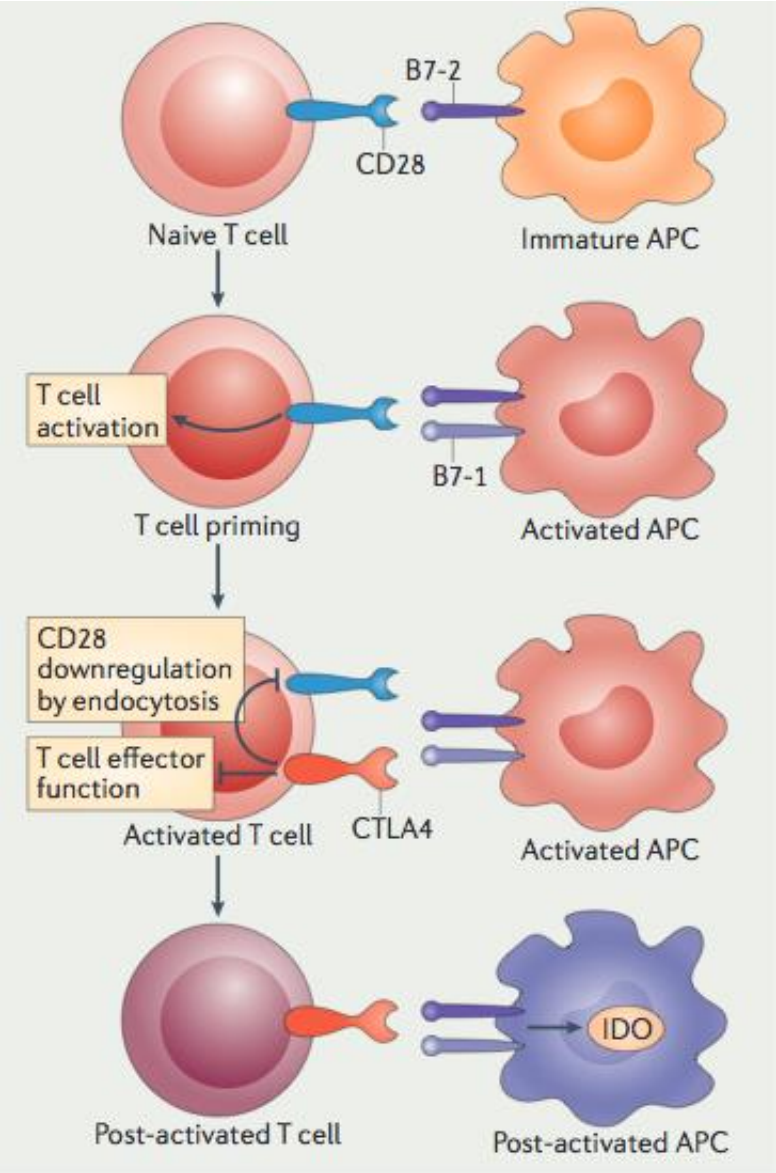


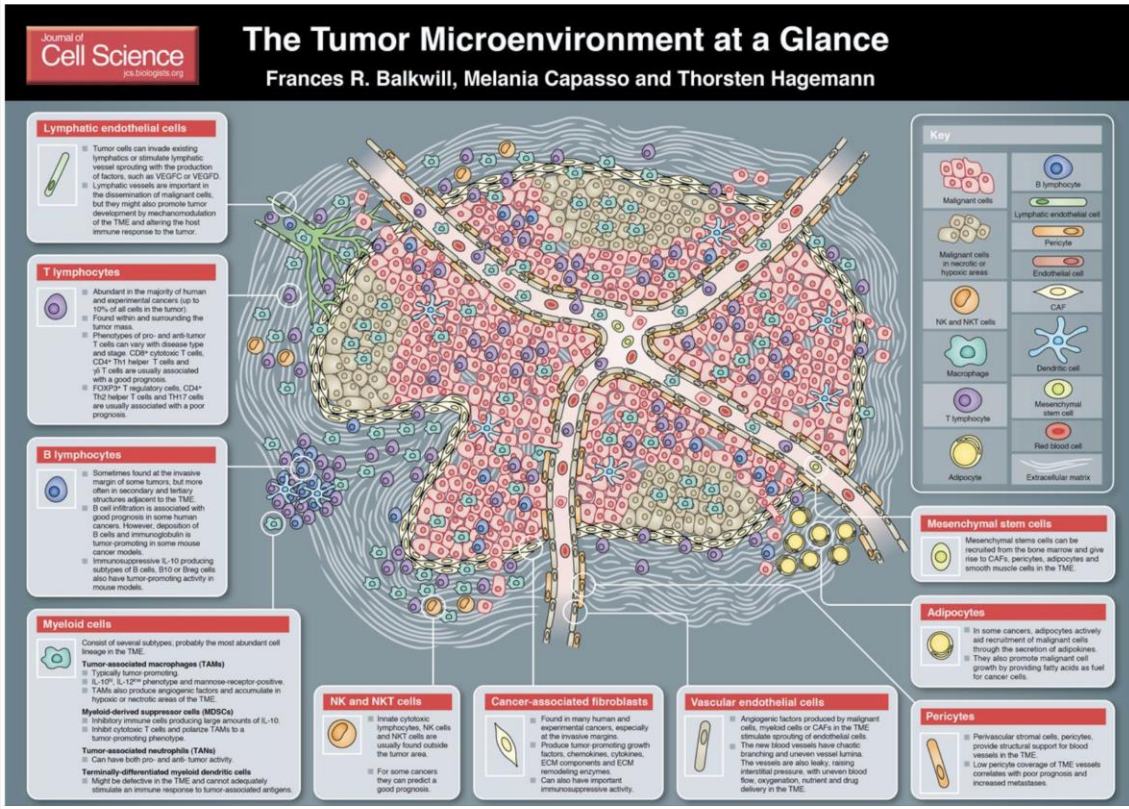
L.Chen et al, Nature Reviews Immunology, 2013 Apr;13(4):227-42

Nobel Prize 2018



Explanation of the Molecular Mechanisms of Checkpoint Inhibitors and Other Key Emerging Immunologic Strategies





Chen L, et al. *Nat Rev Immunol.* 2013;13(4):227-242.

FARMACI

ANTI PD-1

Pembrolizumab

Nivolumab

ANTI PD-L1

Atezolizumab

Durvalumab

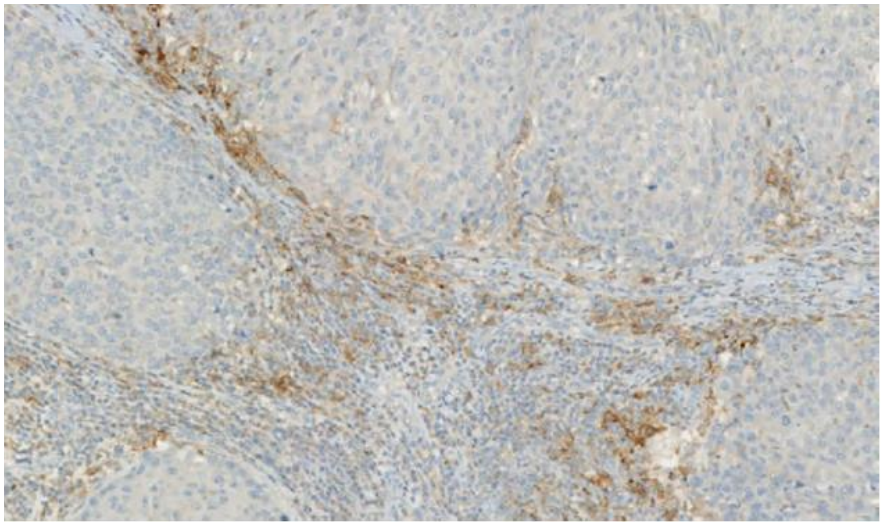
Anti CTLA4

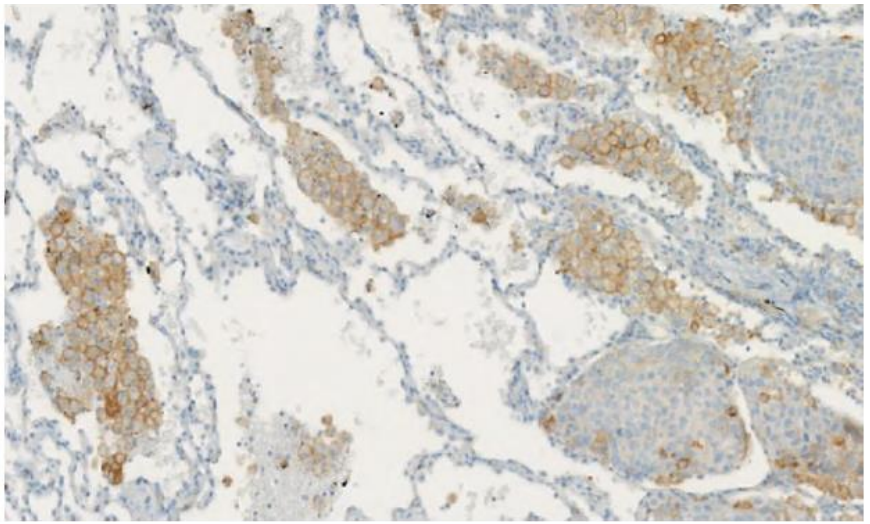
Ipilimumab

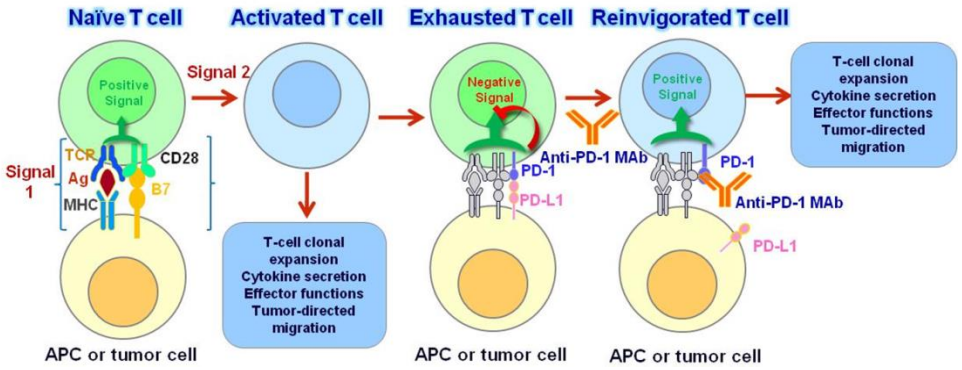
Tremelimumab

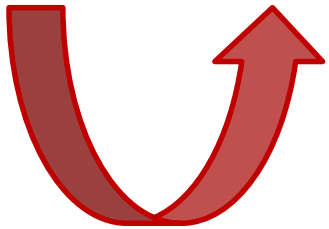
PD-L1 (IHC) as a Biomarker

Expression on tumour cells and on immune cells









The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Nivolumab versus Docetaxel in Advanced Squamous-Cell Non–Small-Cell Lung Cancer

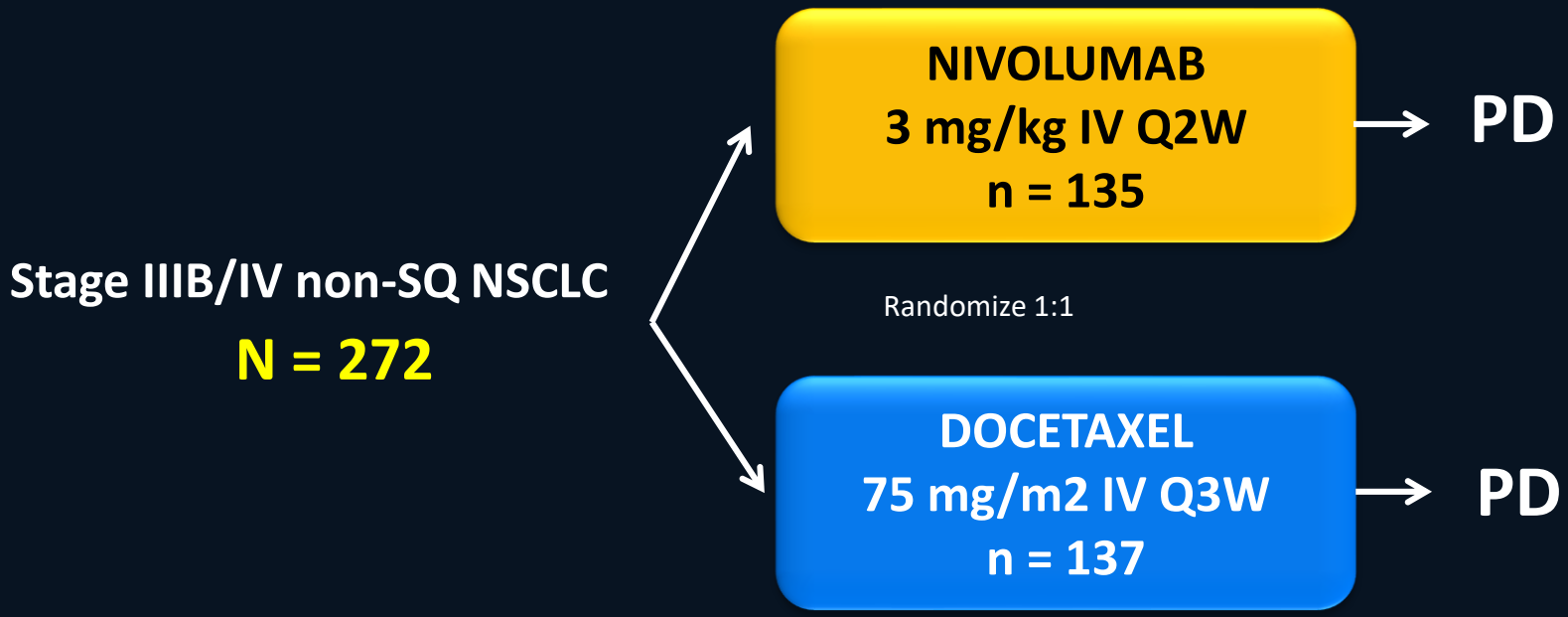
Julie Brahmer, M.D., Karen L. Reckamp, M.D., Paul Baas, M.D., Lucio Crinò, M.D., Wilfried E.E. Eberhardt, M.D., Elena Poddubskaya, M.D., Scott Antonia, M.D., Ph.D., Adam Pluzanski, M.D., Ph.D., Everett E. Vokes, M.D., Esther Holgado, M.D., Ph.D., David Waterhouse, M.D., Neal Ready, M.D., Justin Gainor, M.D., Osvaldo Arén Frontera, M.D., Libor Havel, M.D., Martin Steins, M.D., Marina C. Garassino, M.D., Joachim G. Aerts, M.D., Manuel Domine, M.D., Luis Paz-Ares, M.D., Martin Reck, M.D., Christine Baudelet, Ph.D., Christopher T. Harbison, Ph.D., Brian Lestini, M.D., Ph.D., and David R. Spigel, M.D.

CHECKMATE-017


In NSCLC
CHECKMATE

CHECKMATE 017-squamous

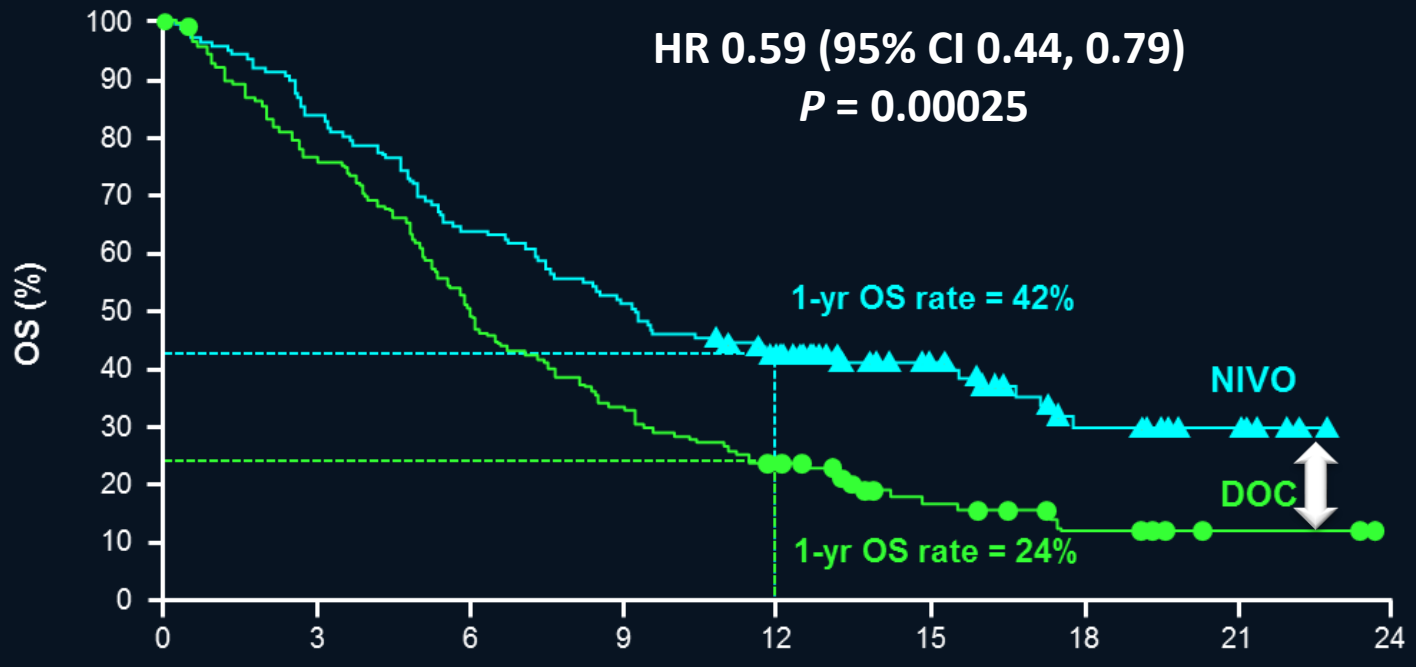
Nivolumab (anti PD-1)



PRIMARY ENDPOINT OS

CHECKMATE-017 interim analysis

Nivolumab vs docetaxel squamous 2nd-line



Number of patients at risk									
NIVO	135	113	86	69	52	31	15	7	0
DOC	137	103	68	45	30	14	7	2	0

ISTITUTO NAZIONALE
PER LO STUDIO
E LA CURA DEI TUMORI

DOC, docetaxel; NIVO, nivolumab
Brahmer J, NEJM, 2015

THE LANCET

Articles

Pembrolizumab versus docetaxel for previously treated, PD-L1-positive, advanced non-small-cell lung cancer (KEYNOTE-010): a randomised controlled trial

Roy S Herbst, Paul Baas, Dong-Wan Kim, Enriqueta Felip, José L Pérez-Gracia, Ji-Youn Han, Julian Molina, Joo-Hang Kim, Catherine Dubos Arvis, Myung-Ju Ahn, Margarita Majem, Mary J Fidler, Gilberto de Castro Jr, Marcelo Garrido, Gregory M Lubiniecki, Yue Shentu, Ellie Im, Marisa Dolled-Filhart, Edward B Garon

Summary

Background Despite recent advances in the treatment of advanced non-small-cell lung cancer, there remains a need for effective treatments for progressive disease. We assessed the efficacy of pembrolizumab for patients with previously treated, PD-L1-positive, advanced non-small-cell lung cancer.

Methods We did this randomised, open-label, phase 2/3 study at 202 academic medical centres in 24 countries. Patients with previously treated non-small-cell lung cancer with PD-L1 expression on at least 1% of tumour cells were randomly assigned (1:1:1) in blocks of six per stratum with an interactive voice-response system to receive pembrolizumab 2 mg/kg, pembrolizumab 10 mg/kg, or docetaxel 75 mg/m² every 3 weeks. The primary endpoints were overall survival and progression-free survival both in the total population, and in patients with PD-L1 expression on at least 50% of tumour cells. We used a threshold for significance of $p<0\cdot00825$ (one-sided). This trial is registered at ClinicalTrials.gov, number NCT01905657.

Published Online
December 19, 2015
http://dx.doi.org/10.1016/S0140-6736(15)01281-7

See Online/Comment
http://dx.doi.org/10.1016/S0140-6736(15)01308-2

Yale School of Medicine, Yale Cancer Center, and Smilow Cancer Hospital, New Haven, CT, USA (Prof R S Herbst MD); **The Netherlands Cancer Institute and The Academic Medical Hospital Amsterdam,**

KEYNOTE-010 Study Design

Patients

- Advanced NSCLC
- Confirmed PD after ≥1 line of chemotherapy[a]
- No active brain metastases
- ECOG PS 0-1
- PD-L1 TPS ≥1%
- No serious autoimmune disease
- No ILD or pneumonitis requiring systemic steroids

R
1:1:1

- Pembrolizumab 2 mg/kg IV Q3W for 24 months
- Pembrolizumab 10 mg/kg IV Q3W for 24 months
- Docetaxel 75 mg/m^2 Q3W per local guidelines[c]

Stratification factors:

- ECOG PS (0 vs 1)
- Region (East Asia vs non-East Asia)
- PD-L1 status[b] (TPS ≥50% vs 1%-49%)

End points in the TPS ≥50% stratum and TPS ≥1% population

- Primary: PFS and OS
- Secondary: ORR, duration of response, safety

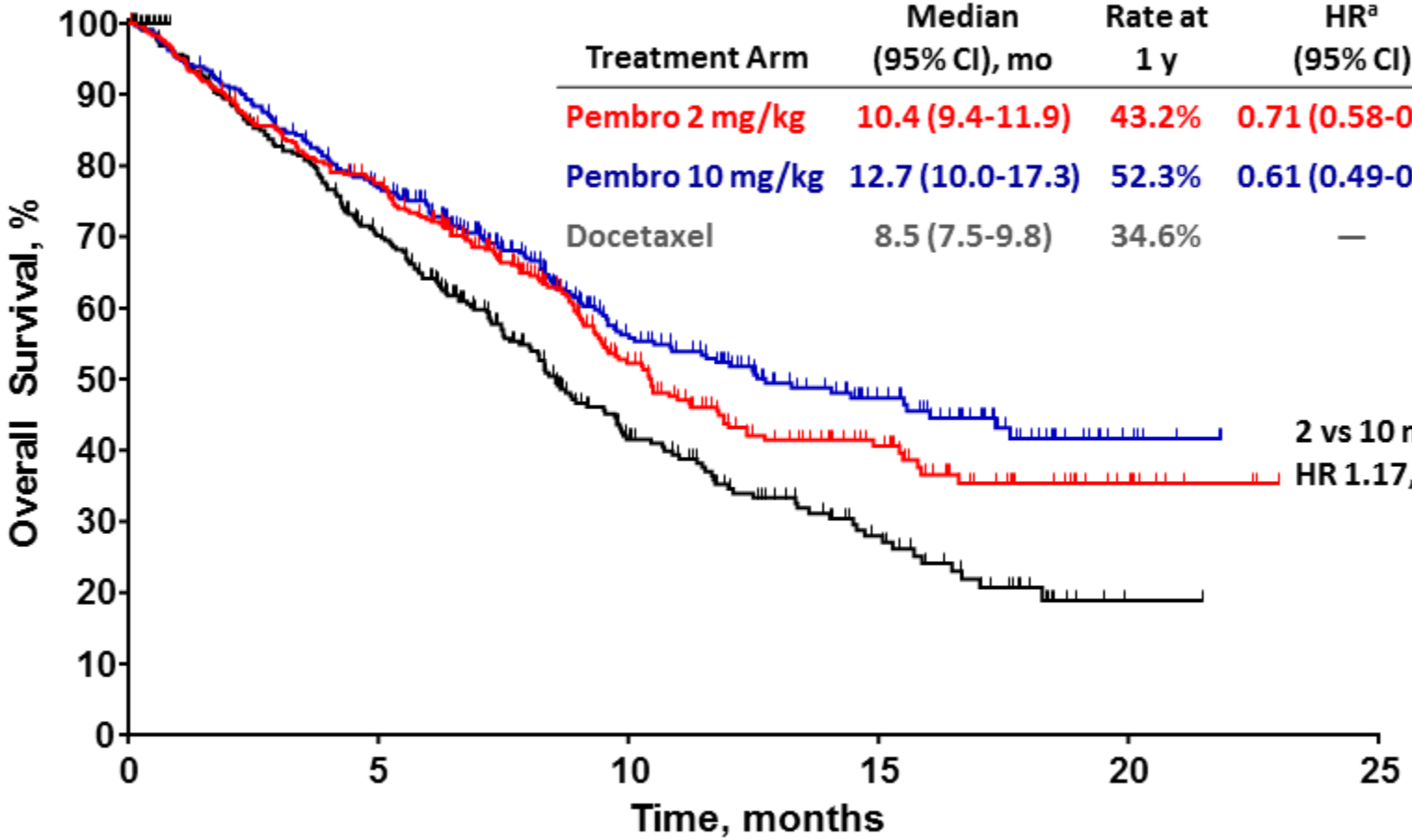
ClinicalTrials.gov, NCT01905657.

[a]Prior therapy must have included ≥2 cycles of platinum-doublet chemotherapy. An appropriate tyrosine kinase inhibitor was required for patients whose tumors had an *EGFR* sensitizing mutation or an *ALK* translocation.

[b]Added after 441 patients enrolled based on results from KEYNOTE-001 (Garon EB et al. *N Engl J Med*. 2015;372:2018-28).

[c]Patients received the maximum number of cycles permitted by the local regulatory authority.

OS, PD-L1 TPS ≥1% (Total Population)



Treatment Arm	Median (95% CI), mo	Rate at 1 y	HR[a] (95% CI)	*P*
Pembro 2 mg/kg	10.4 (9.4-11.9)	43.2%	0.71 (0.58-0.88)	0.0008
Pembro 10 mg/kg	12.7 (10.0-17.3)	52.3%	0.61 (0.49-0.75)	<0.0001
Docetaxel	8.5 (7.5-9.8)	34.6%	—	—

2 vs 10 mg/kg:
HR 1.17, 95% CI 0.94-1.45

Analysis cut-off date: September 30, 2015.

[a]Comparison of pembrolizumab vs docetaxel.

ORIGINAL ARTICLE

Pembrolizumab versus Chemotherapy for PD-L1–Positive Non–Small-Cell Lung Cancer

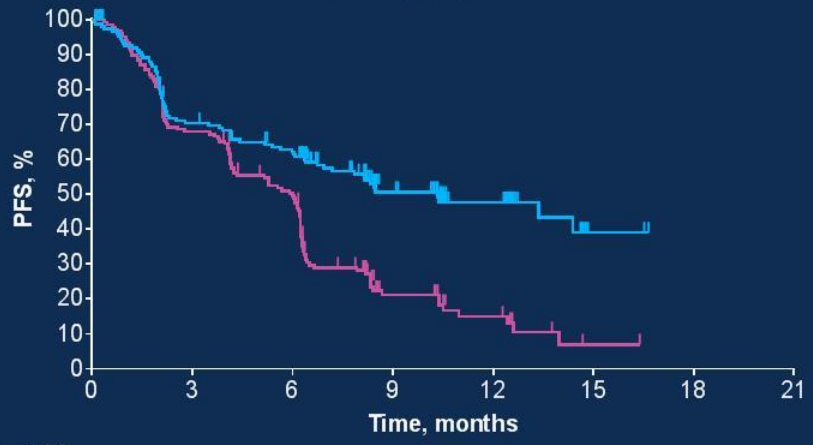
Martin Reck, M.D., Ph.D., Delvys Rodríguez-Abreu, M.D., Andrew G. Robinson, M.D., Rina Hui, M.B., B.S., Ph.D., Tibor Csőszi, M.D., Andrea Fülöp, M.D., Maya Gottfried, M.D., Nir Peled, M.D., Ph.D., Ali Tafreshi, M.D., Sinead Cuffe, M.D., Mary O'Brien, M.D., Suman Rao, M.D., et al., for the KEYNOTE-024 Investigators*

KEYNOTE-024

KEYNOTE-024: Primary Analysis
(Median Follow-Up: 11.2 months)

Progression-Free Survival[a]

HR 0.50 (95% CI 0.37-0.68)
***P* < 0.001**

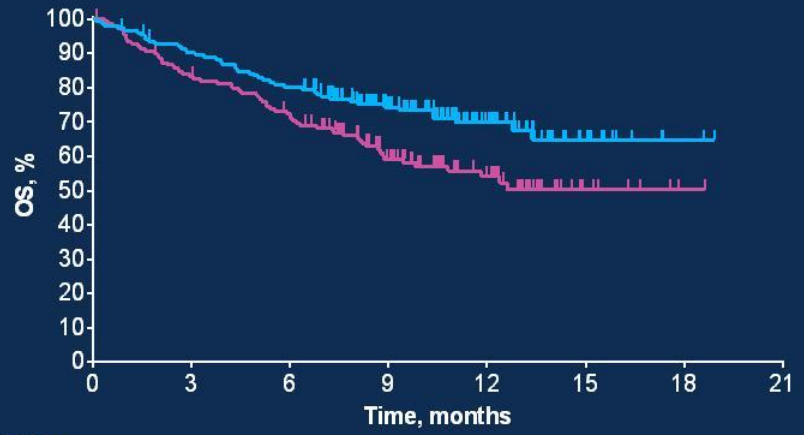


No. at risk

Pembro	154	104	89	44	22	3	1	0
Chemo	151	99	70	18	9	1	0	0

Overall Survival

HR 0.60 (95% CI 0.41-0.89)
***P* = 0.005**



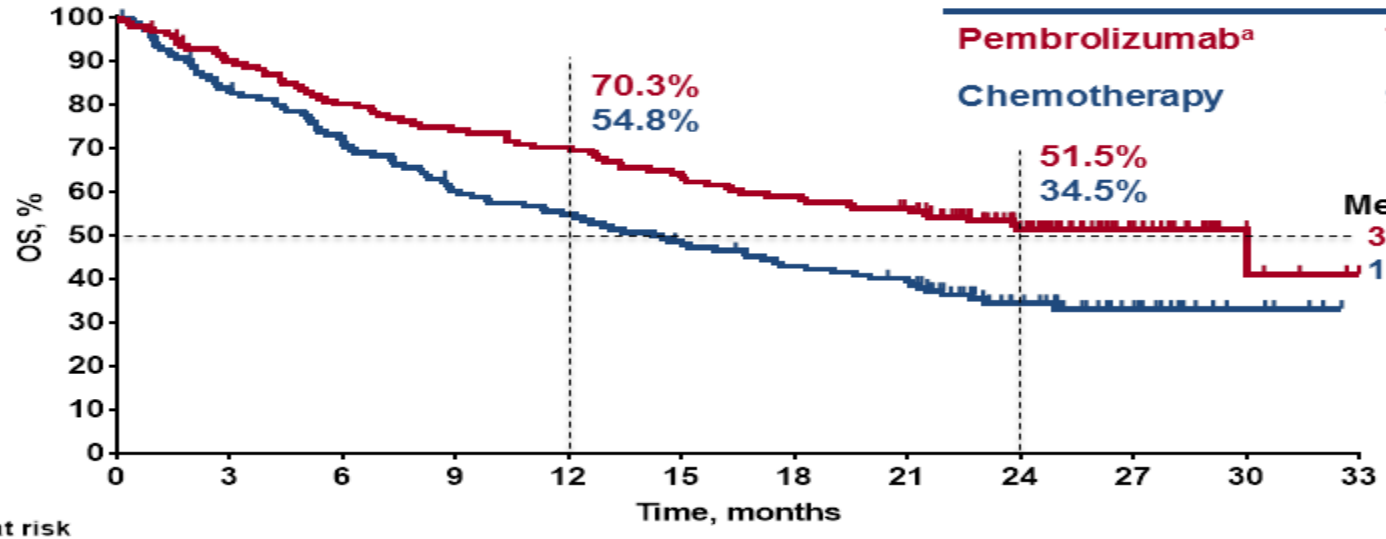
No. at risk

Pembro	154	136	121	82	39	11	2	0
Chemo	151	123	106	64	34	7	1	0

[a]RECIST v1.1 by blinded, independent central review.
Reck M et al. *N Engl J Med* 2016;375:1823-33.
Data cutoff: May 9, 2016.

Brahmer WCLC 2017

Overall Survival: Updated Analysis



	Events, n	HR (95% CI)
Pembrolizumab[a]	73	0.63 (0.47–0.86) *P* = 0.002[b]
Chemotherapy	96	

Median (95% CI)
30.0 mo (18.3 mo–NR)
14.2 mo (9.8 mo–19.0 mo)

No. at risk

Pembro	154	136	121	112	106	96	89	83	52	22	5	0
Chemo	151	123	107	88	80	70	61	55	31	16	5	0

[a]Effective crossover rate from chemotherapy to anti-PD-L1 therapy, 62.3% (82 patients crossed over to pembrolizumab during the study and 12 received anti-PD-L1 therapy outside of crossover). [b]Nominal *P* value. NR, not reached.
Data cutoff: July 10, 2017.

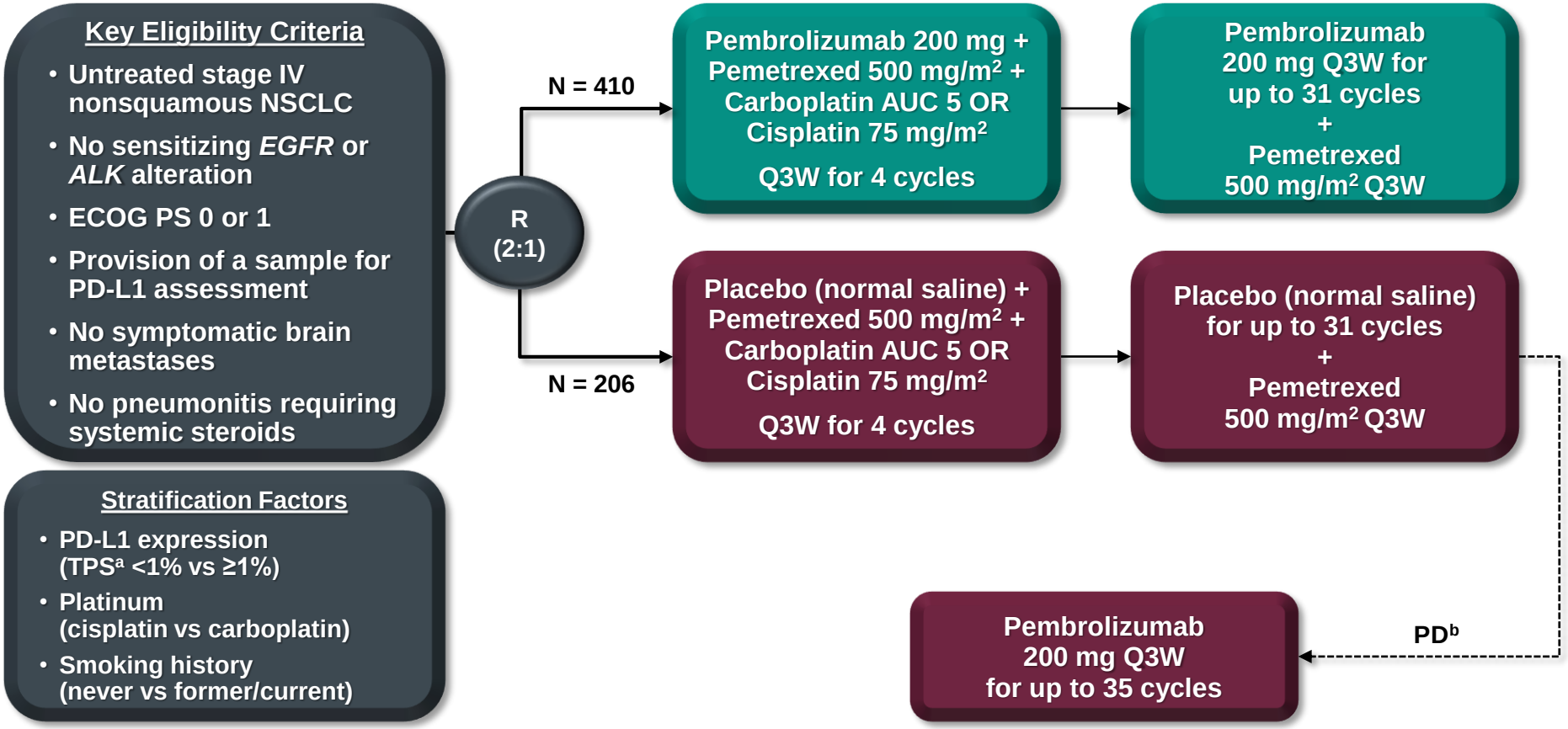
The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Pembrolizumab plus Chemotherapy in Metastatic Non–Small-Cell Lung Cancer

L. Gandhi, D. Rodríguez-Abreu, S. Gadgeel, E. Esteban, E. Felip, F. De Angelis, M. Domine, P. Clingan, M.J. Hochmair, S.F. Powell, S.Y.-S. Cheng, H.G. Bischoff, N. Peled, F. Grossi, R.R. Jennens, M. Reck, R. Hui, E.B. Garon, M. Boyer, B. Rubio-Viqueira, S. Novello, T. Kurata, J.E. Gray, J. Vida, Z. Wei, J. Yang, H. Raftopoulos, M.C. Pietanza, and M.C. Garassino, for the KEYNOTE-189 Investigators*

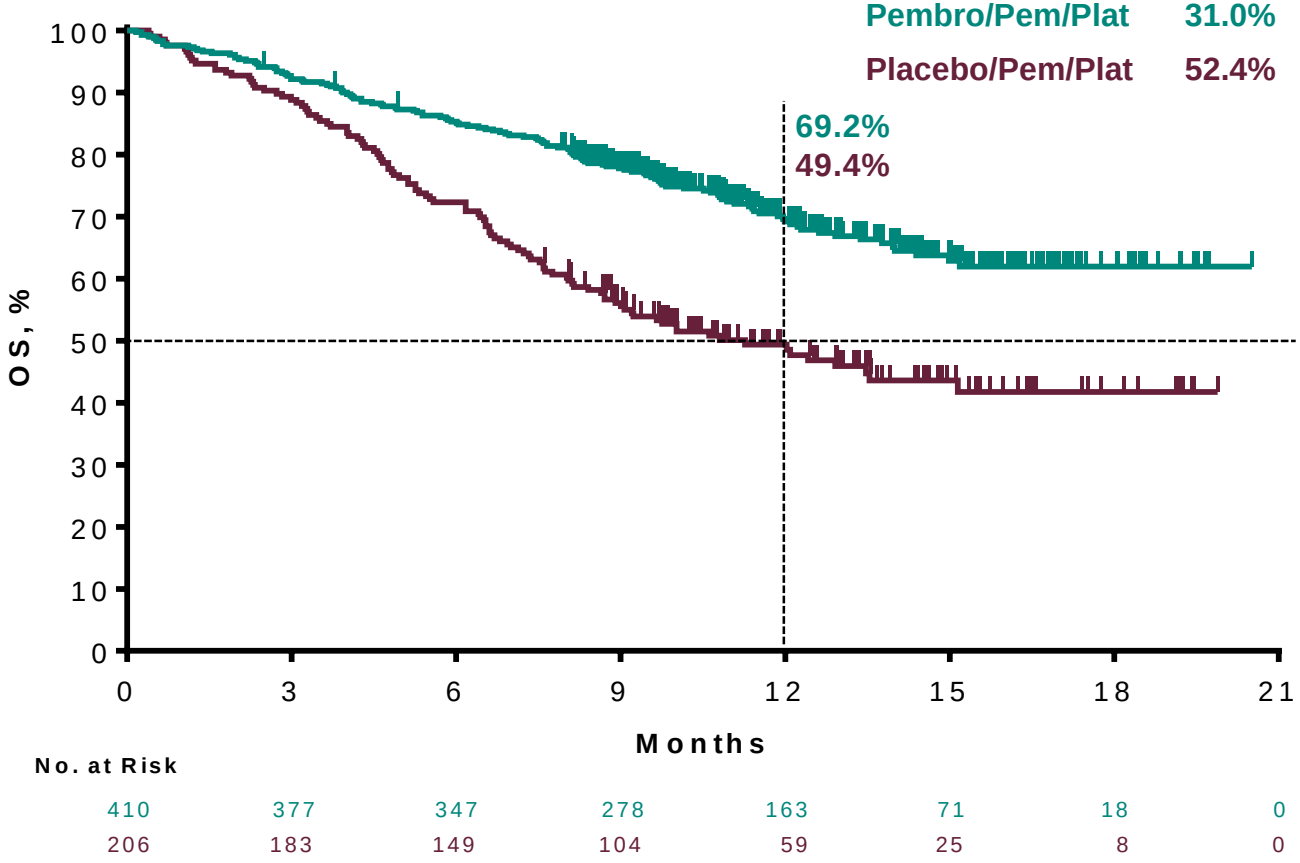
KEYNOTE-189 Study Design (NCT02578680)



[a]Percentage of tumor cells with membranous PD-L1 staining assessed using the PD-L1 IHC 22C3 pharmDx assay. [b]Patients could crossover during the induction or maintenance phases. To be eligible for crossover, PD must have been verified by blinded, independent central radiologic review and all safety criteria had to be met.

Overall Survival, ITT

	Events	HR (95% CI)	*P*
Pembro/Pem/Plat	31.0%	0.49 (0.38-0.64)	<0.00001
Placebo/Pem/Plat	52.4%		



Median (95% CI)
NR (NE-NE)
11.3 mo (8.7-15.1)

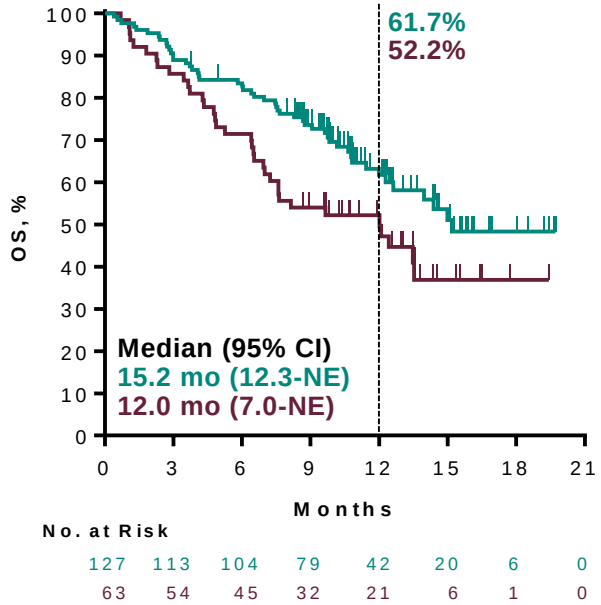
ISTITUTO NAZIONALE
PER LO STUDIO
E LA CURA DEI TUMORI

Data cutoff date: Nov 8, 2017.

Overall Survival by PD-L1 TPS

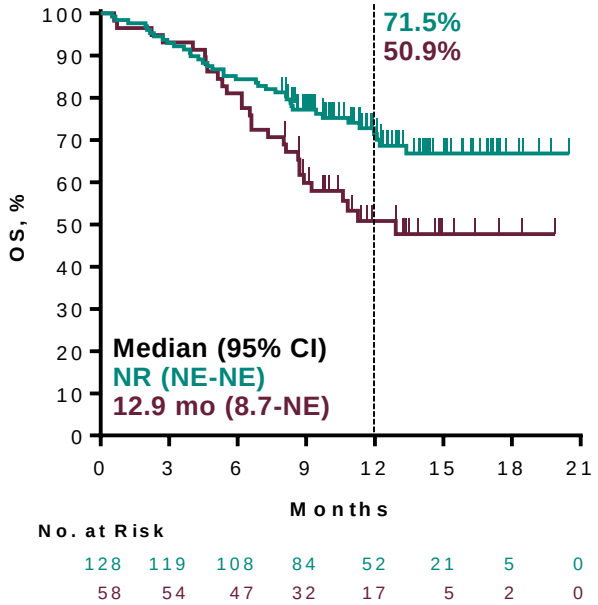
TPS <1%

	Events	HR (95% CI)	P[a]
Pembro/Pem/Plat	38.6%	0.59 (0.38-0.92)	0.0095
Placebo/Pem/Plat	55.6%		



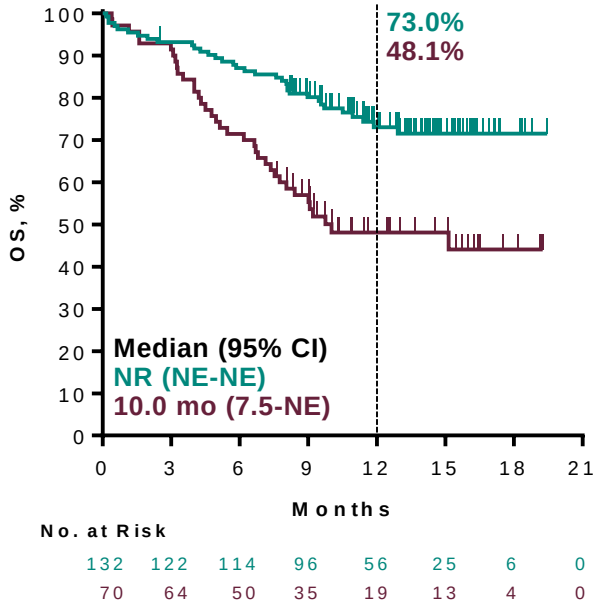
TPS 1-49%

	Events	HR (95% CI)	P[a]
Pembro/Pem/Plat	28.9%	0.55 (0.34-0.90)	0.0081
Placebo/Pem/Plat	48.3%		



TPS ≥50%

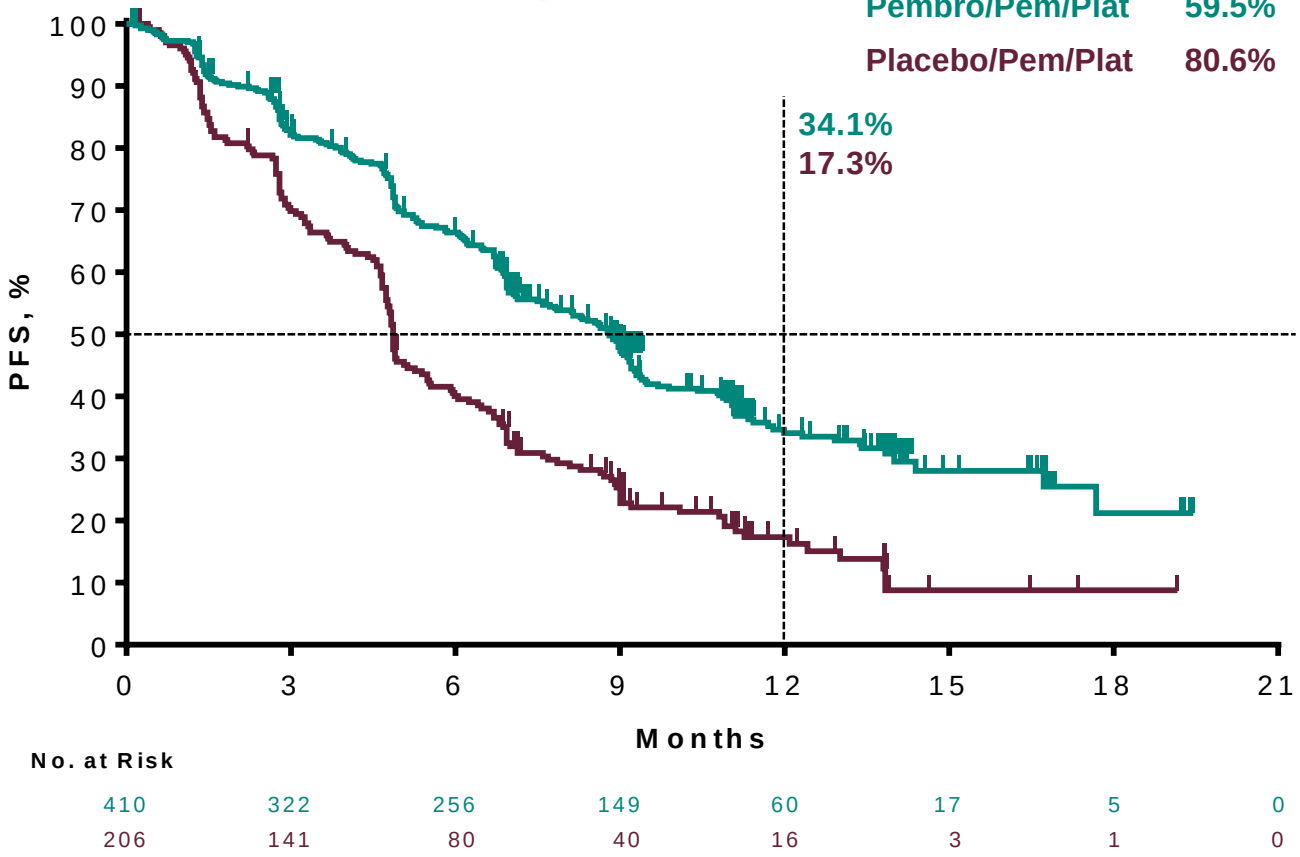
	Events	HR (95% CI)	P[a]
Pembro/Pem/Plat	25.8%	0.42 (0.26-0.68)	0.0001
Placebo/Pem/Plat	51.4%		



[a]Nominal and one-sided. Data cutoff date: Nov 8, 2017.

Progression-Free Survival, ITT
(RECIST v1.1, BICR)

	Events	HR (95% CI)	*P*
Pembro/Pem/Plat	59.5%	0.52 (0.43-0.64)	<0.00001
Placebo/Pem/Plat	80.6%		



Median (95% CI)
8.8 mo (7.6-9.2)
4.9 mo (4.7-5.5)

No. at Risk

410	322	256	149	60	17	5	0
206	141	80	40	16	3	1	0

BICR, blinded, independent central review. Data cutoff date: Nov 8, 2017.

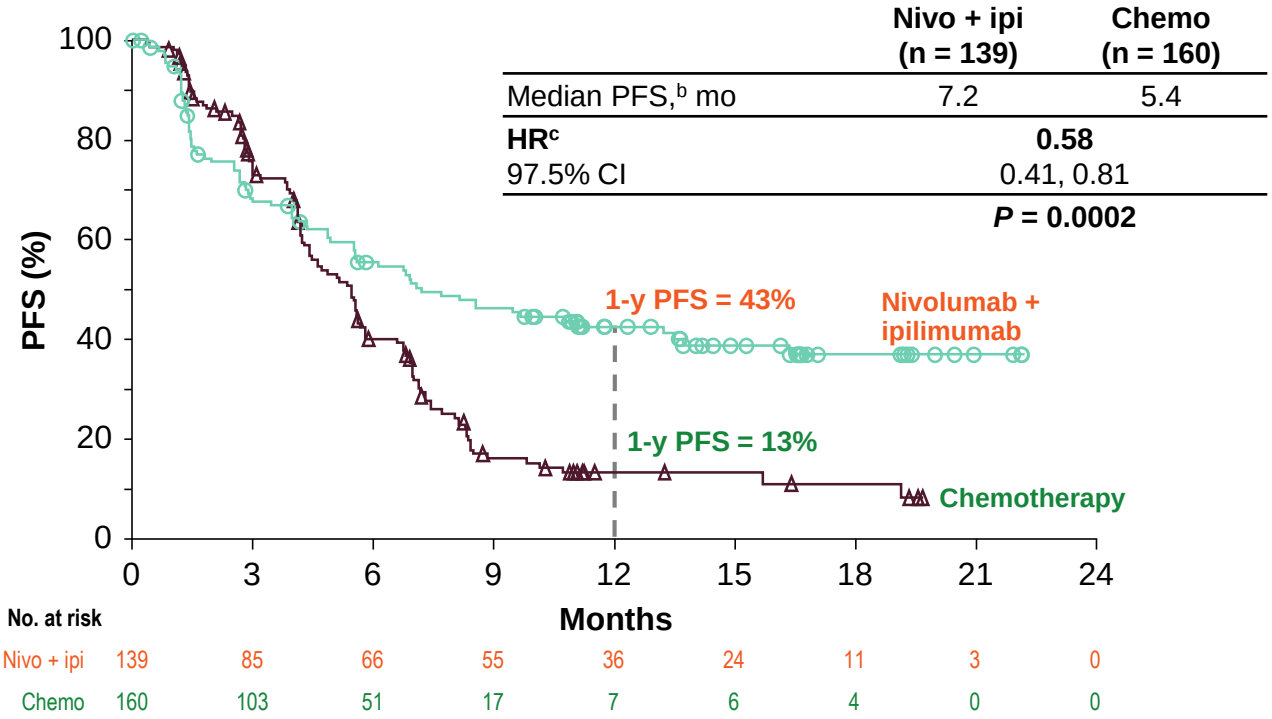
ORIGINAL ARTICLE

Nivolumab plus Ipilimumab in Lung Cancer with a High Tumor Mutational Burden

Matthew D. Hellmann, M.D., Tudor-Eliade Ciuleanu, M.D., Adam Pluzanski, M.D., Jong Seok Lee, M.D., Gregory A. Otterson, M.D., Clarisse Audigier-Valette, M.D., Elisa Minenza, M.D., Helena Linardou, M.D., Sjaak Burgers, M.D., Pamela Salman, M.D., Hossein Borghaei, D.O., Suresh S. Ramalingam, M.D., et al.

April 16, 2018
DOI: 10.1056/NEJMoa1801946

Co-primary Endpoint: PFS With Nivolumab + Ipilimumab vs Chemotherapy in Patients With High TMB (≥10 mut/Mb)[a]



	Nivo + ipi (n = 139)	Chemo (n = 160)
Median PFS,[b] mo	7.2	5.4
HR[c]	**0.58**	
97.5% CI	0.41, 0.81	
	***P* = 0.0002**	

- In patients with TMB <10 mut/Mb treated with nivo + ipi vs chemo, the HR was 1.07 (95% CI: 0.84, 1.35)[d]

[a]Per blinded independent central review (BICR); median (range) of follow-up in the co-primary analysis population was 13.6 mo (0.4, 25.1) for nivo + ipi and 13.2 mo (0.2, 26.0) for chemo; [b]95% CI: nivo + ipi (5.5, 13.2 mo), chemo (4.4, 5.8 mo); [c]95% CI: 0.43, 0.77 mo; [d]The *P*-value for the treatment interaction was 0.0018

Three main phenotypes and multiple uncertainties

Immune-excluded

CT+IO
CT→IO

Immune-desert

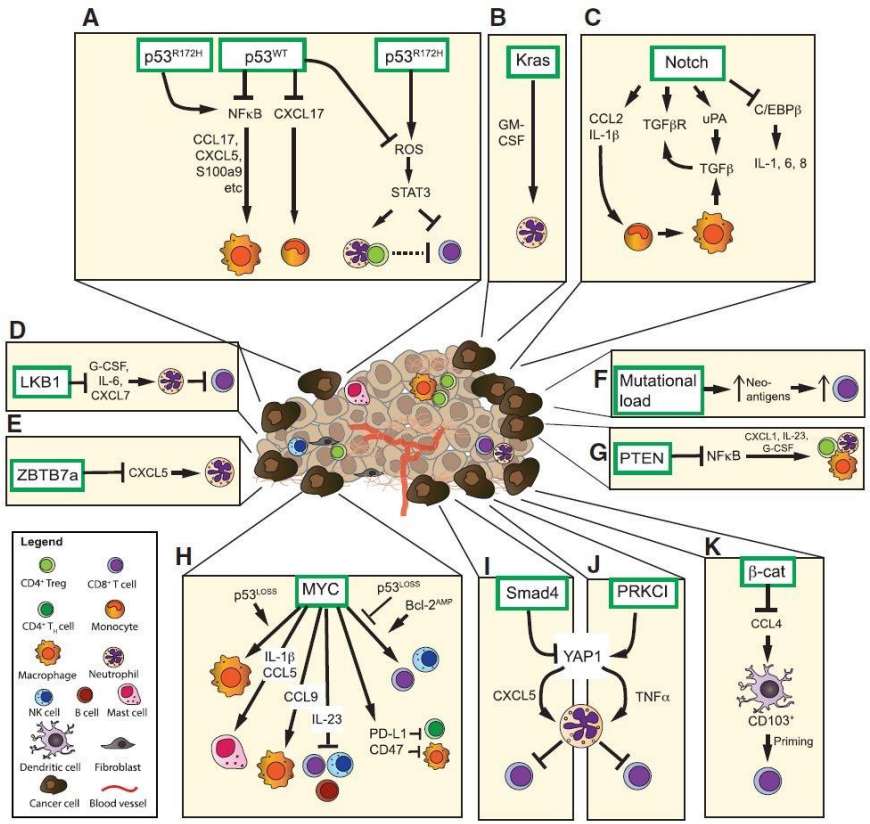
CT+IO
CT→IO

Inflamed

IO→CT
CT+IO

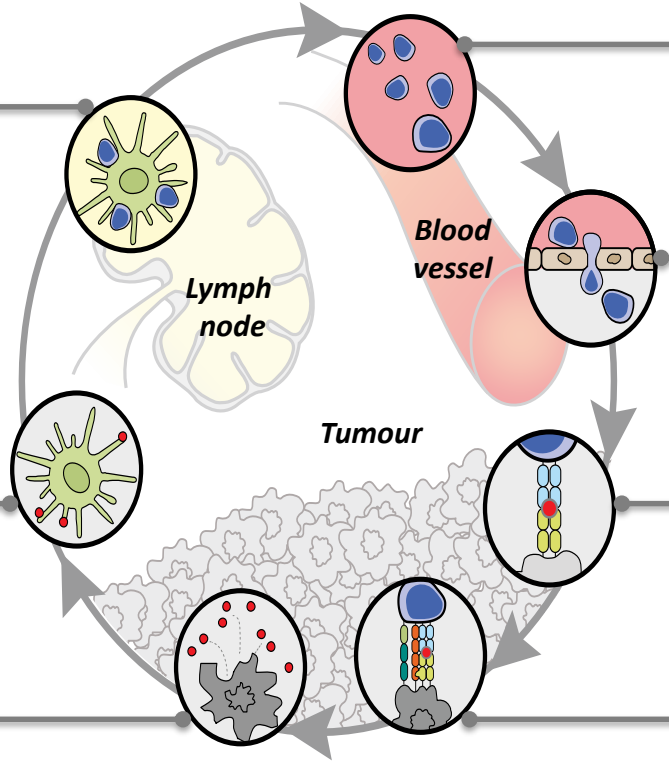
Can we increase the patient population that responds to Immunotherapy?

Genotype and immunephenotype



Wellenstein, MD, Cell 2018

Tumors Use Complex, Overlapping Mechanisms to Evade and Suppress the Immune System



3

Priming and Activation

CD28/B7.1, CD137/CD137L
OX40/OX40L, CD27/CD70
HVEM, GITR, IL-2, IL-12

CTLA4/B7.1
PD-L1/PD-1
PD-L1/B7.1 Prostaglandins

2

Cancer antigen presentation

TNF-α, IL-1
IFN-α, CD40L/CD40
CDN, ATP
HMGB1, TLR

IL-10, IL-4, IL-13

1

Release of cancer cell antigens

Immunogenic cell death

Tolergenic cell death

4

Trafficking of T cells to tumours

CX3CL1, CXCL9, CXCL1
CCL5

5

Infiltration of T cells into tumours

LFA1/ICAM1
Selectins

VEGF
Endothelin-B receptor

6

Recognition of cancer cells by T cells

T cell receptor

Reduced pMHC on cancer cells

7

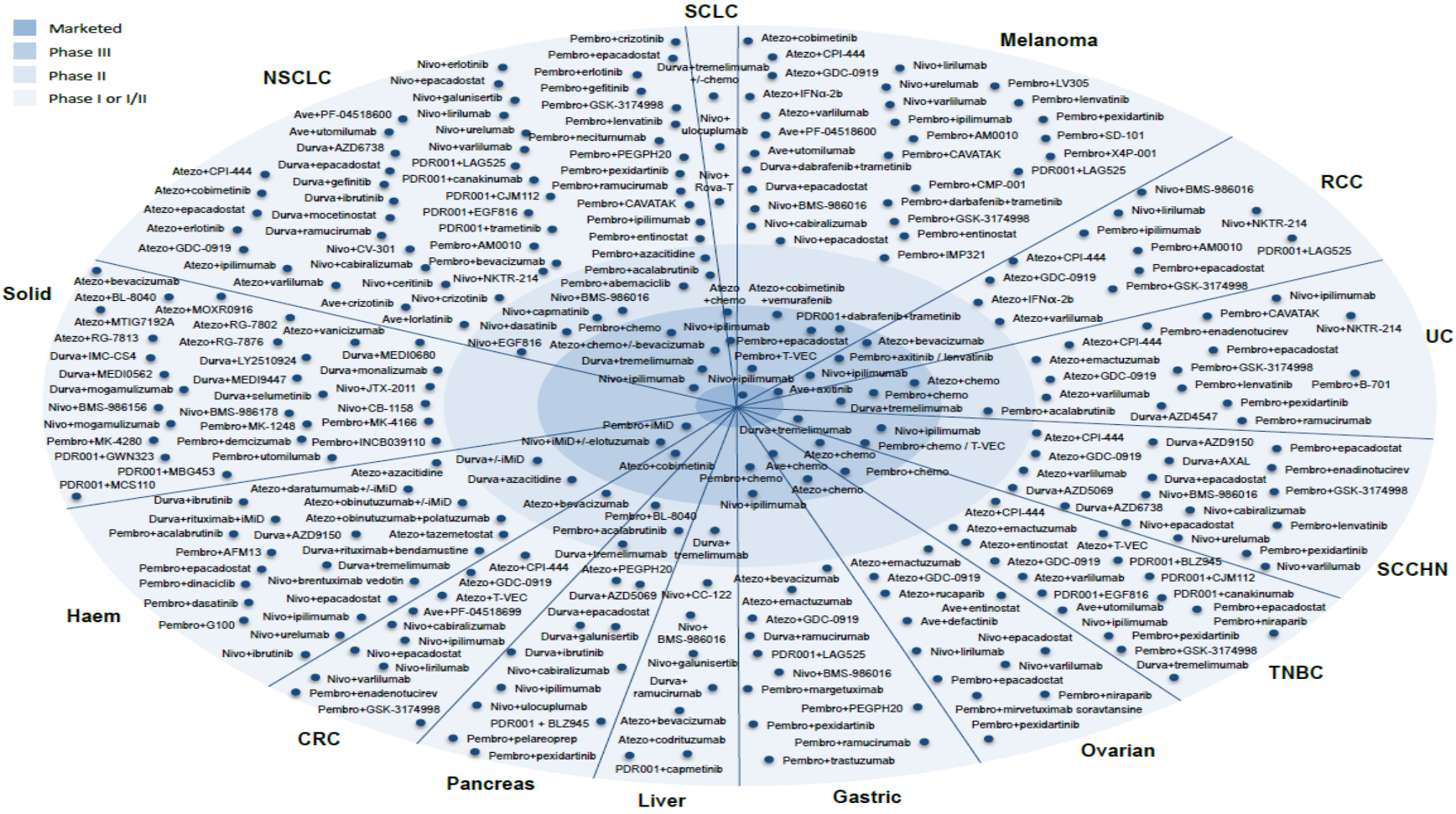
Killing of cancer cells

IFN-γ , T-cell granule content
TIM-3/phospholipids

PD-L1/PD-1	VISTA
PD-L1/B7.1	LAG-3
IDO	Arginase
TGF-β	MICA/MICB
BTLA	B7-H4

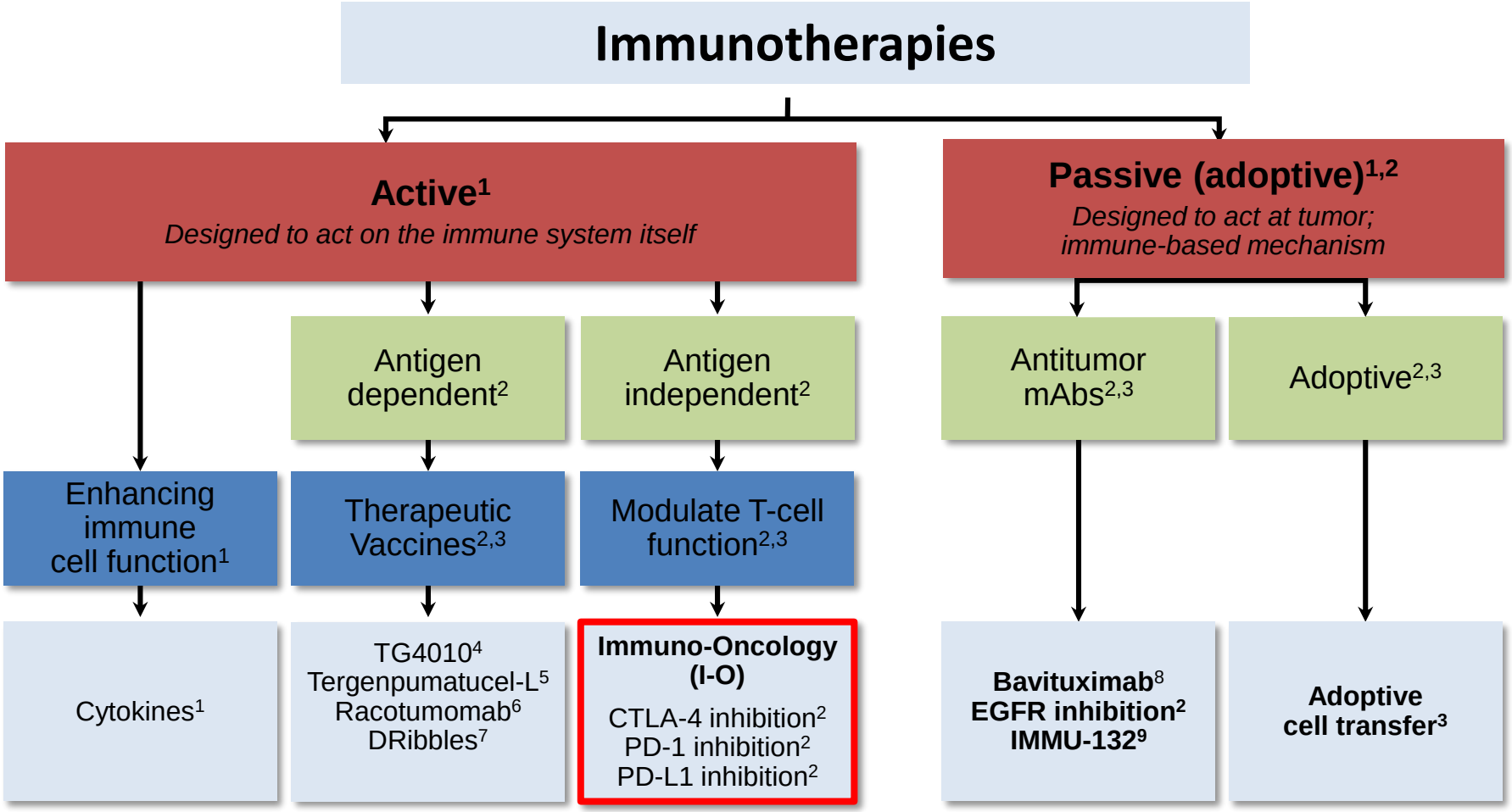
Chen DS and Mellman I. *Immunity.* 2013 Jul 25;39(1):1-10.

Drug Development for IO Drugs

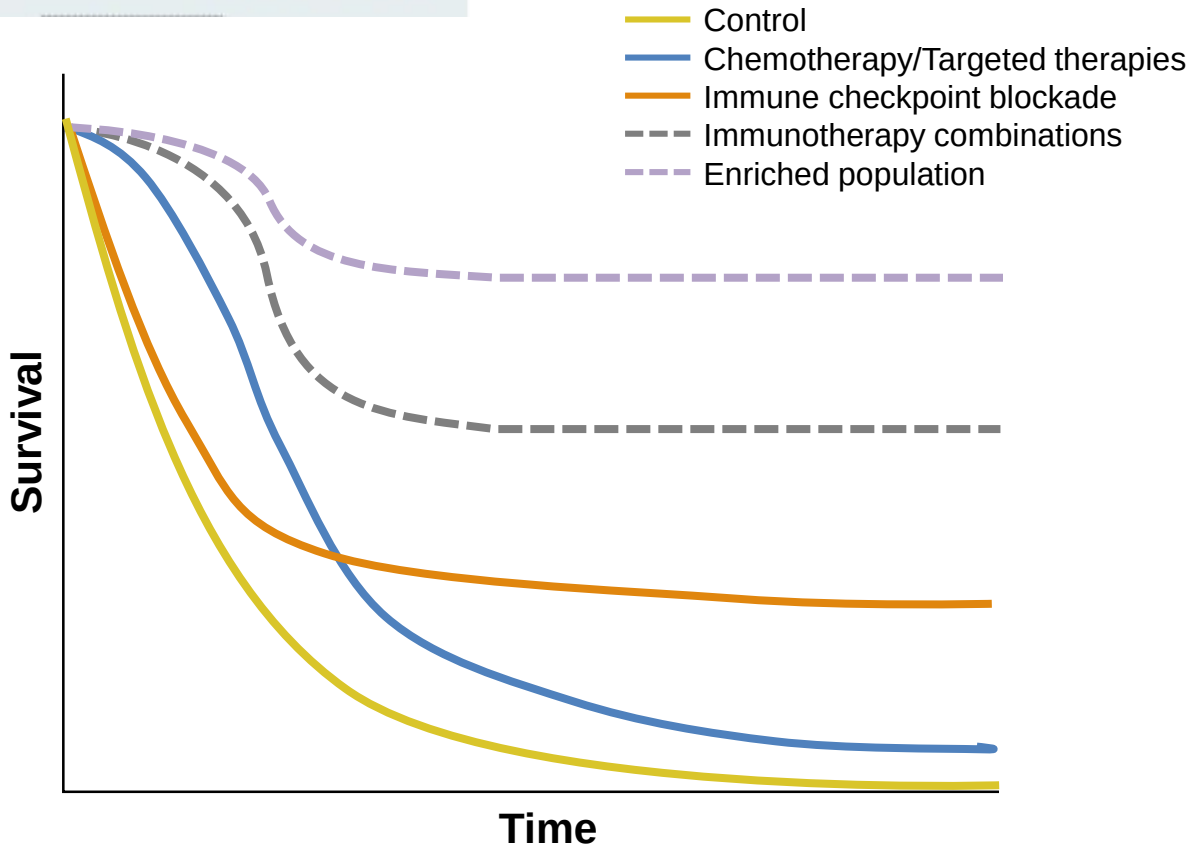


Chen and Mellman, Nature 2017

Investigational immunotherapeutic approaches in lung cancer



Conclusions: "The Promise"



1. Adapted from Ribas A, presented at WCM, 2013
2. Ribas A, et al. *Clin Cancer Res* 2012;18:336–341
3. Drake CG. *Ann Oncol* 2012;23(suppl 8):viii41–viii46

"Time for me is double-edged: Every day brings me further from the low of my last cancer relapse, but every day also brings me closer to the next cancer recurrence --- and eventually, death."

Paul Kalanithi, MD'07 Author of "*When Breath Becomes Air*"

Neurosurgeon, Writer, Patient with *EGFR* mutant lung cancer

Marina Chiara Garassino

President

1 : 10


Rossana Berardi
Ketta Lo Russo
Valentina Guarneri
Marina Garassino
Rita Chiari
Fabiana Cecere
Nicla La Verde
Erika Martinelli
Laura Locati
QUESTIONI di MANAGEMENT al FEMMINILE in ONCOLOGIA
16 Settembre 2016
SDA Bocconi • Milano
RESEARCH & COMMUNICATION
12•13 Maggio 2016
WO
Oncology

2016 - Our mission

Doing something for ourselves

Some activities

Inspiring women

Courses with famous journalists

University of Economics «Luigi Bocconi»
Leadership programmes

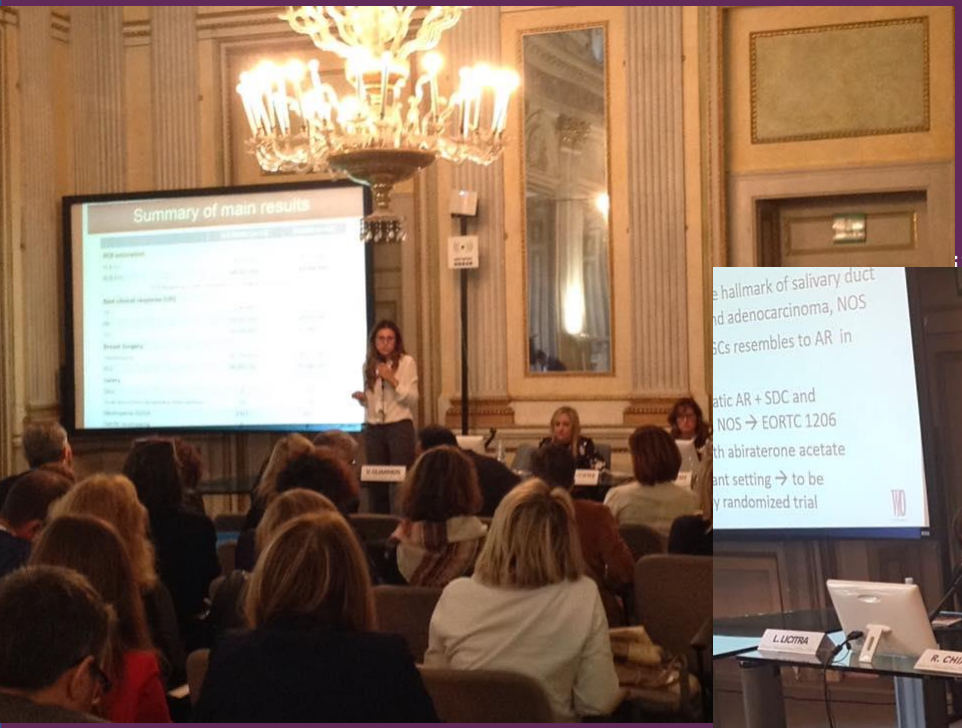
Communication Skills

Psycological skills

Patient communication

Social network skills

POST ESMO-2017



Rome, Parliament, 2017- Women who care

In our diversities

We learnt the power of sharing

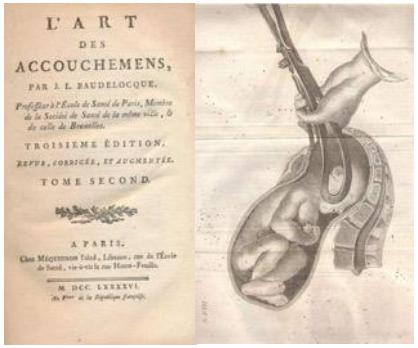

We are in sharing era

FEMINIZATION AND RECOMPOSITION OF PROFESSIONAL HIERARCHIES: CHALLENGES AND OPPORTUNITIES FOR MEDICINE

Francesco Panese, Professor of Sociology, UNIL

MEDICINE MEN MARGINALIZING MEDICINE WOMEN
A PROBLEM AS OLD AS MODERN MEDICINE

Men	Women
"Professors"	"Modest females"
"Instruments" (forceps)	"Hands"
"Ignorance"	"Experience"
"Impatience"	"Patience"
"Cruelty"	"Compassion"
…	…



BAUDELOCQUE (Jean-Louis). L'Art des accouchemens. Paris : Méquignon l'aîné, **1796**

John Blunt [i.e. S.W. Fores], Man-midwifery dissected ; or, the obstetric family-instructor ., London: **1793**. Wellcome images.

BOURSIER DU COUDRAY, Angelique Marguerite le (1714–1794) Abrégé de l'art accouchements, **1773**.

STEREOTYPICAL GENDER DIVISION

“Cure” vs “Care”

Pablo Picasso (1881-1973). Science and charity. **1897**.
Museo Picasso, Barcelona, Spain.

A physician examining a child, who is being comforted by a nurse in the ward of a childrens' hospital. J.Löwy, 1901, after I. Knopp, **1892**.

STEREOTYPICAL GENDER DIVISION

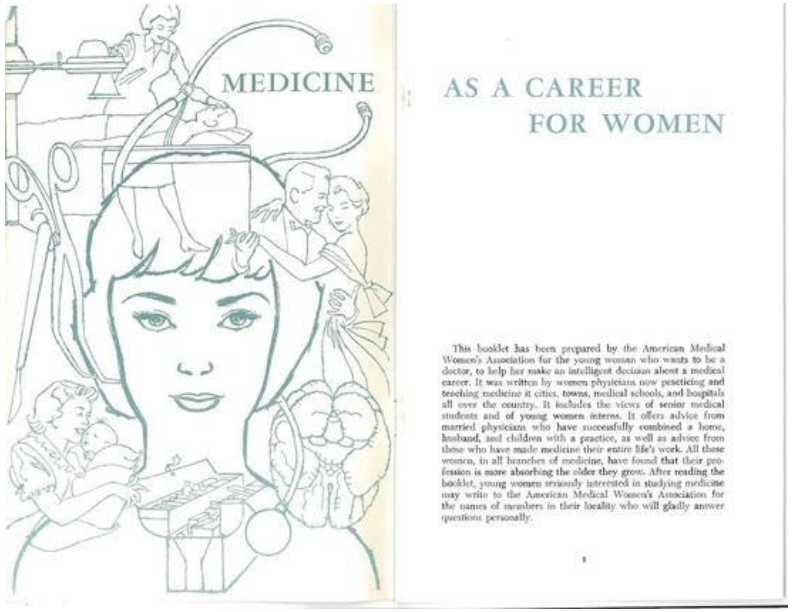
A long and difficult *REAL* integration

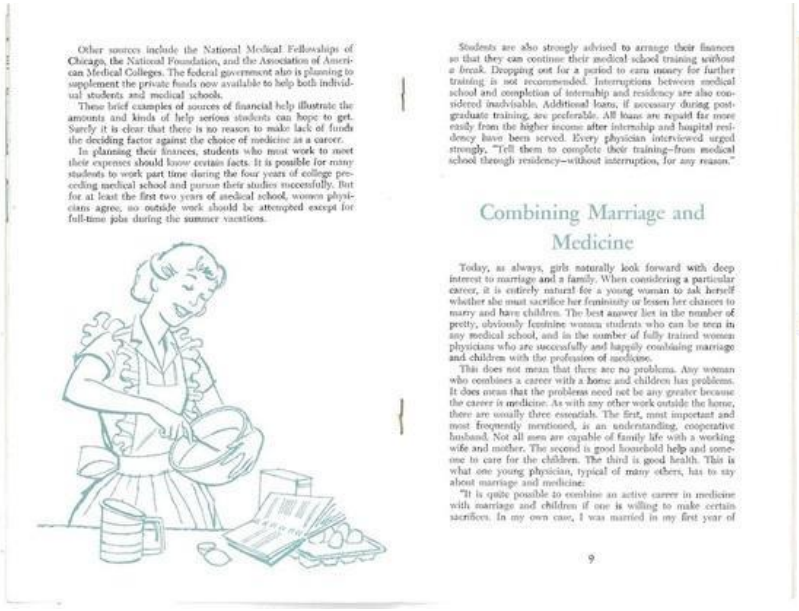
New England Female Medical College, originally Boston Female Medical College, founded in **1848**. The oldest medical school in the United States exclusively for women.

First coeducational class of women admitted to Harvard Medical School, **1945**.

Countway Library of Medicine

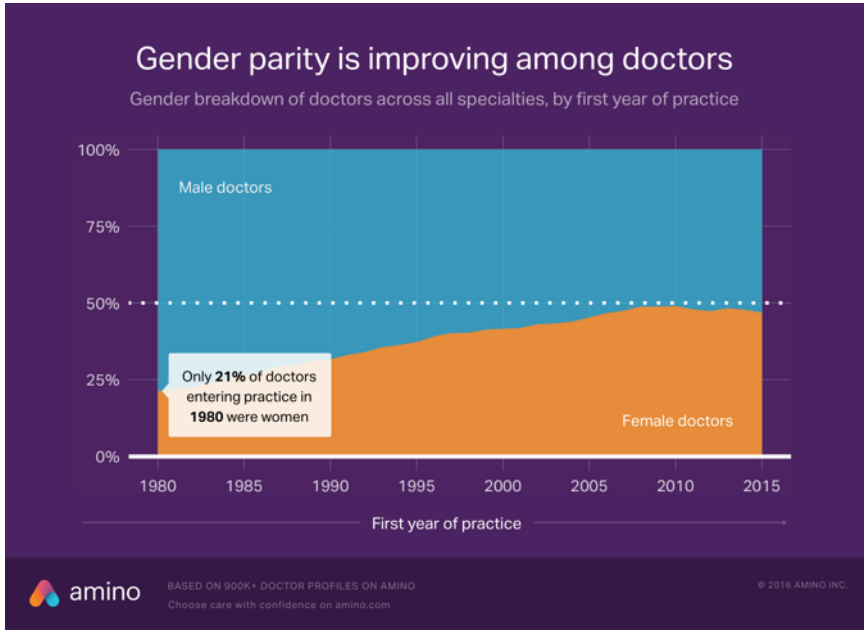
STEREOTYPES JUSTIFYING EXCEPTION
MULTI-ROLE, MULTI-TASKING AND HEROISM



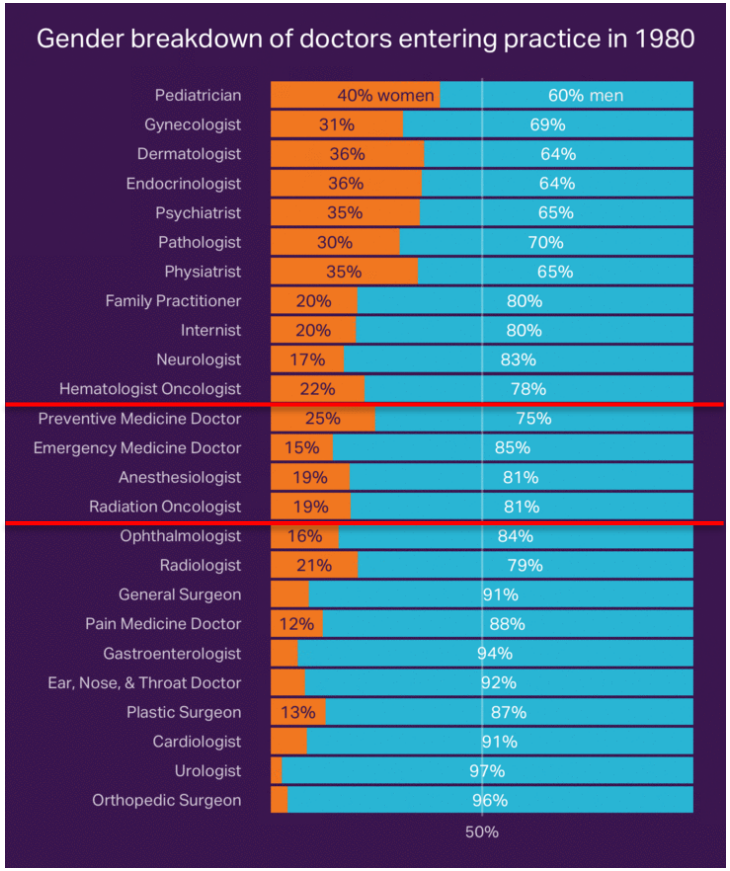


Ruth Morris Bakwin (1898-1985) (MD 1923 Cornell University Medical College). « Pediatrician, researcher, writer, wife, mother and art collector ». Pamphlet of edited papers (ca. 1984). Weill Cornell Medicine library.

GENDER IN MEDICINE – POSITIVE DIFFERENTIAL INCREASE…

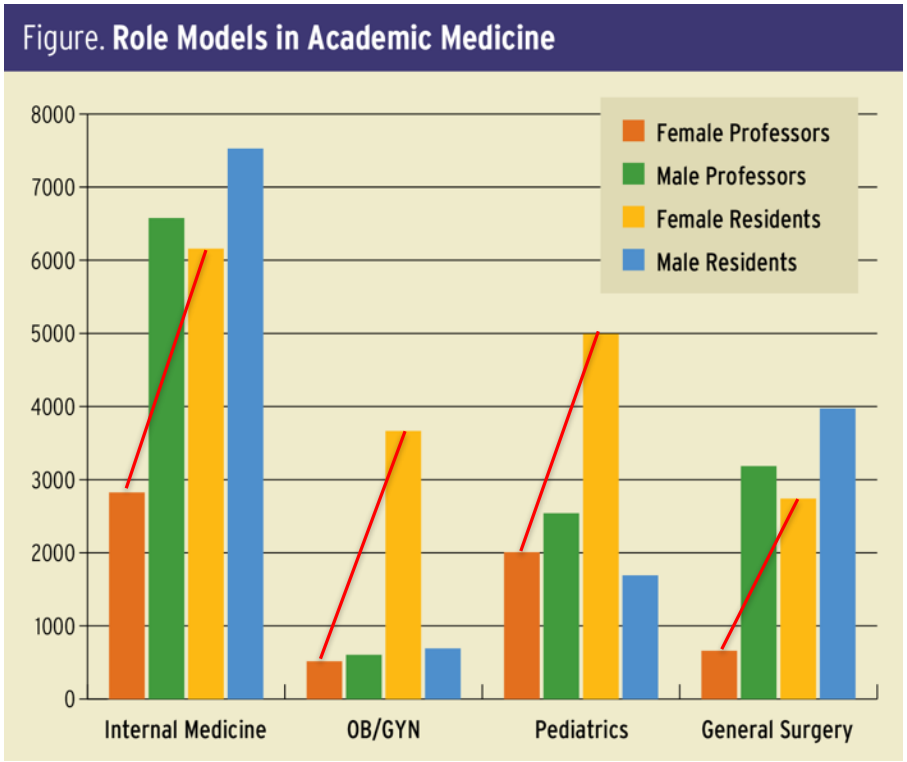


Hannah Levy (2016) How the gender gap is shifting in medicine, by specialty. © Amino digital health company.
https://amino.com/blog/how-the-gender-gap-is-shifting-in-medicine-medical-specialties-by-gender/



An ESMO Initiative

GENDER IN MEDICINE – …NEGATIVE GAPS



"Distribution of U.S. Medical School Faculty by Sex, Race/Ethnicity, Tenure Status, and Department". Resident data based on 2014-2015 AAMC "Report on Residents".

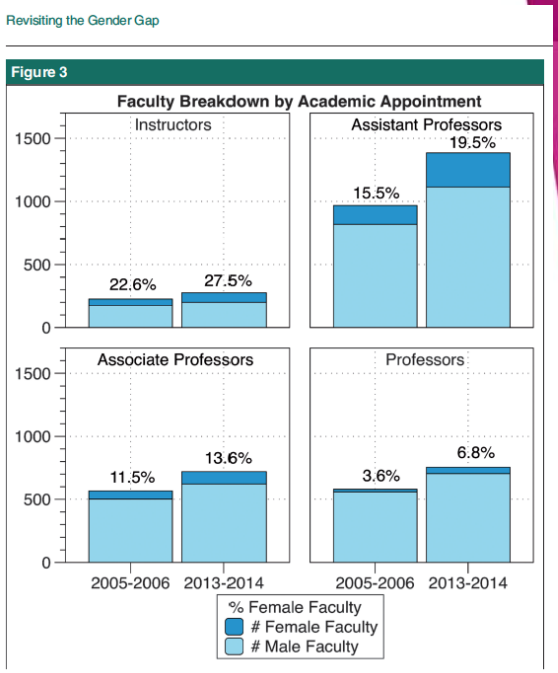


Chart showing the proportion of female orthopaedic surgery instructors, assistant professors, associate professors, and professors for the academic years of 2005 to 2006 and 2013 to 2014.

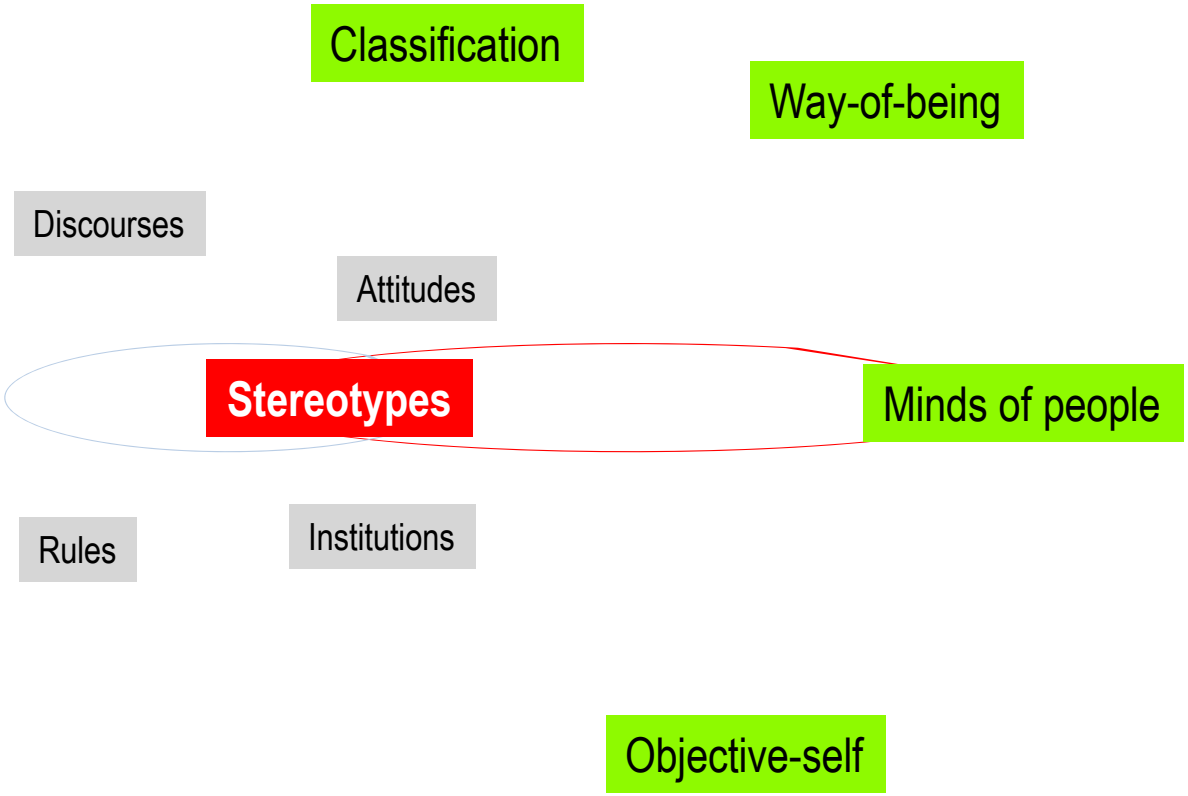
Journal of the American Academy of Orthopaedic Surgeons. Publish Ahead of Print(), OCT 2018
DOI: 10.5435/JAAOS-D-17-00686, PMID: 30278014
Issn Print: 1067-151X
Publication Date: 2018/10/01

Revisiting the Gender Gap in Orthopaedic Surgery: Investigating the Relationship Between Orthopaedic Surgery Female Faculty and Female Residency Applicants

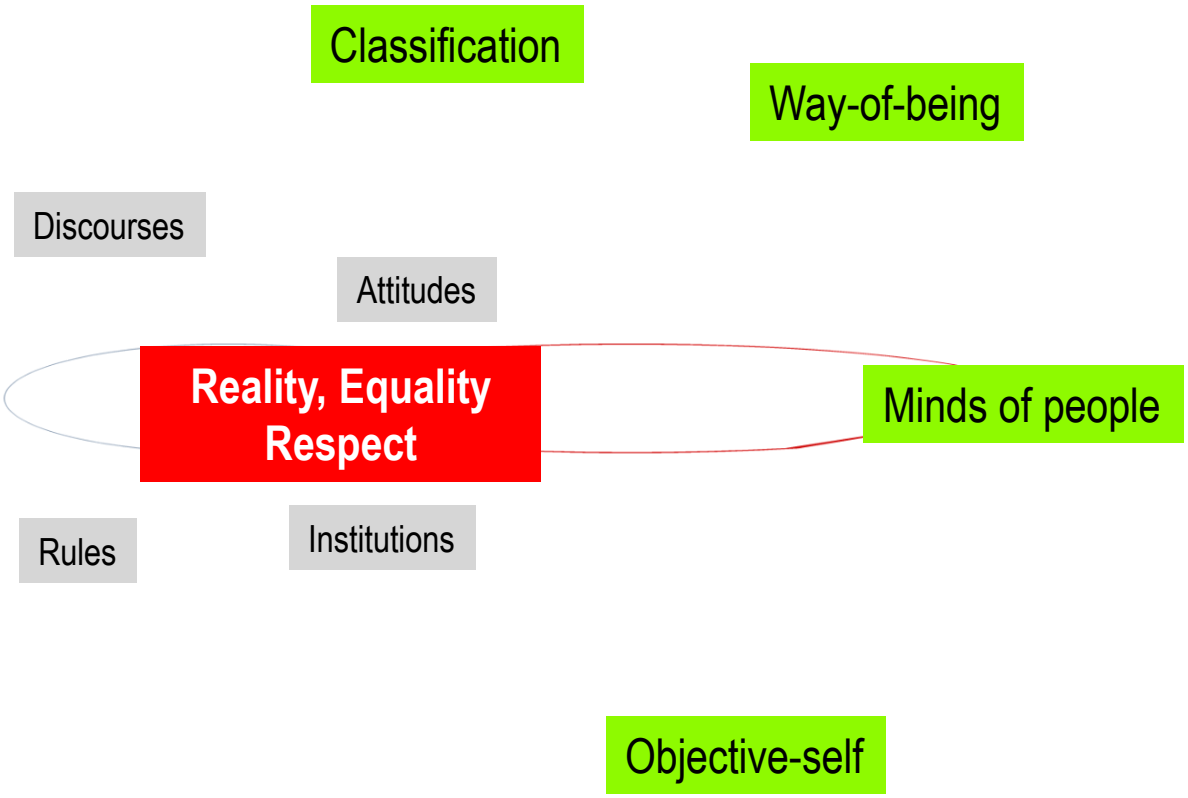
Alana M. Munger; Nathanael Heckmann; Braden McKnight; Marie N. Dusch; George F. Hatch; Reza Omid

An ESMO Initiative

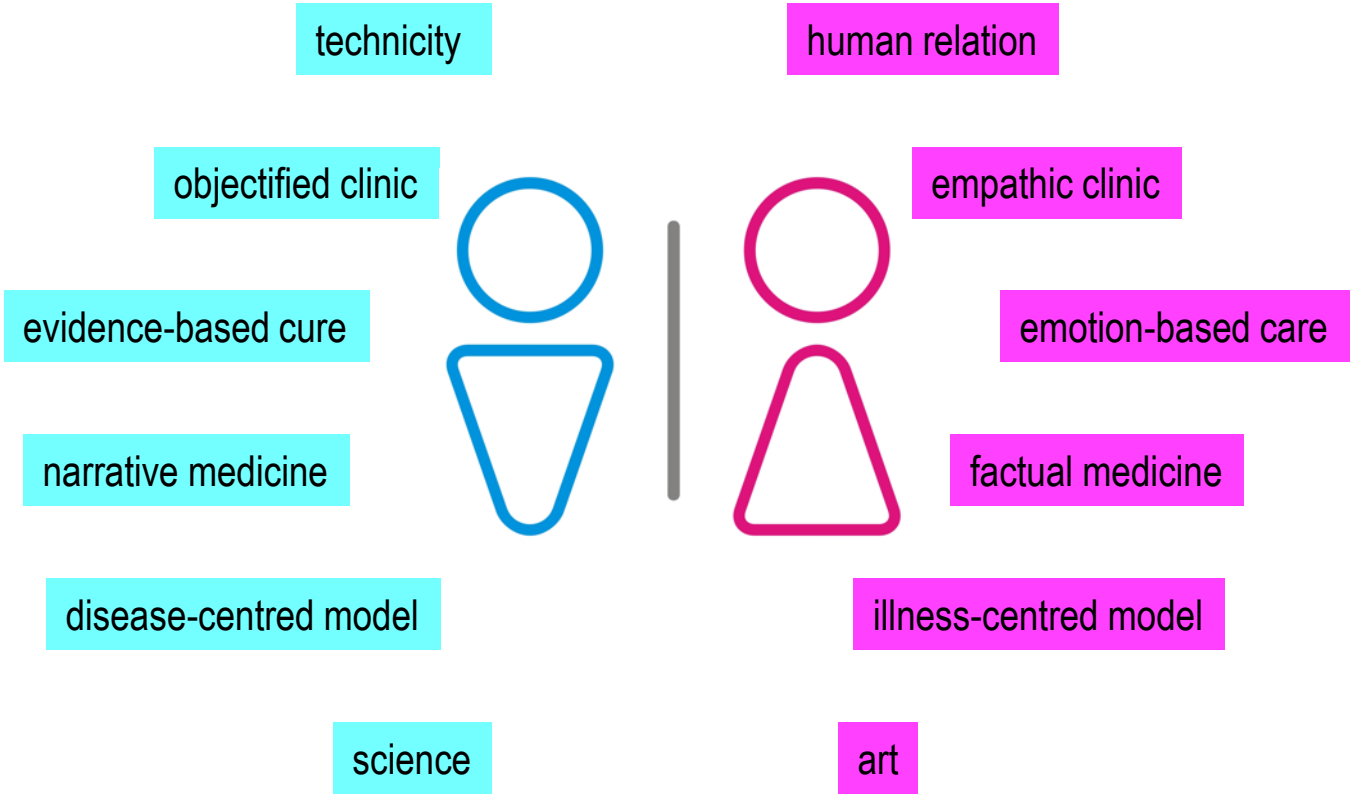
WHAT STEREOTYPES DO ?
"STEREOTYPES AS CLASSIFYING MACHINE"



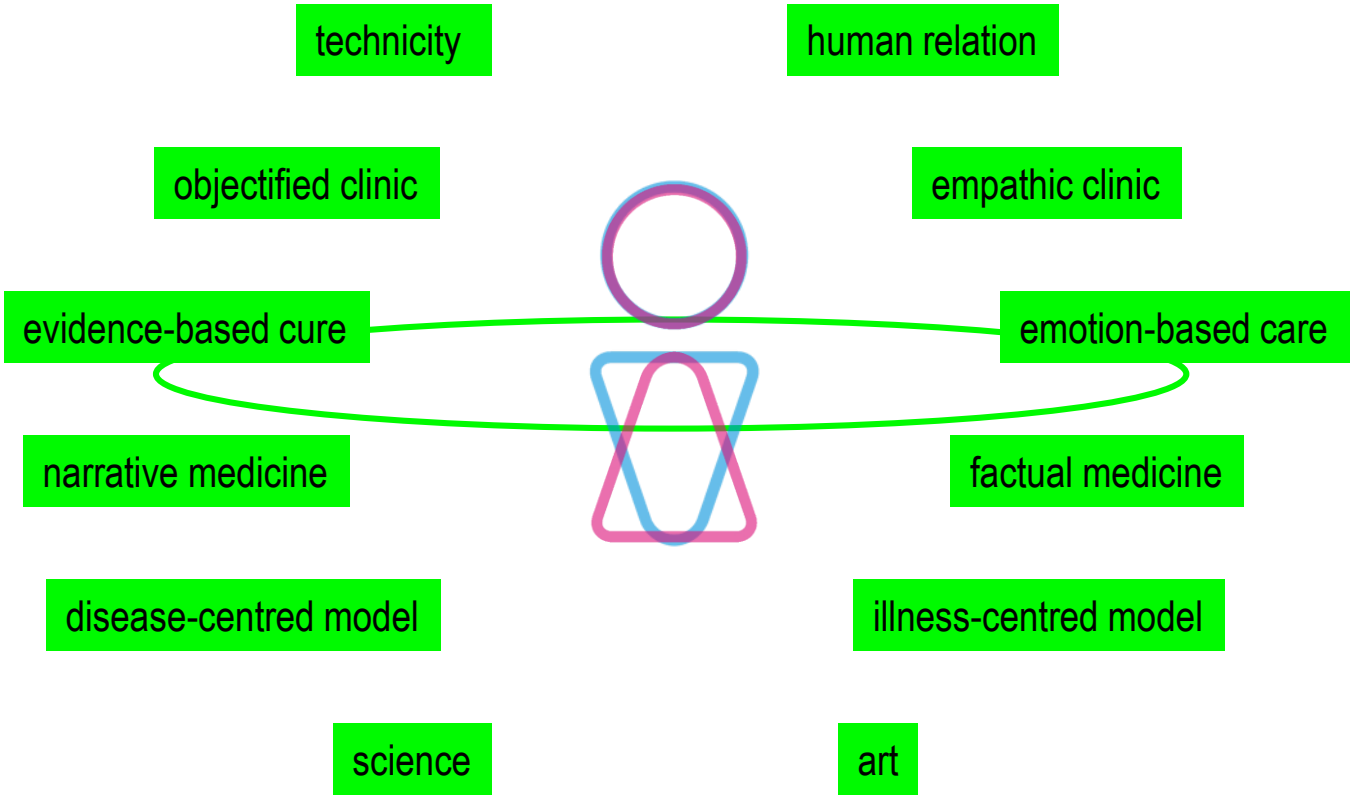
CHALLENGING STEREOTYPES
"STEREOTYPES RECTIFICATION MACHINE"



CHALLENGING STEREOTYPICAL OPPOSITIONS IN CLINICAL PRACTICE



DIFFERENCES ⇒
COMPLEMENTARITIES
COMPLEMENTARITIES ≠ HIERARCHIES



BIG ISSUE : CHALLENGING STEREOTYPES

Françoise Mouly. The New Yorker. March 27, 2017