PLATAFORMA DE ONCOLOGIA

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ONCOLOGÍA INTEGRADA: EQUIPO MULTIDISCIPLINARIO Y ENFOQUE PERSONAL

FUNDACIÓN TEDECA
PLATAFORMA DE ONCOLOGÍA

HOSPITAL QUIRÓN Torrevieja

UCAM
UNIVERSIDAD CATÓLICA SAN ANTONIO

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PLATFORM OF ONCOLOGY
DEVELOPMENT OF SELECT UNIQUE PROGRAMS

Isolated limb perfusion/liver perfusion with hyperthermia & TNF Monitoring
Intra-arterial chemotherapy (CNS, hepatic, limbs)
Cell therapy: Adoptive immunotherapy (TIL), Dendritic cell vaccines, High Dose IL_2
High dose chemotherapy and autologous hematopoietic progenitor transplant
Intra-operative radiotherapy
Radio-metabolic therapy. PET imaging evaluation of response
Therapeutic biopsy procedure and tumor tissue bank. Molecular monitoring of MRD
Full time Psico-oncologic care, Dignity therapy
New drug (TKIs) monitoring. Pharmacokinetic-dynamic guided chemotherapy
One step therapy in breast cancer
Hyperthermic peritoneal chemotherapy (HIPEC) in carcinomatosis
Assisted robotic surgery (Da Vinci)
Re-irradiation, Concurrent Chemoradiotherapy, IMRT, IMGRT, Stereotactic Radiosurgery
Surgical rescue for oligometastatic and residual disease. Pelvic exenteration.
Gene Target Therapy, Tumor profiling, and signature guided chemotherapy
Radiofrequency ablation, Photodynamic surgery, Electrochemotherapy

PLATFORM OF ONCOLOGY: 10 YEAR RESULTS IN FREQUENT TUMORS

CHARACTERISTICS	COLORECTAL		BREAST		LUNG	
	LOCAL	METS	LOCAL	METS	LOCAL	METS
NUMBER	68	148	176	107	90	133
Median AGE	69	61.5	53	52	64	58
≥ 70 y.o. (%)	35.3	11.5	9.7	6.5	23.6	12.8
Residency ≥ 150 Km.(%)	55.8	43.2	40.9	35.5	38.9	31.2
E.U. (%)	30.8	10.1	26.2	14	22.2	13.8
Other in Spain (%)	13.2	39.8	33	50.5	38.9	55
Co-morbidity, any (%)	54.7	53.5	37.5	35	83.3	68.8
Severe (%)	42.6	16.9	36,9	25.2	44.5	38.5
Second CANCER (%)	22.1	12.8	25.6	15	20.8	18.3
MEDIAN SURVIVAL TIME	80%≥6y	29 mo	90%≥6y	36 mo	45%≥6y	13 mo.
DFS (%)	91.2	12.2	92.6	6.5	44.4	3,2

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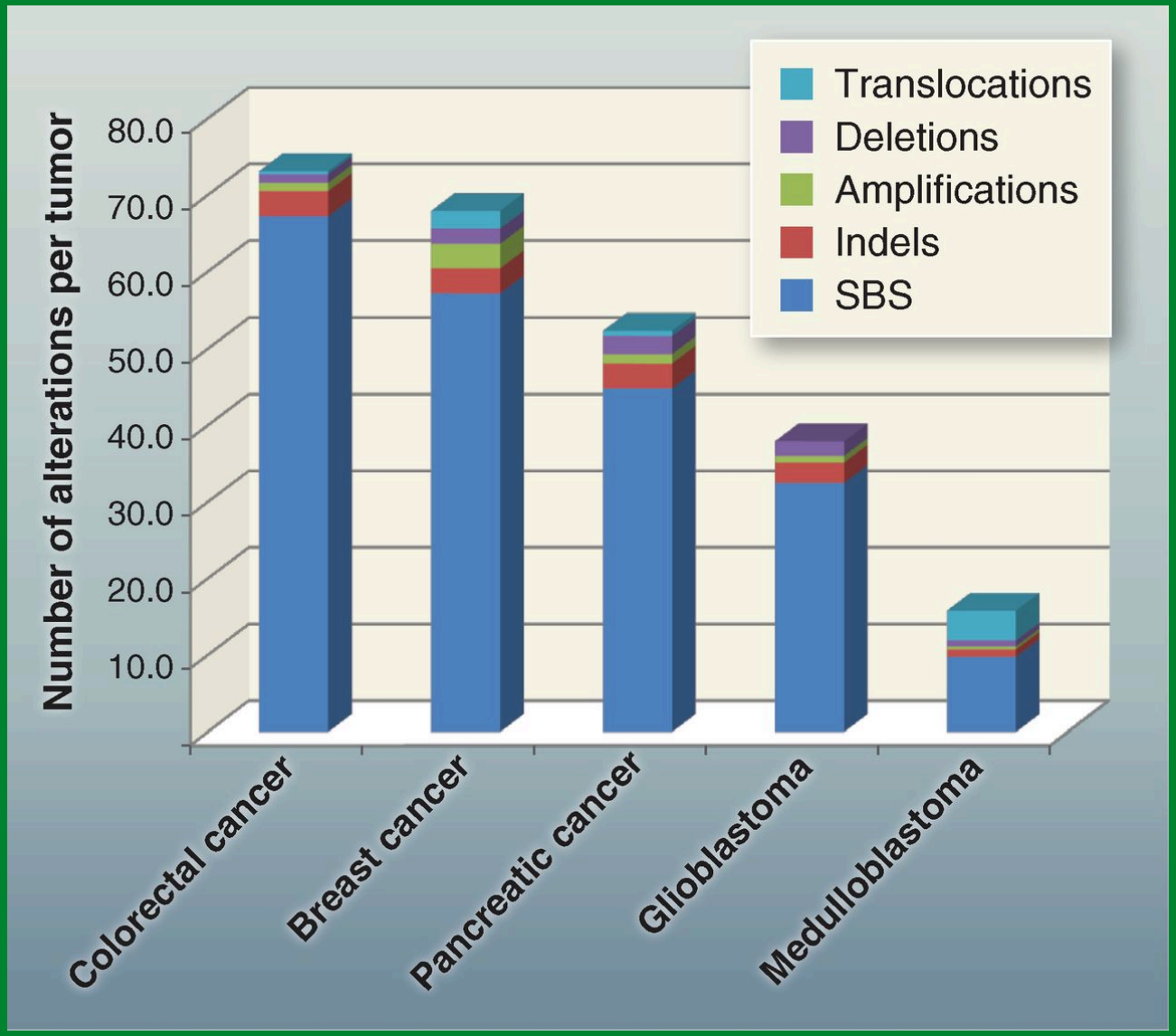
Fig. 3 Total alterations affecting protein-coding genes in selected tumors.



B Vogelstein et al. Science 2013;339:1546-1558

Science
AAAS

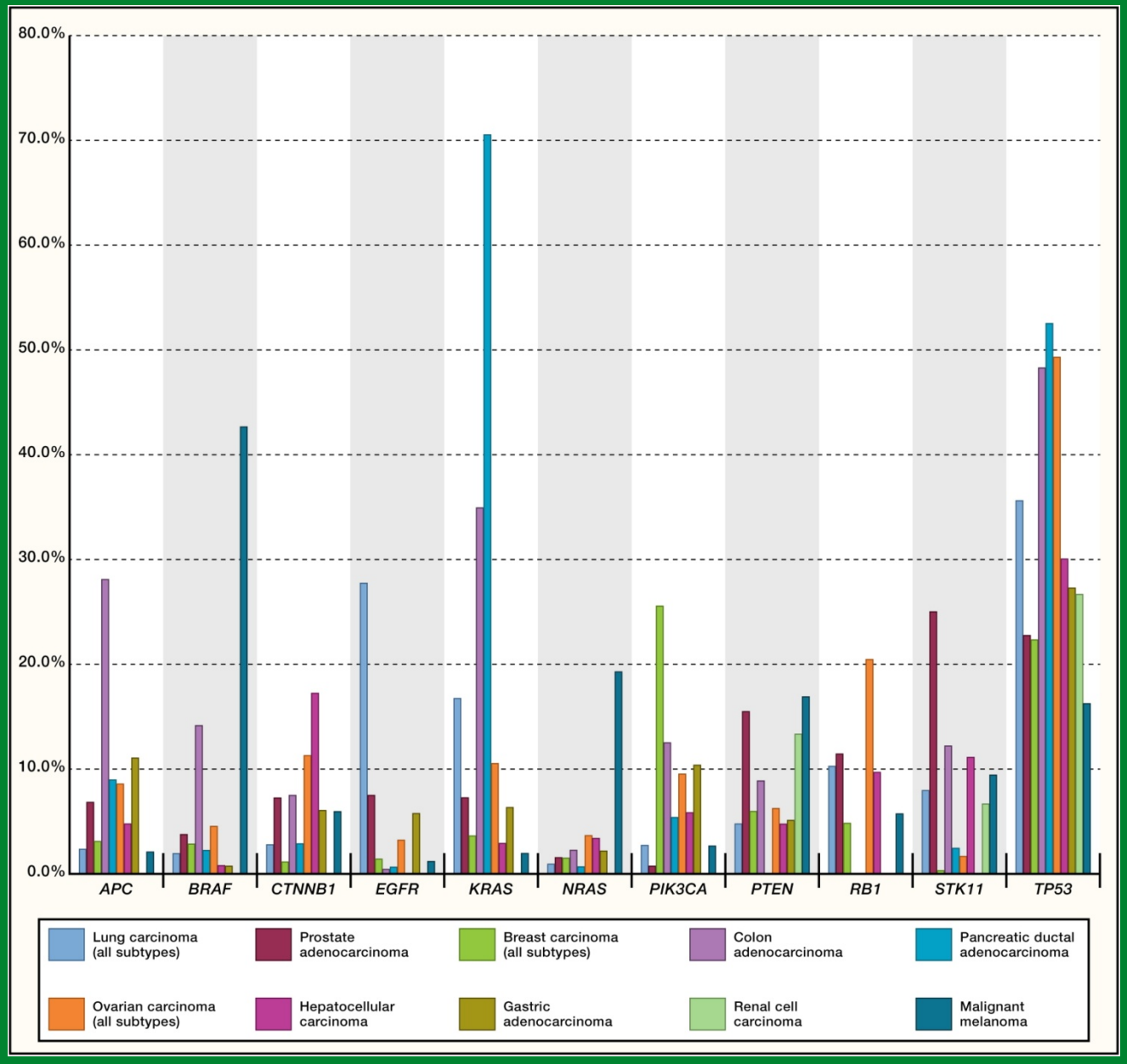
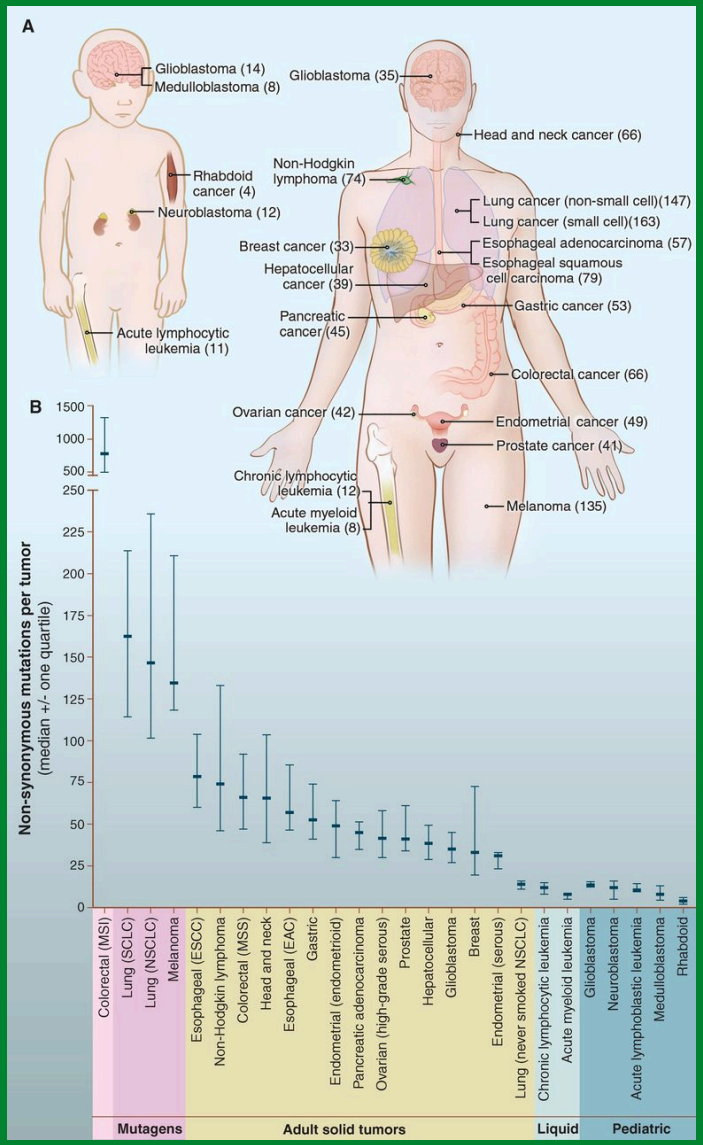

80.0%
70.0%
60.0%
50.0%
40.0%
30.0%
20.0%
10.0%
0.0%
APC
BRAF
CTNNB1
EGFR
KRAS
NRAS
PIK3CA
PTEN
RB1
STK11
TP53
Lung carcinoma (all subtypes)
Prostate adenocarcinoma
Breast carcinoma (all subtypes)
Colon adenocarcinoma
Pancreatic ductal adenocarcinoma
Ovarian carcinoma (all subtypes)
Hepatocellular carcinoma
Gastric adenocarcinoma
Renal cell carcinoma
Malignant melanoma

Fig. 1 Number of somatic mutations in representative human cancers, detected by genome-
sequencing studies.



B Vogelstein et al. Science 2013;339:1546-1558

THE CANCER GENOME PROJECT 2008

INTERNATIONAL CANCER GENOME CONSORTIUM

To study 50 cancers: 500 tumors each:

1.Gene expression profiling: coding regions of exons, conserved regions, regulatory elements, split-site regions, non repetitive unique regions, etc.

2.RNA sequencing: pre-mRNA consensus, micro-RNA, etc.

3.DNA methylation analysis

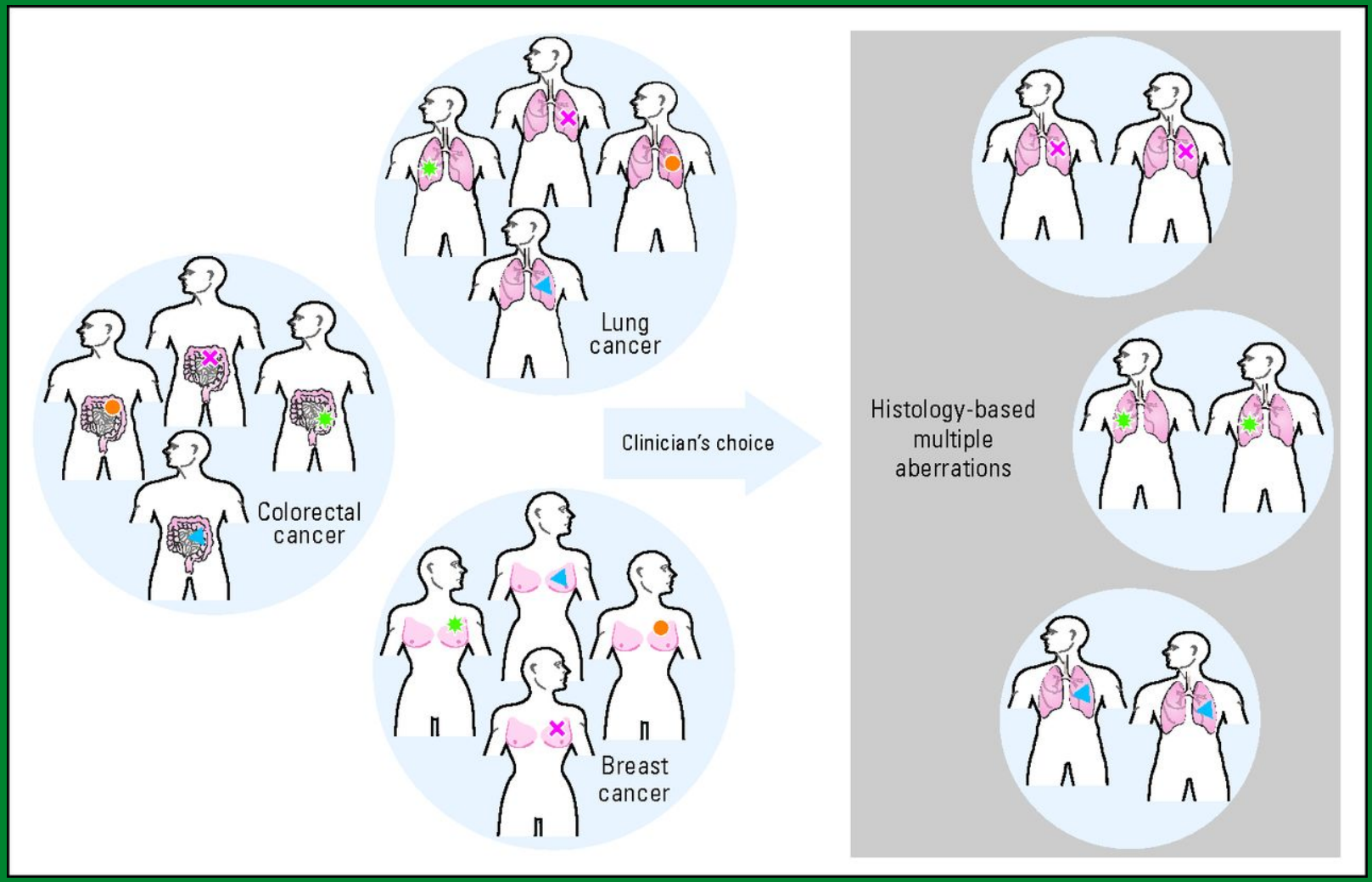
PARTICIPATING COUNTRIES

USA: OvCa, CNS (GBM), NSCLC (scc, adca), AML, CRC; **UK**: Breast ductal (TN, lobular, ER+HER2-); **Canada and Australia**: Pancreas (ductal); **France**: Breast (HER2+), Liver (alcohol), RCC; **Spain**: CLL; **Italy**: Pancreas (NET); **Germany**: Pediatric CNS (MB, Pilocytic); **India**: Oral Ca; **China**: Stomach ca; **Japan**: Liver (Virus)

OUTLINE OF CANCER GENOME RESEARCH HIGHLIGHTS

- Cancer cell compared to normal cell genome revealed 1000-10.000 somatic substitutions in most adult cancers; some have less (MB, GCT, AL, NET), and a few have >100.000 (NSCLC, Mel)
- Systematic sequencing allows the distinction of driver/passenger alterations: Driver *mut* cluster in the subset of cancer genes, while passenger *mut* are randomly distributed (errors are possible)
- The known ~400 *mut* genes represent 2% of protein coding genes of human genome. Dominant genes are activated by *mut* in one allele (80% of cancers), while recessive genes require both alleles *mut* (suppressor genes)
- Studies of cancer evolution demonstrate that are necessary 5 *mut* cancer genes (some require < 5)
- Mutated genes have a central role in the genesis and maintenance of cancer clones. There is evidence of mutagenic and repair processes
- There is evidence of a tree of clonal evolution with distinct dominant sub-clones which can persist or disappear, influenced by biological selection or therapy
- Highly personalized cancer genome
- Driver mut proteins are actionable and targetable by specific drugs

Histology-based clinical trial design to evaluate multiple molecular aberrations.



Sleijfer S et al. JCO 2013;31:1834-1841

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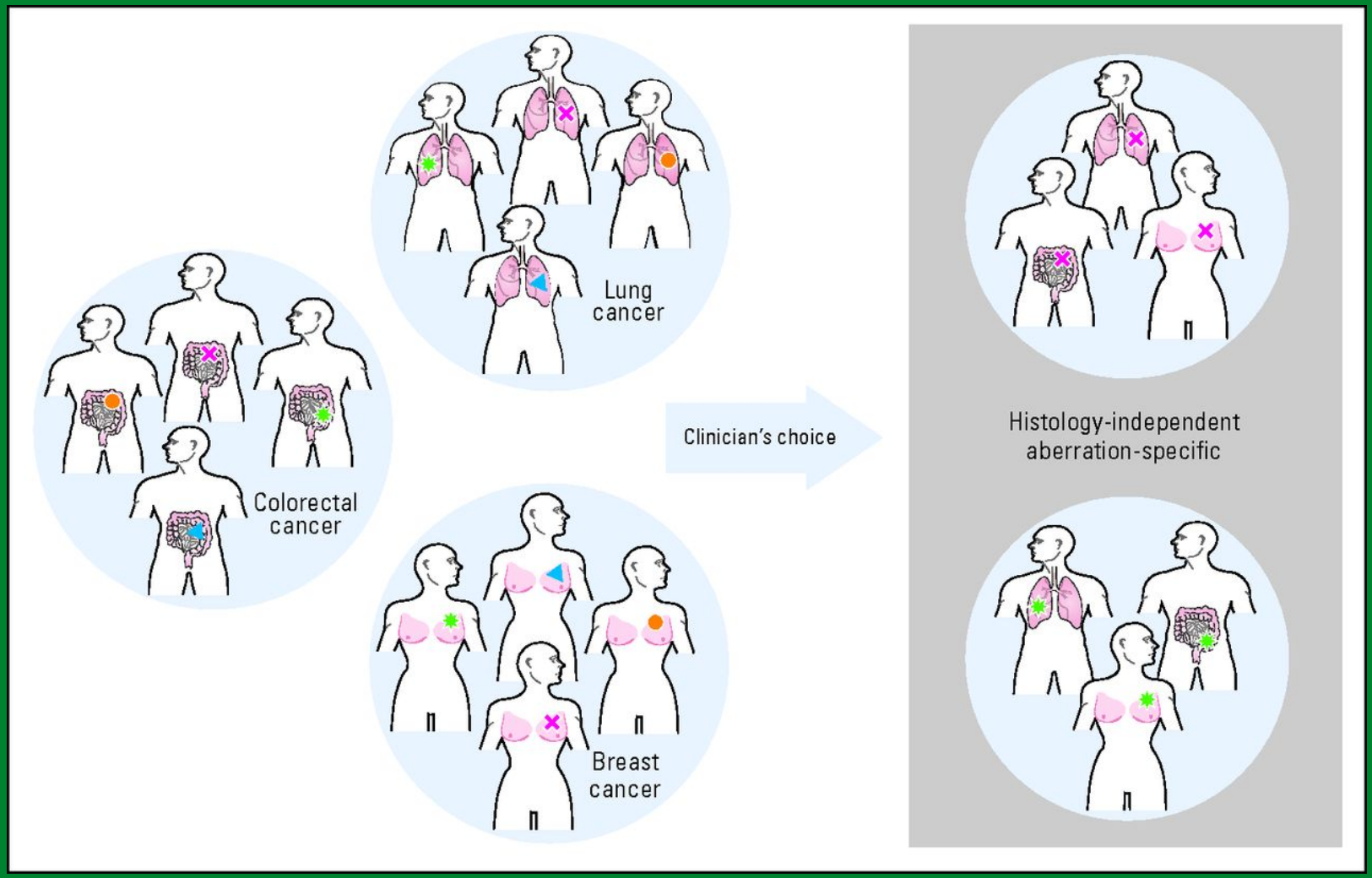
JOURNAL OF CLINICAL ONCOLOGY ASCO

Histology-independent, aberration-specific clinical trial design.



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JOURNAL OF CLINICAL ONCOLOGY ASCO

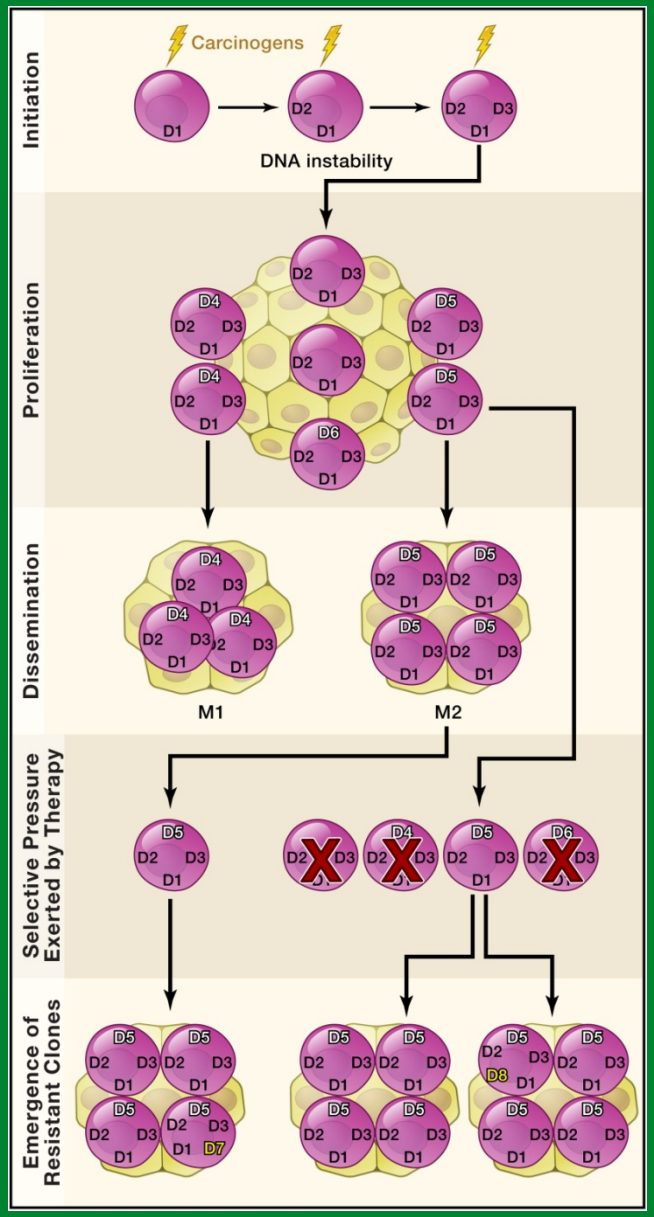
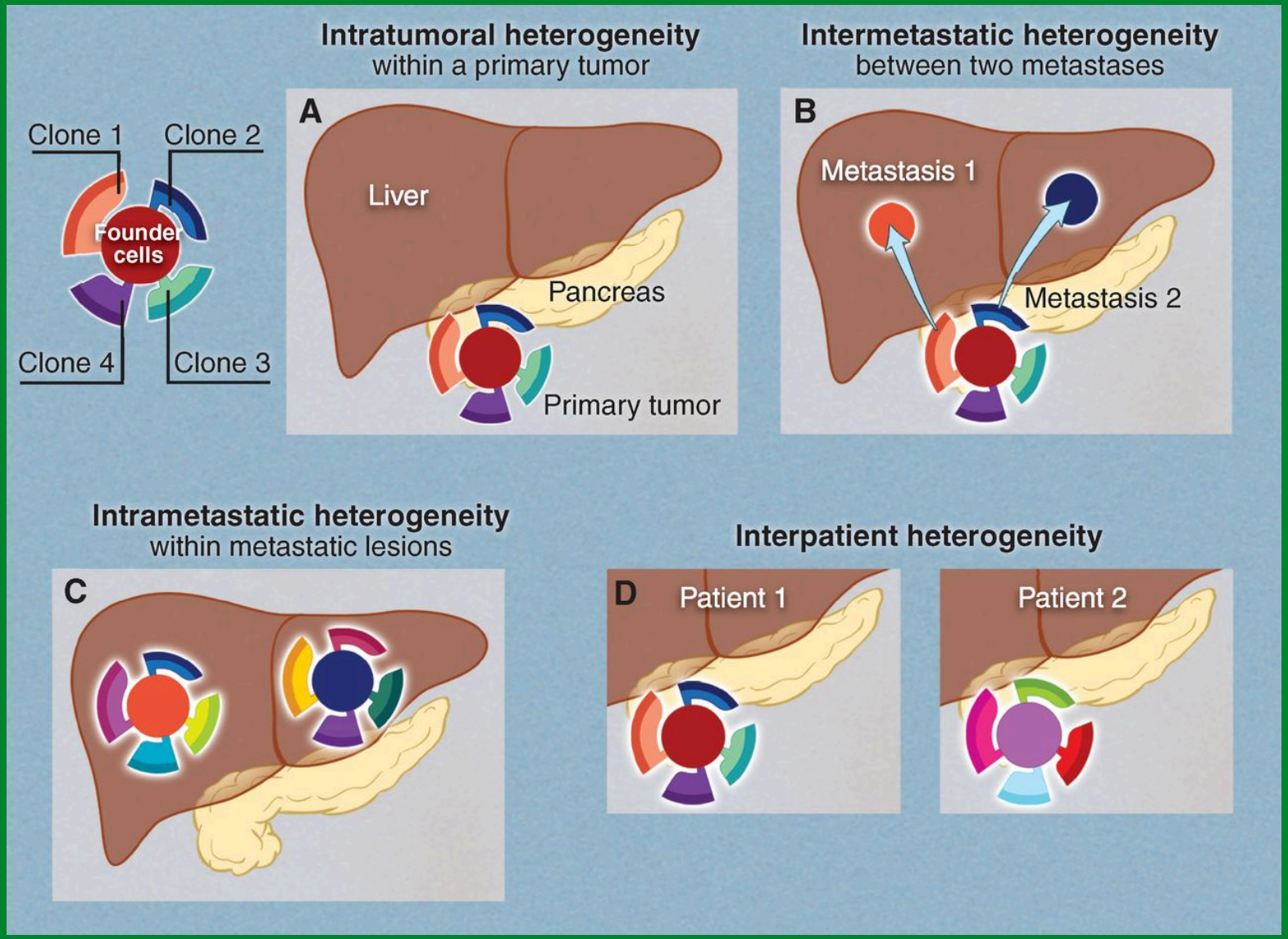

Initiation
Carcinogens
D1
D2 D1
D2 D3 D1
DNA instability
Proliferation
D2 D3 D1
D4 D2 D3 D1
D5 D2 D3 D1
D6 D2 D3 D1
Dissemination
D4 D2 D3 D1
D5 D2 D3 D1
M1
M2
Selective Pressure Exerted by Therapy
D5 D2 D3 D1
D4 D5 D6
Emergence of Resistant Clones
D5 D2 D3 D1
D7
D8

Fig. 6 Four types of genetic heterogeneity in tumors, illustrated by a primary tumor in the pancreas and its metastatic lesions in the liver.



B Vogelstein et al. Science 2013;339:1546-1558

PERSONALIZED CANCER MEDICINE

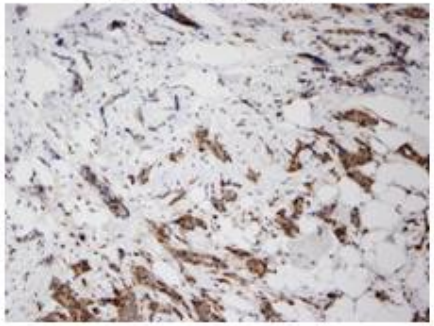
“The tumor clonal evolution should be measured”

- Current generation diagnostic genomic tests for cancer are binary (present/absent) and take no account of the proportion of the tumor mutations or amplifications, so fail to deal with clonality
- Tumors are heterogeneous
- All gene directed therapies select for resistant clones
- Develop techniques for disease monitoring (multiple biopsies, studies of sensitive/resistant populations, etc.)

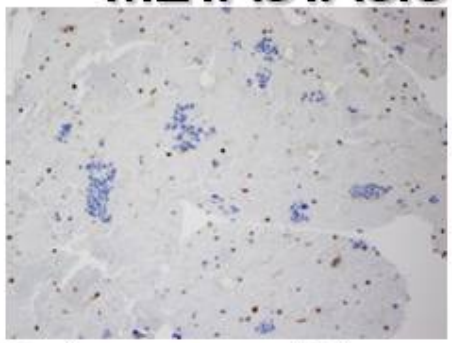
S Aparicio et al (N Engl J Med 2013;¡368:842-51)

Heterogeneity and Molecular Evolution
n=51 tumors and 56 metastases

PRIMARY



METASTASIS



Assay	Loss	Gain	Discordance
PTEN	10% (5/46)	15% (8/46)	25%
PIK3CA	8% (4/21)	8% (4/21)	16%
marked change	8% (4/21)	12% (6/21)	20%
ER (38)	7% (4/38)	0% (0/38)	7%
PR	12% (7/38)	3% (2/38)	15%
HER2	2% (1/12)	2% (1/12)	4%

Ana Gonzalez, Funda Meric, Kat Hale

HIGHLY PERSONALIZED TUMOR GENOME

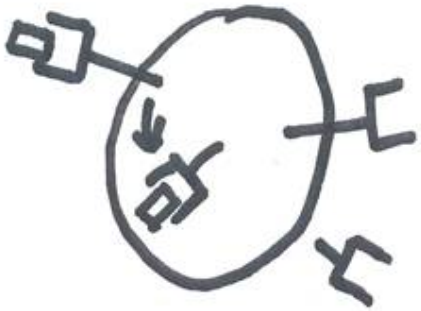
Applications for the prevention, detection, diagnosis and therapy:

- Biology of the neoplastic change
- Evolution of the cancer clone
- Understanding metastasis
- Mechanisms of DNA damage and repair processes
- Identifying drug targets
- Monitoring cancer burden
- Early diagnosis
- Classification of cancer tissue origin
- Progression and response to therapy
- Drug resistance

Paul Ehrlich: Birth of Targeted Therapy

(1) Antibodies: Nobel Prize for Serum Therapy in 1908
(2) Targeted Chemotherapy: 1910-11

Receptors on Cells



Postulated "side-chains," or "receptors" specific for external substances (dyes), antigens and nutrients.

A Bacterial Toxin AND A Targeted Chemotherapy

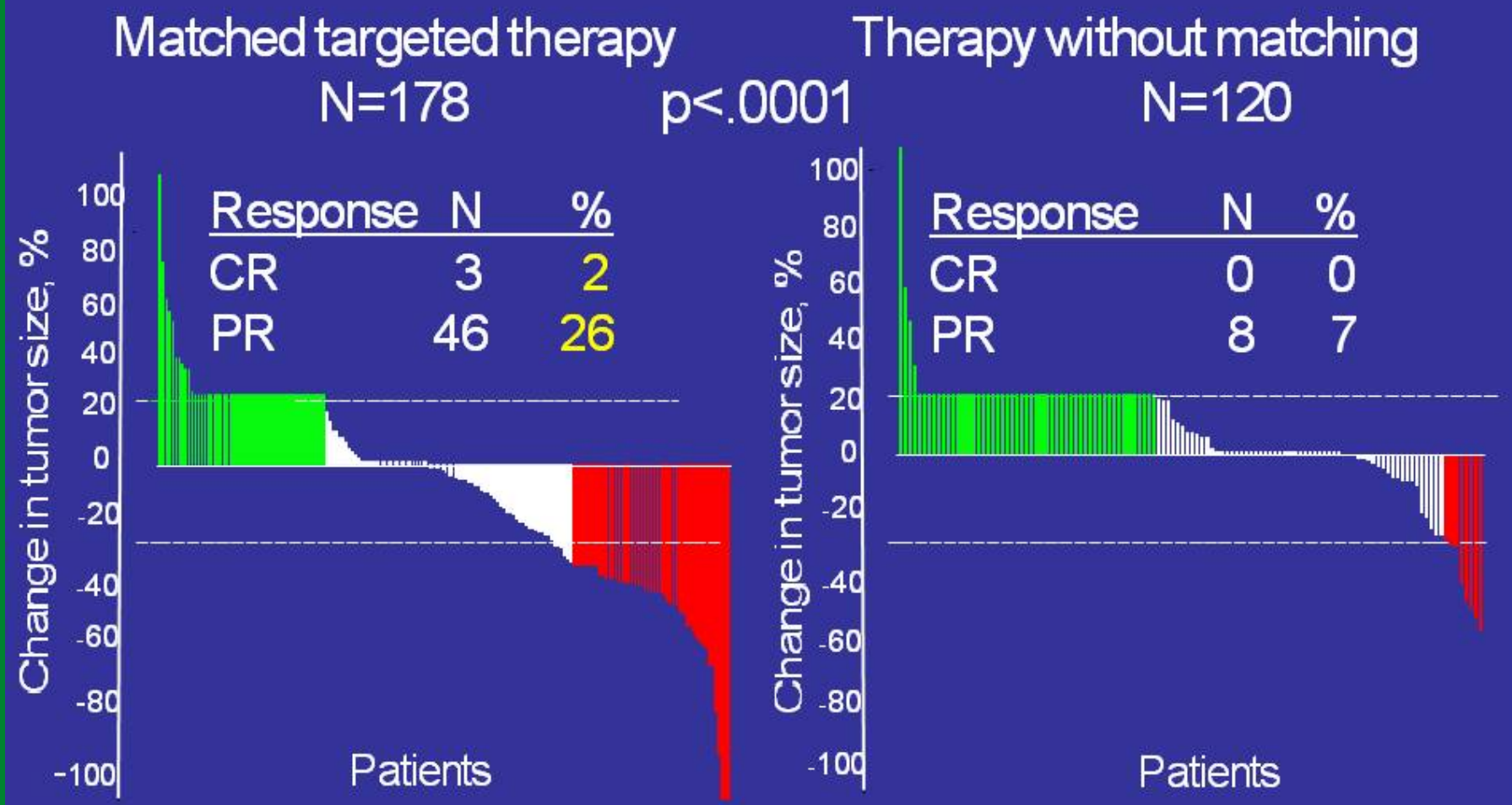
Model: bifunctional agent, containing a chemical structure that binds to the "receptor" linked to a toxic molecule.

Tissue Molecular Aberrations

	No. of pts.	% *
Molecular analysis ordered	955	
Adequate tissue available	852	
No. of mutations		
0	498	58.5
1	313	36.7
2	38	4.5
3	3	0.4
No. of pts. with any aberration	354	41.5

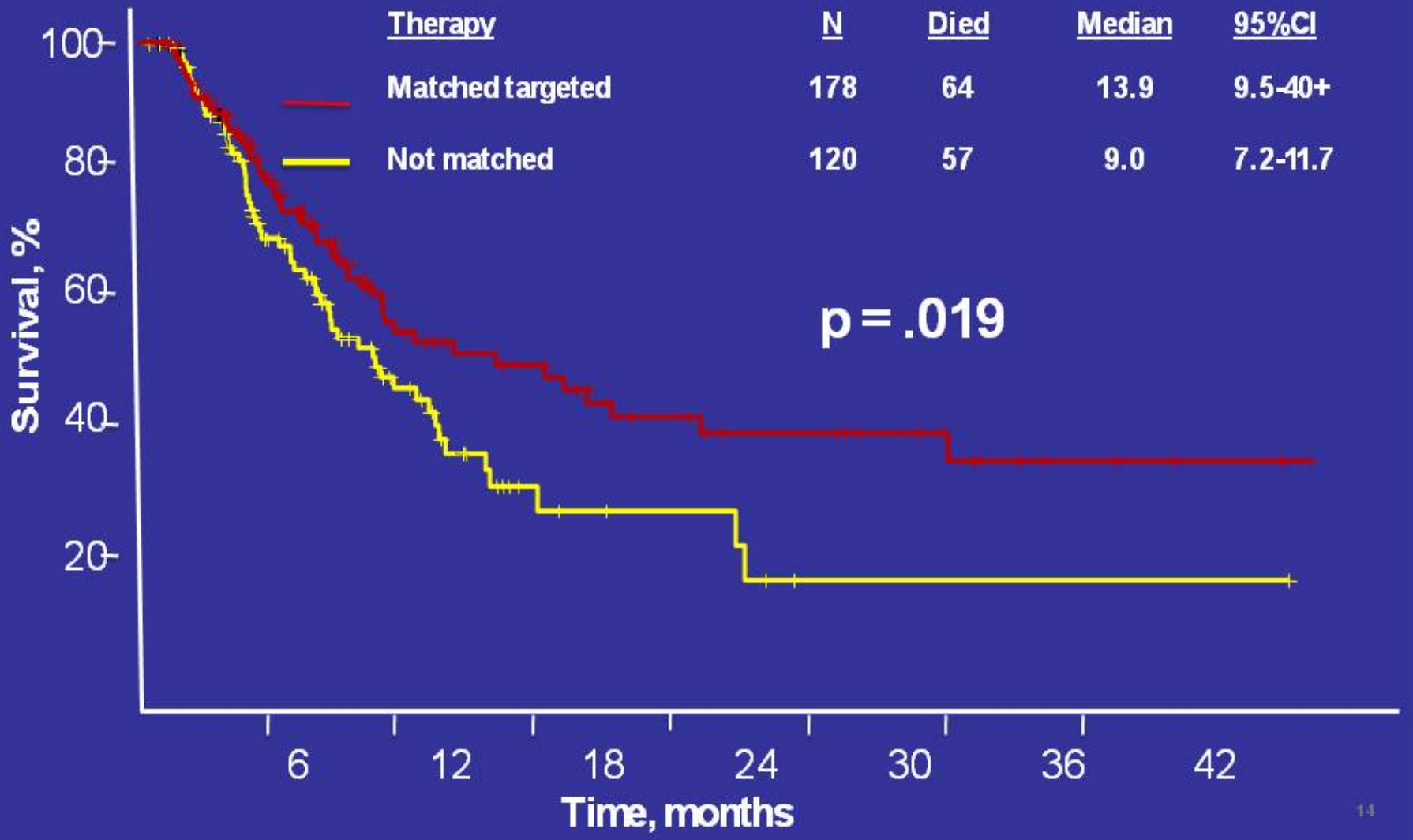
** Proportion was calculated for patients whose tissue was analyzed for ≥ 1 molecular aberration*

Best RECIST Response



Presented By John Mendelsohn, MD at 2011 ASCO Annual Meeting

Survival by Therapy. Patients with 1-3 Molecular Aberrations



Presented By John Mendelsohn, MD at 2011 ASCO Annual Meeting

PLATAFORMA DE ONCOLOGÍA
MICROARRAY GENOMIC EXPRESSION-BASED THERAPY IN REFRACTORY SOLID TUMORS

CONVENTIONAL CHEMOTHERAPY GENES

5Fluoro-uracil: DPYD, TYMS
Capecitabine: ECGF1, DPYD, TYMS
Doxorubicin: TOP2A, TOP2B, LAPTM4B, TIMP1,YWHAZ, ABCG2, ABCB1
Epipodophyllotoxins: TOP2A, TOP2B, ABCB1
Platinum derivs: ABCC2, ATP7B, POLH, SLC31A1, GSTP1, BRCA1, ERCC1, MT1A
Taxanes: ABCC3, TACC3, TUBB3, ABCB1
Gemcitabine: RRM1, DCK, CDA, CPTS
Ara C: DCK,RRM1, POLA1, POLA2
Irinotecan: TOP1, CES1, CES2, APTX, ABCG2
Methotrexate: FOLR2, DHFR, ABCC1
Pemetrexed: DHFR, GART, TYMS, FOLR2
Thiopurines: HPRT1, TPMT
Cyclophosphamide, Ifosfamide: CYP2B6, CYP3A4, CYP2A6, CYP3A5
Interleukin 2: IL2RA, IL2RB, IL2RG
Mitomycin C: NQO1
Nano-albumin paclitaxel: SPARC
Pentostatin: ADA
Temozolamide, DTIC: MGMT
Tamoxifen, Fulvestrant, Ais: ESR1, PGR
Asparaginase: ASNS
5 Azacitidine, Decitabine: DNMT1
Bleomicin: BLMH
Trastuzunmab, Pertuzumab, Lapatinib: ERBB2
Cetuximab, Erlotinib, Gefintinib, Afatinib: EGFR
Cetuximab, Panitumumab, (CRC wt k-ras): AREG, ERE

TARGET CHEMOTHERAPY GENES

PARP1: Olaparib, Velaparib
PTGS2: AINES AGTR!: Losartan
CD52: Alemtuzumab
HGF: Crizotinib, Cozantinib, Foretinib
AURKA/B: Alisertib, Tozaseritb
VDR: Calcitriol
JAK1/2/3: Ruxolitinib, Tofacitinib
GLI1/2, SMO: Vismodegib
HDAC1: Vorinostat
GHR: Pegvisomant
CD33: Gemtuzumab-ozogamicin
ALK: Crizotinib
NFKB1/2: Bortezomib
PRKCB1: Enzastaurin
PLK1: Specific inhibitors
NTRK1/2/3: Lestaurtinib
SSTR1/2/3/5: Octreotide, Lanreotide, Pasireotide
AR: Flutamide, Bicalutamide, Goserelin, Abiraterone
RXRA/B: 13-cisRA, Bexarotene,9-cisRA
RARA/B/G: ATRA, Fenretinide, 9-cisRA
IGF1R: Figitumumab, Cixutumumab, Ganitumumab
NOTCH1/2/3/4: Gamma-secretase inhibitors
ABL1, KIT, PDGFA, PDGFC, PDGFRA/B: Imatinib, Nilotinib, Ponatinib
PRKCD/E/H/Q: Midostaurin, UCN-01
BCL2: Oblimersen, Navitoclax, Obatoclax
AKT1/2/3: Perifosine, MK2206
PIK3CA/R1/R2, PTEN: PI3K and mTOR inhibitors
MET: Crizotinib, Cozantinib, Foretinib
EPHA, YES1, SRC: Dasatinib
FGFR1/2/3: Dovitinib, Brivanic, Ponatinib
FRAP1: Sirolimus, Everolimus, Temsirolimus
BRAF, RAF1, RET, FLT3/1/4 KDR: Sorafenib, Sunitinib, Pazopanib, Vandetanib, Axitinib, Cediranib, others
VHL, HIF1A, VEGFA/B/C: Bavacizumab, Sorafenib, others

PLATAFORMA DE ONCOLOGÍA
MICROARRAY GENOMIC EXPRESSION-BASED THERAPY IN REFRACTORY SOLID TUMORS

- TOTAL PROCEDURES 136
- AGE (y) Median (range) 56 (12-83)
- Prior therapies: Median (range) 3 (1-6)
- Tumor-types: Breast 24; CRC 17; NSCLC 16; STS 11; Ov 10; Stomach 9; Pancreas 8; H&N SCC 4; Salivary gland 4; Esophagus 5; Cholangioca 4; NHL 4; Melanoma 3; SCLC 3; Uterus 2; Bladder 2; GCT 2; Mesothelioma 2; Others 3 (Thymoma, Thyroid ca, Skin non melanoma)

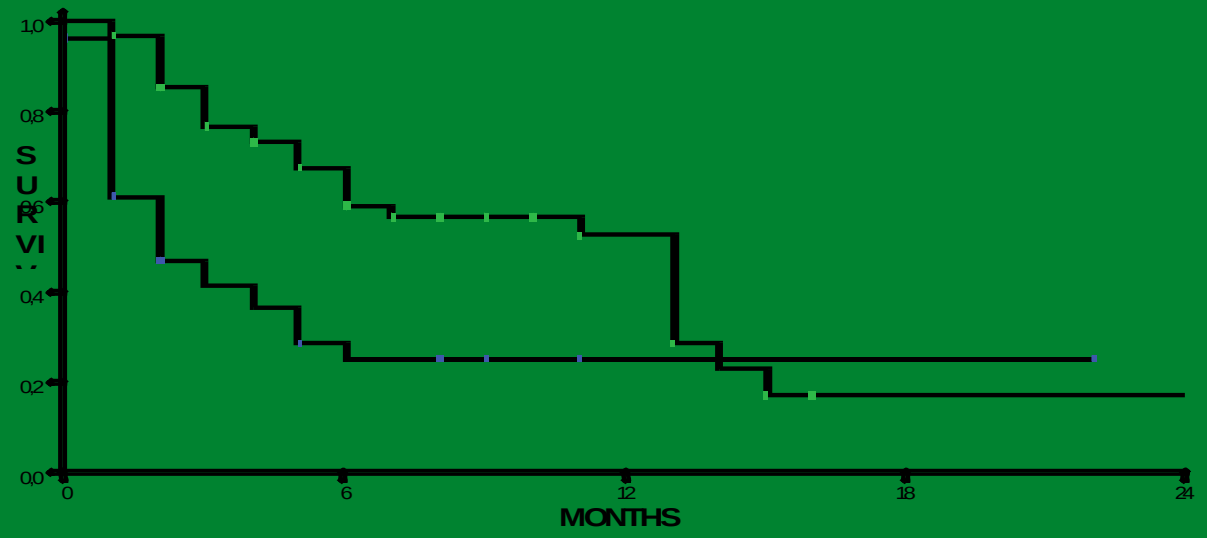
PLATAFORMA DE ONCOLOGÍA
MICROARRAY GENOMIC EXPRESSION-BASED THERAPY IN REFRACTORY SOLID TUMORS

CLINICAL RESULTS: POSITIVE TESTING	119 (87.5%)
PATIENTS TREATED	64 (53,8%)
EVALUABLE FOR RESPONSE	55 (85.9%)
PARTIAL RESPONSE	**18 (32.7%)**
STABLE DISEASE	**22 (40%)**
PROGRESSION	**15 (27.2%)**

MEDIAN SURVIVAL TIMES in months (range)

	PFS	OS
Whole series	8 (5.3-107)	13 (9.3-16.7)
Tumor response	9 (6.4-11-6)	13 (7.5-18.5)
Stable disease	4 (1.2-6.8)	13 (0-27.3)
Progression	--------	4 (2.5-5-5)
Log-rank(p)	0.009	<0.001

PLATAFORMA DE ONCOLOGÍA
MICROARRAY GENOMIC EXPRESSION-BASED THERAPY IN REFRACTORY SOLID TUMORS



Survival according to Kaplan-Meyer for the treated and non-treated groups

(p=0.001)

PLATAFORMA DE ONCOLGÍA
MICROARRAY GENOMIC EXPRESSION-BASED THERAPY IN REFRACTORY SOLID TUMORS

Survival according to response groups (Mantle-Cox)

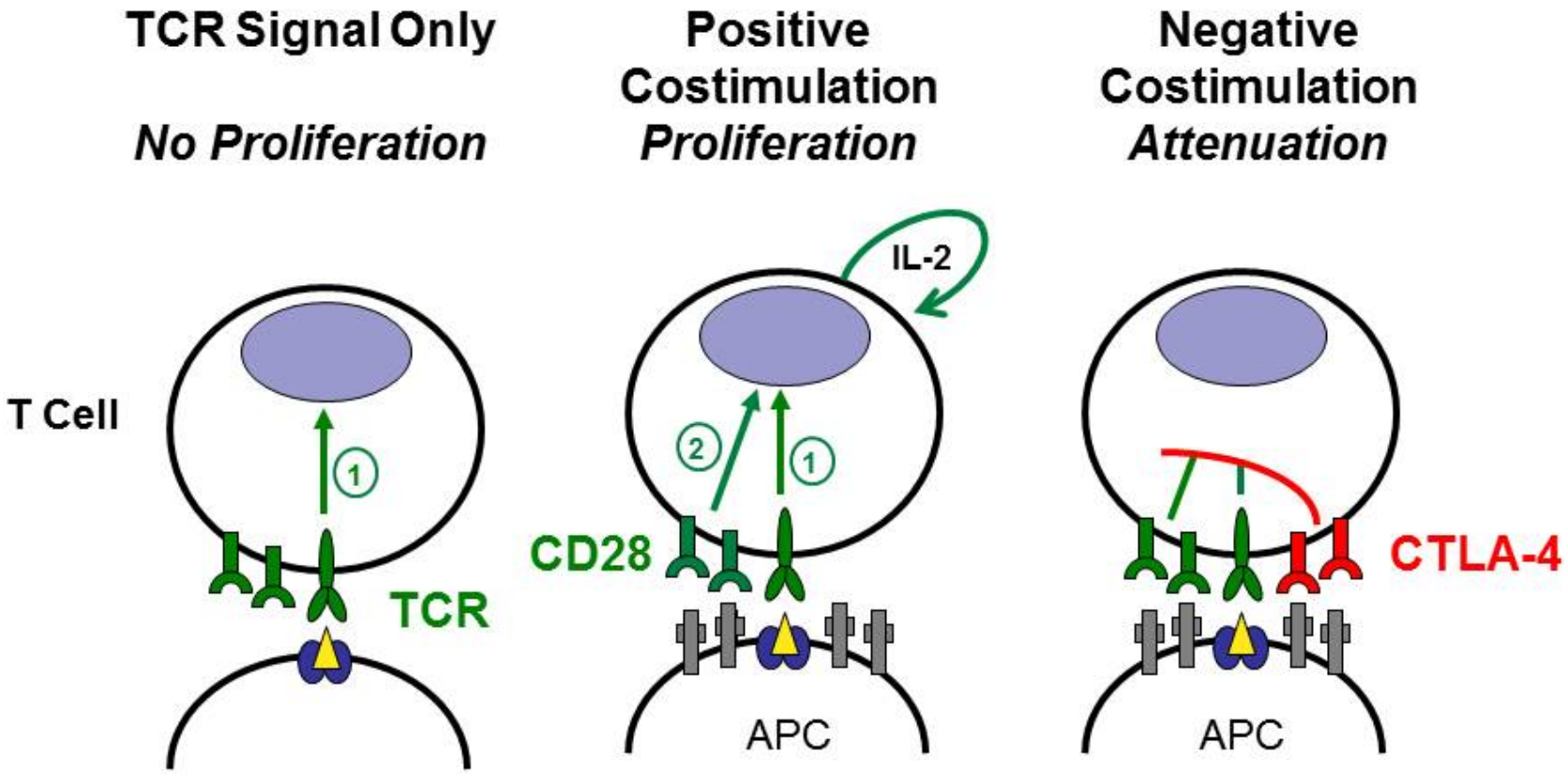
	PR vs SD		PR vs PD		SD vs PD	
	x^2	p	x^2	p	x^2	p
PFS	6.927	0.008	---	---	---	---
OS	6.773	0.009	29.138	<0.001	5.063	0.024

PERSONALIZED CANCER MEDICINE

Tailoring therapy to the particular tumor considering all of these factors:

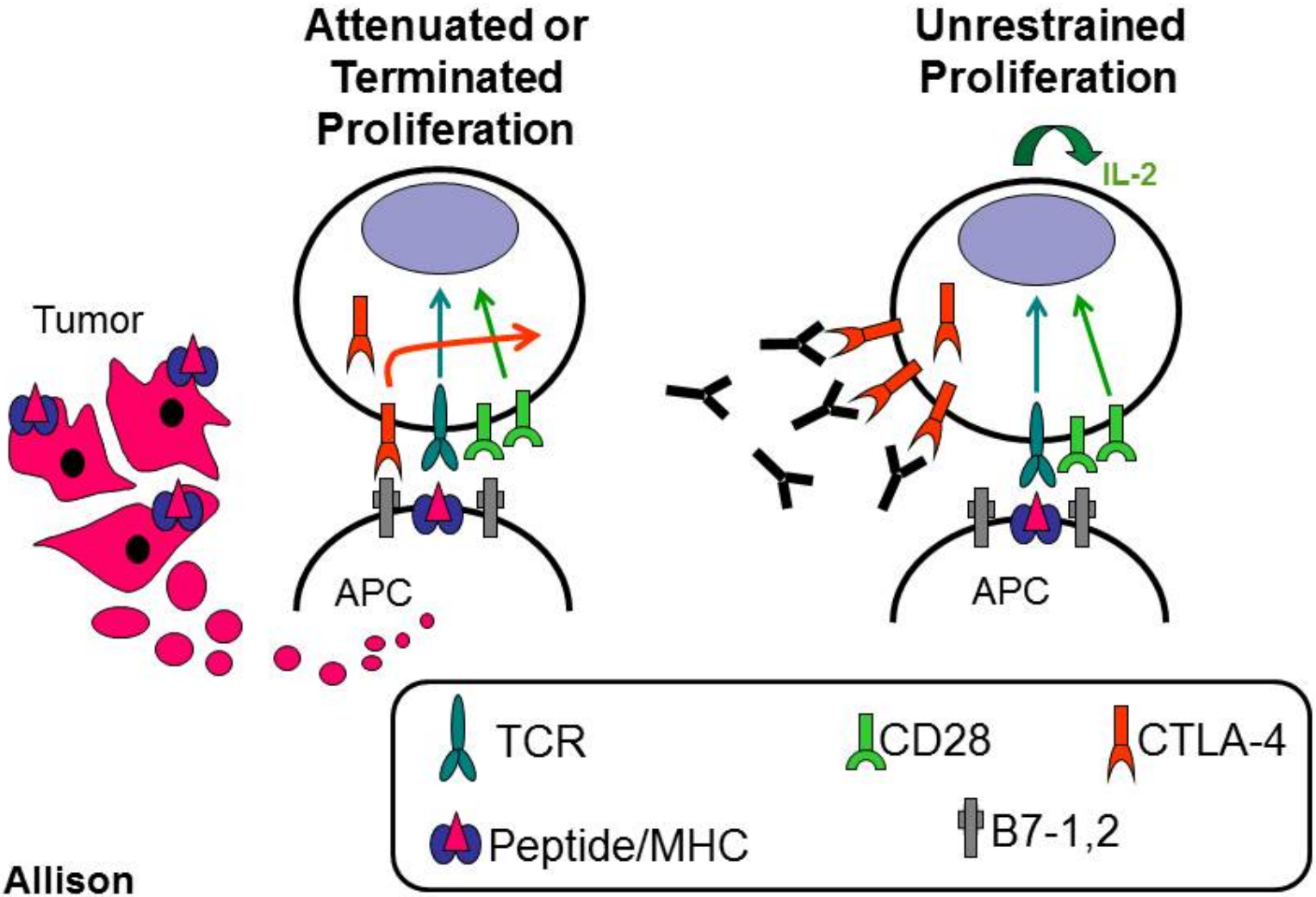
1. **Tumor profiling (somatic cell):** Driver mutations, signal pathway inhibitors, genetic dependency (addiction), chemotherapy profiling, etc .
2. **Clinical characteristics of the patient:** Pharmacogenomics; Functional assessment, Co-morbidity, Age, etc.
3. **Clinical features of the tumor:** Stage and Extension of the disease, Option for a local-regional Rescue, Oligometastatic spread of the disease, MRD, etc.
4. **Differentiation therapy; Cancer Stem cell therapy.**
5. **Immunetherapy & vaccines.**

Understanding T cell Biology:
Positive and Negative Signals Regulate T cell Activation



JP Allison

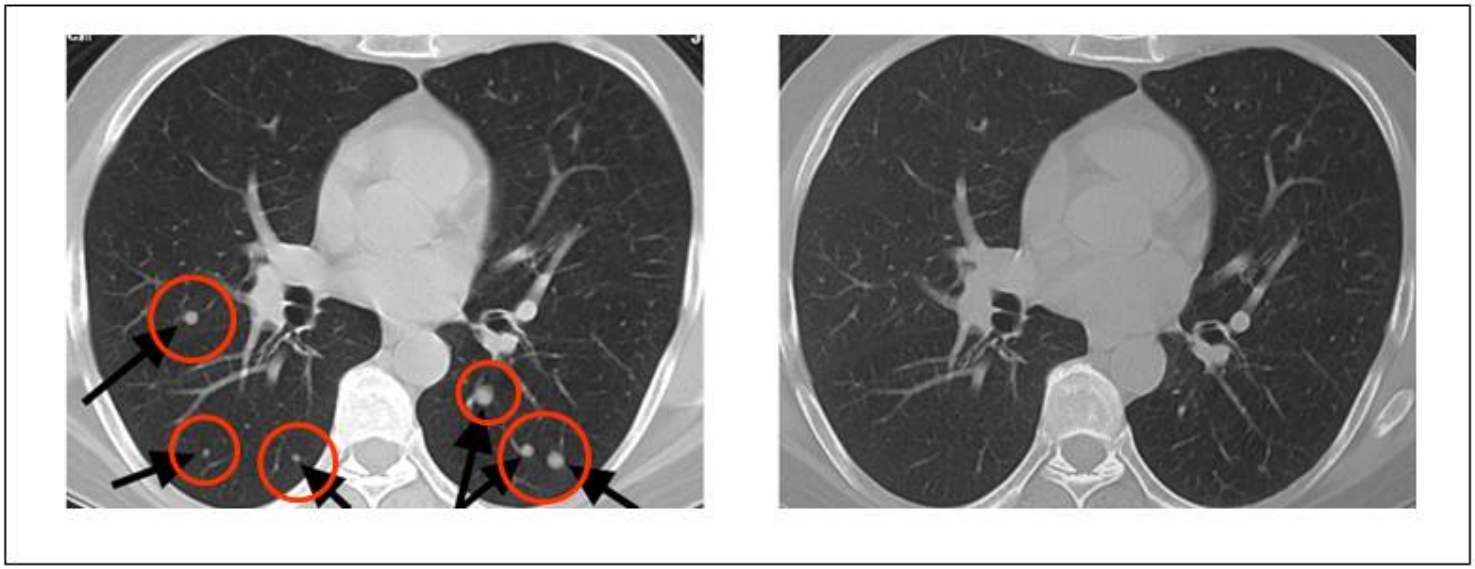
CTLA-4 blockade enhances T cell anti-tumor responses, regardless of tumor type



JP Allison

Anti-CTLA-4 (Ipilimumab) Complete Responder: Melanoma

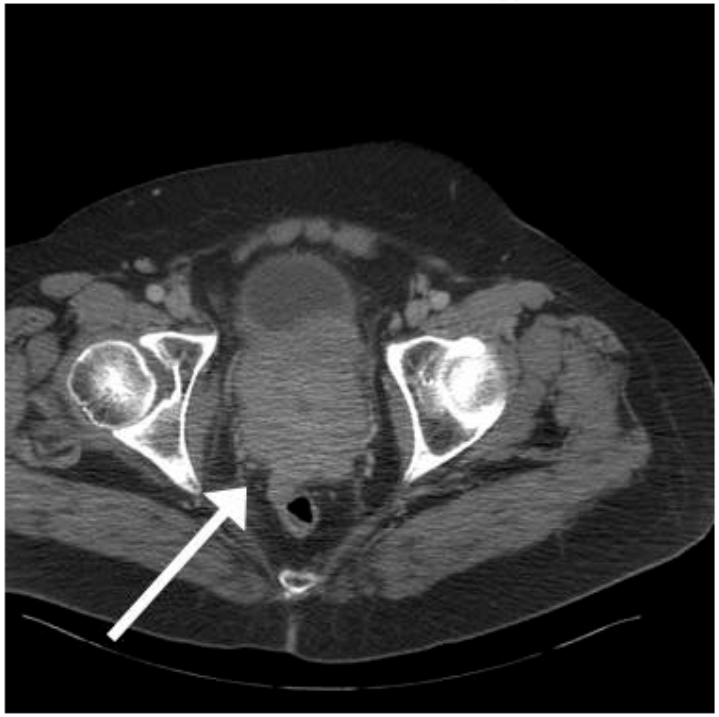
Experienced complete resolution of 2 subcutaneous nodules, 31 lung metastases and 0.5 cm brain metastasis.



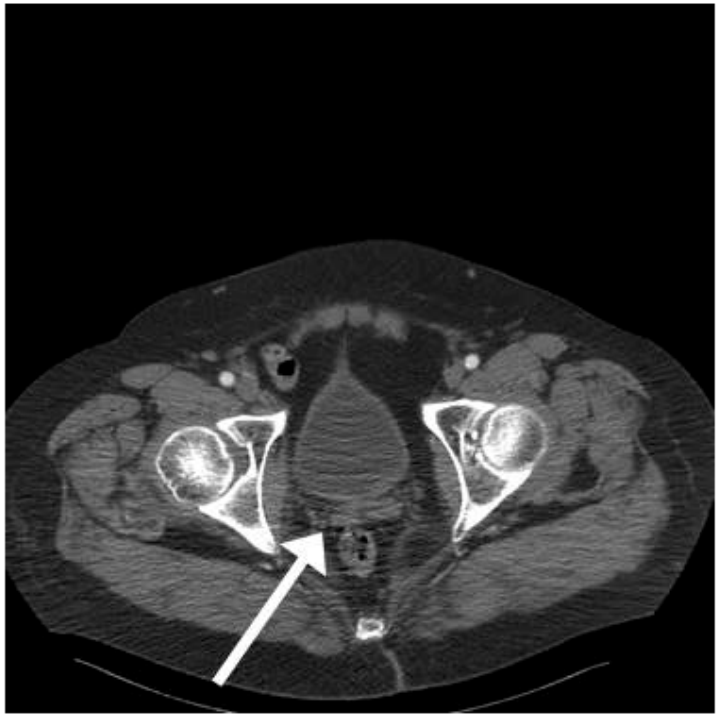
Phase III trials completed: survival benefit: FDA-approved therapy

Anti-CTLA-4 (Ipilimumab)
Complete Responder: Prostate Cancer

Screening

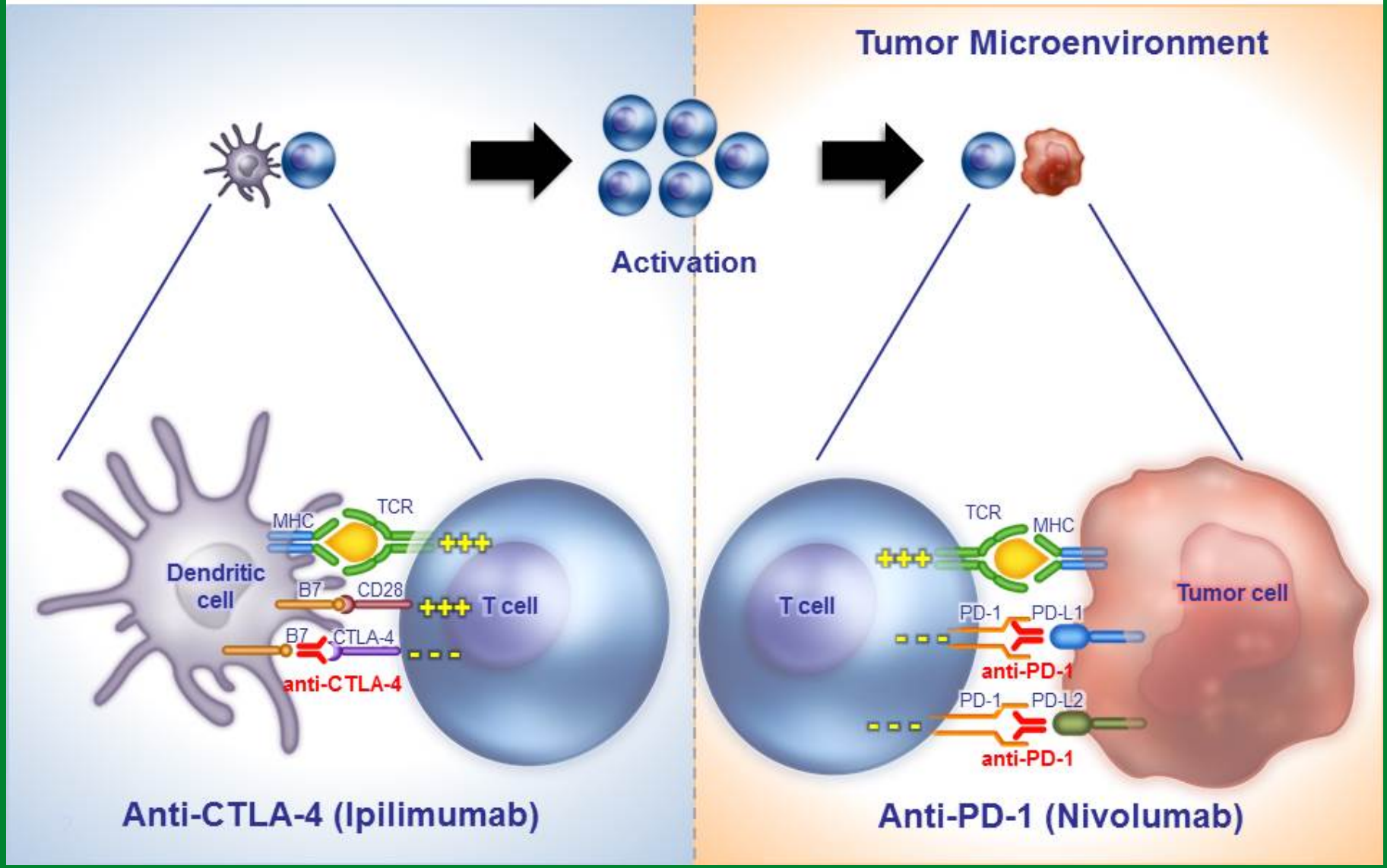


14 months

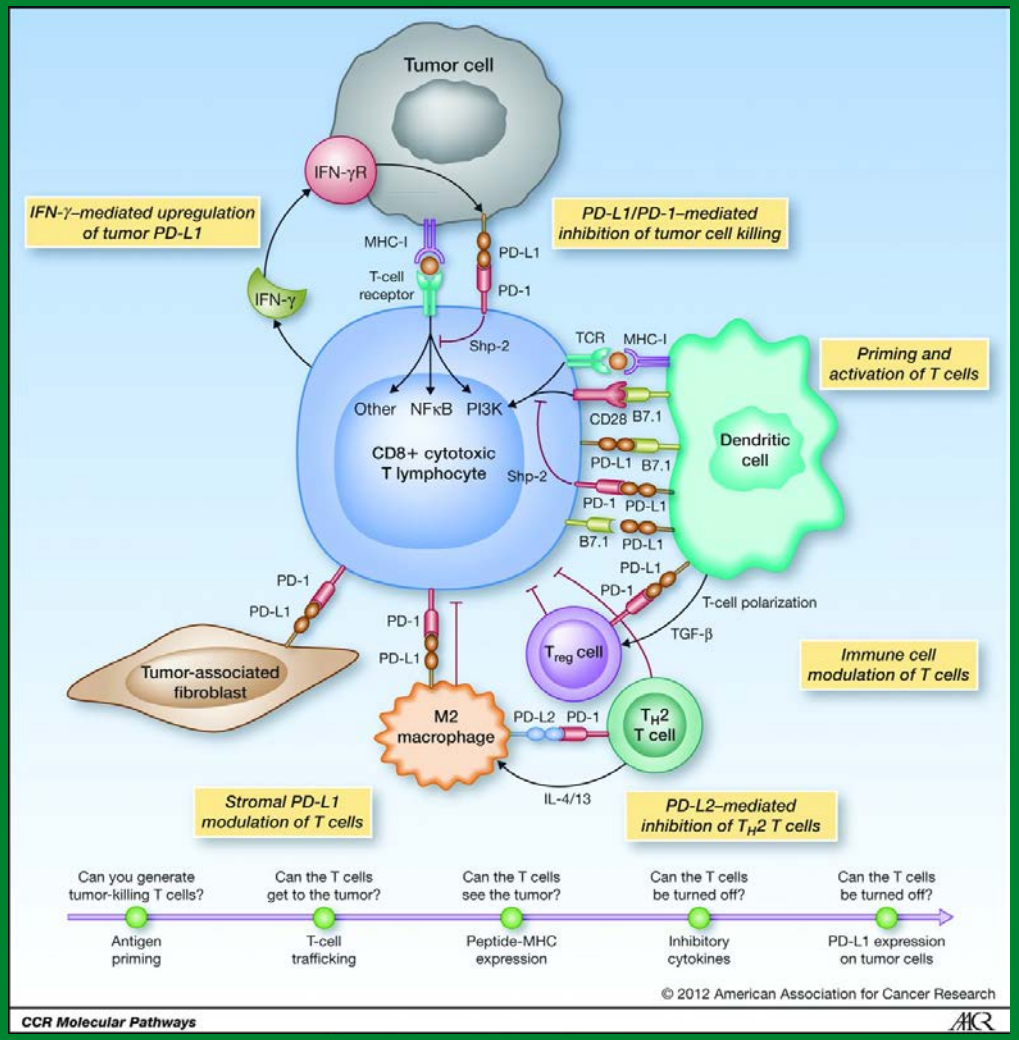


Phase III trials ongoing

CTLA-4 and PD-1: Distinct Immune Checkpoints



Tumor immunology and the PD-L1/PD-1 pathway.



Chen D S et al. Clin Cancer Res 2012;18:6580-6587

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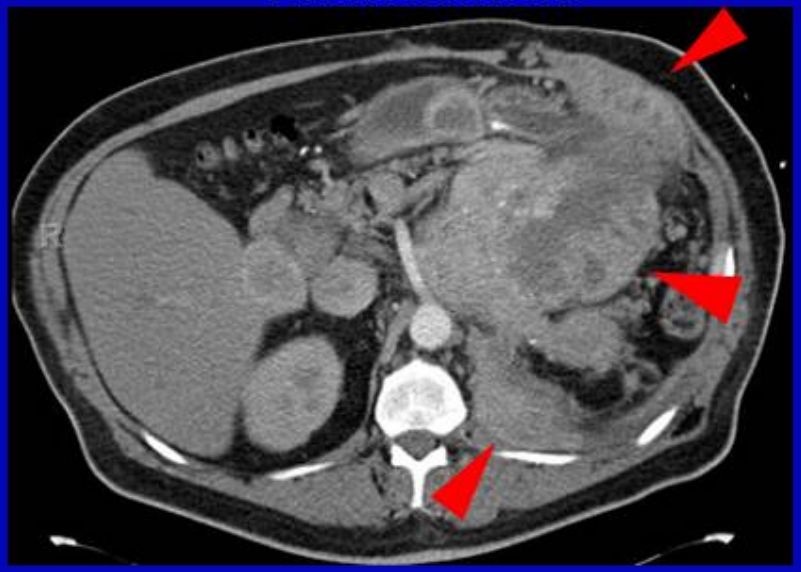
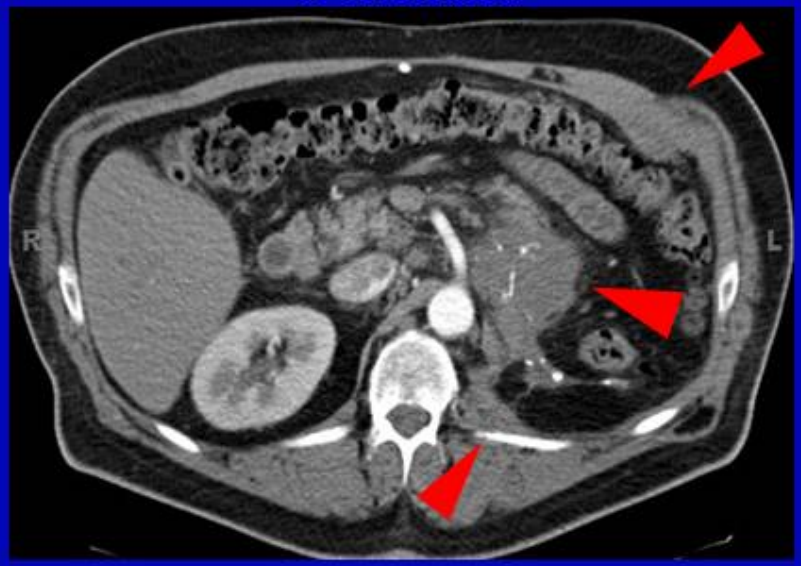
Clinical Cancer Research

Partial Regression of Metastatic RCC in a Patient Treated with 1 mg/kg BMS-936558

Pretreatment



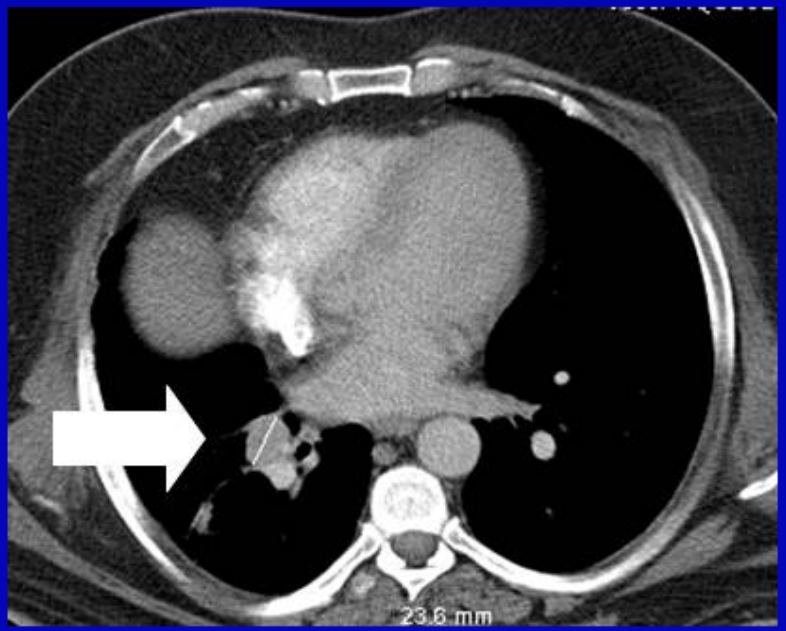
6 months

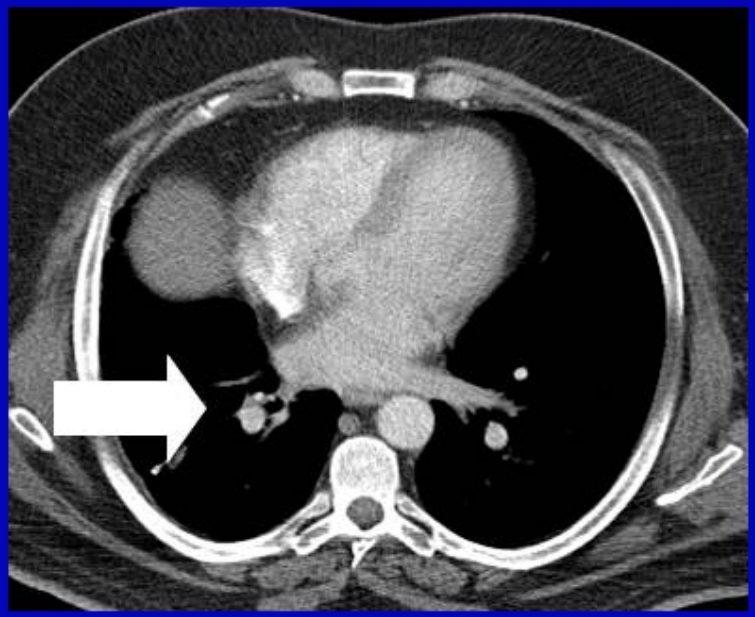


- The 57-year-old patient had developed progressive disease after receiving sunitinib, temsirolimus, sorafenib, and pazopanib
- Currently in cycle 6 with ongoing PR

Courtesy of C. Drake, Johns Hopkins Univ

Partial Regression of Metastatic RCC in a Patient Treated with 1 mg/kg BMS-936558: Durable Benefit off Therapy



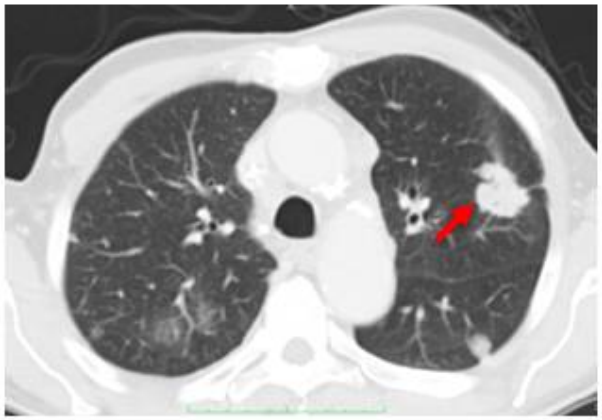


- The 48-year-old patient with low volume but poorly differentiated mRCC
- Developed progressive disease after sunitinib, sorafenib, and thoracic surgery
- Therapy held after 3 cycles due to near CR
- Response has continued for 3 years, while off therapy

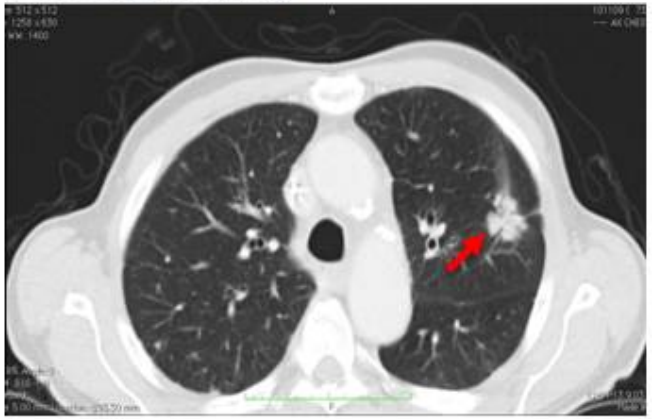
Courtesy of M. Sznol, Yale Cancer Center

Clinical Activity of MPDL3280A in NSCLC (Squamous)

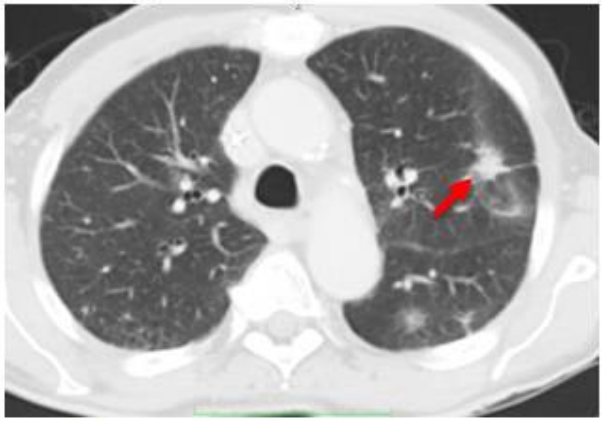
Baseline



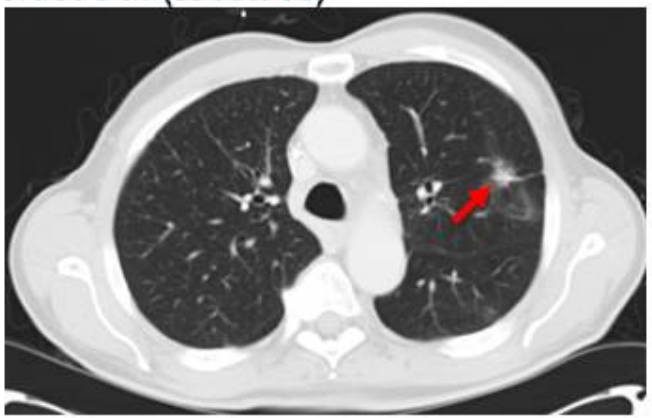
Post C2 (Week 6)



Post C6 (Week 18)



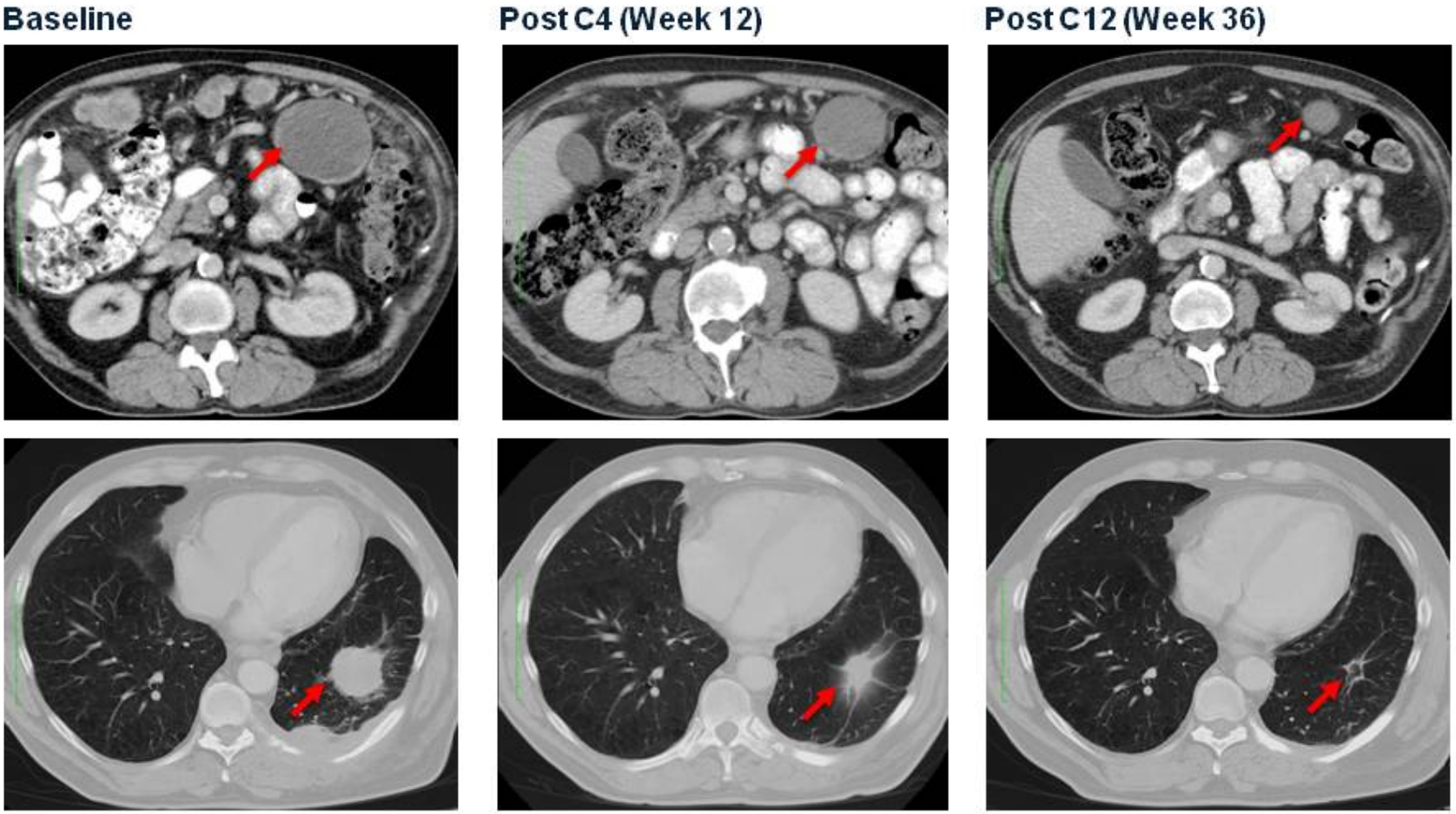
Post C16 (Week 48)



73-year-old male, s/p deep neck mass excision, ramucirumab + gemcitabine + carboplatin
PD-L1 positive

Images include data from after Feb 1, 2013.
Pinnacle Oncology Hematology (Gordon).

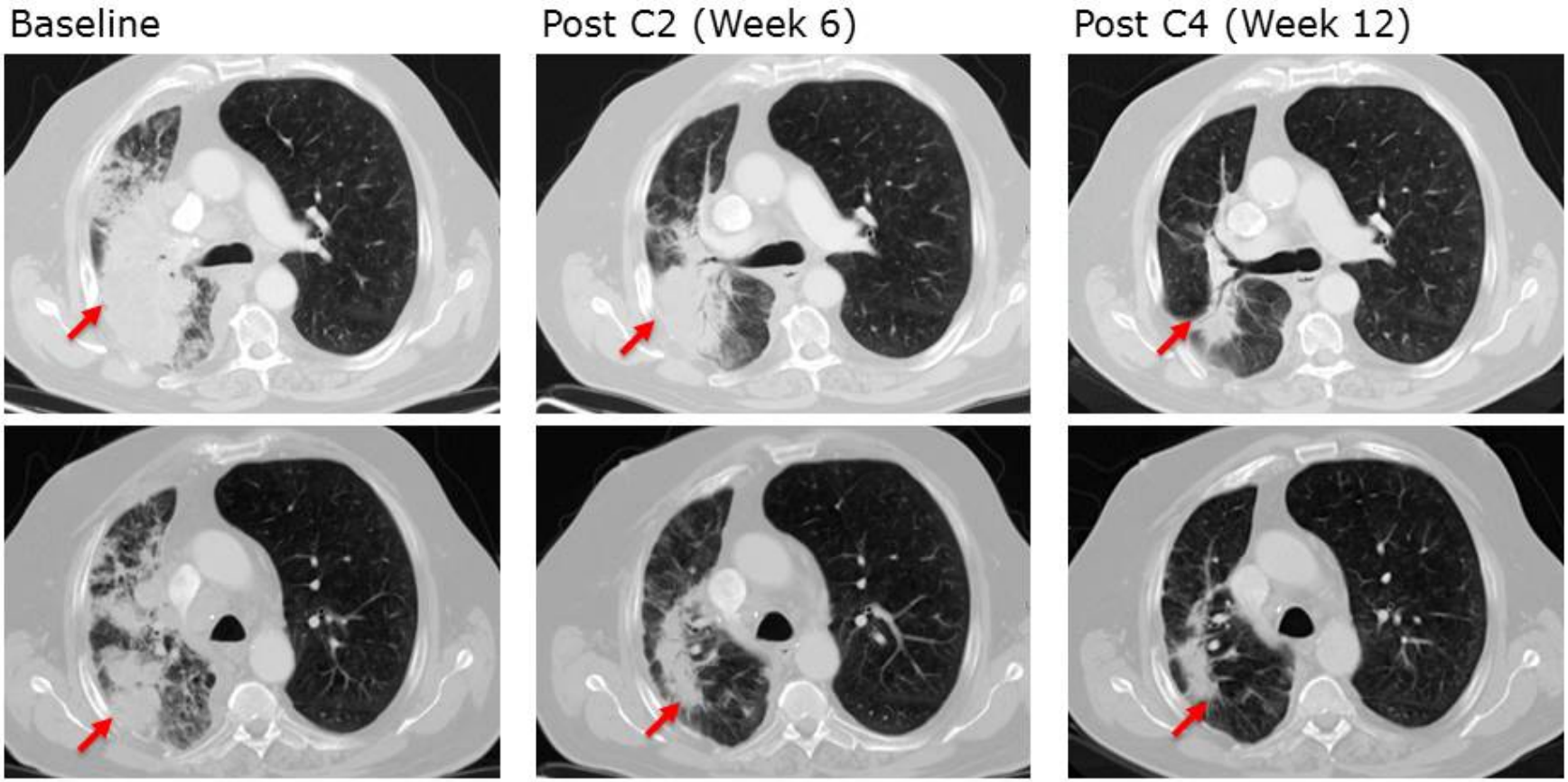
Clinical Activity of MPDL3280A in NSCLC (Adeno)



62-year-old male, pretreated including carboplatin + paclitaxel, bevacizumab + erlotinib, pemetrexed, PD-L1 positive

Images include data from after Feb 1, 2013. Virginia Oncology Associates (Conkling).

Rapid Response in an NSCLC Patient Treated With MPDL3280A Monotherapy

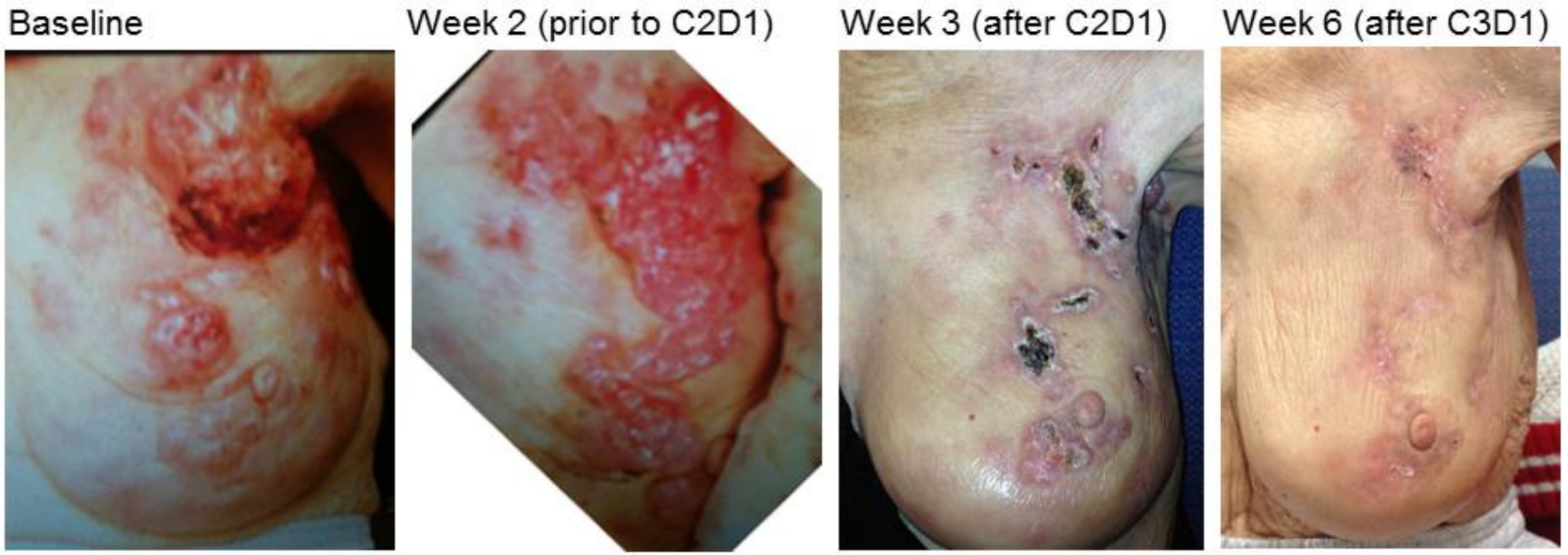


64-year-old male with squamous NSCLC s/p R lobectomy, cisplatin + gemcitabine, docetaxel, erlotinib, PD-L1 positive

Images represent data from patient enrolled after Aug 1, 2012.
Hospital Universitario Vall D´Hebron (Cruz/Tabernero).

MPDL3280A Phase Ia

Rapid Response to MPDL3280A in Head and Neck Cancer With Metastatic SCC of the Tongue



78-year-old female with HNSCC s/p carboplatin + radiation, paclitaxel + cetuximab, carboplatin + paclitaxel + cetuximab, cisplatin + 5FU, PD-L1 positive

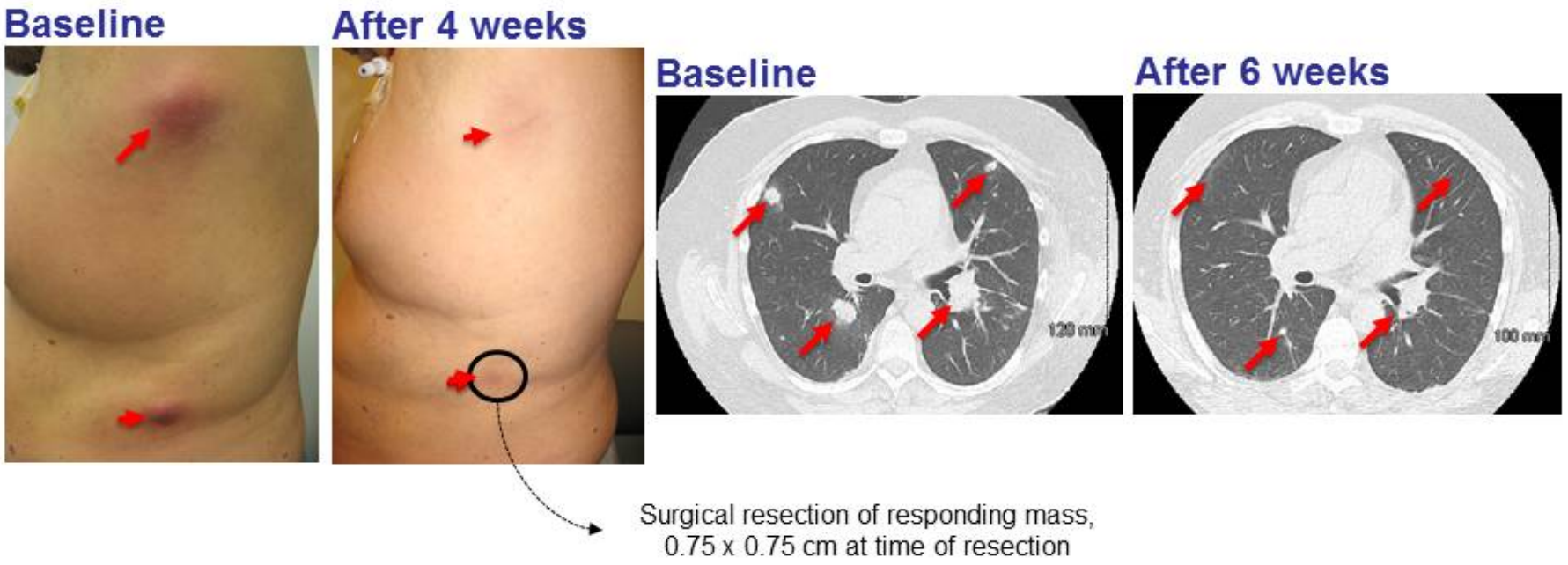
Images represent data from patient enrolled after Aug 1, 2012.
Comprehensive Cancer Center of Nevada (Braiteh).

MPDL3280A Phase Ia

PRESENTED AT: ASCO Annual '13 Meeting

Presented By Roy S. Herbst, MD, PhD at 2013 ASCO Annual Meeting

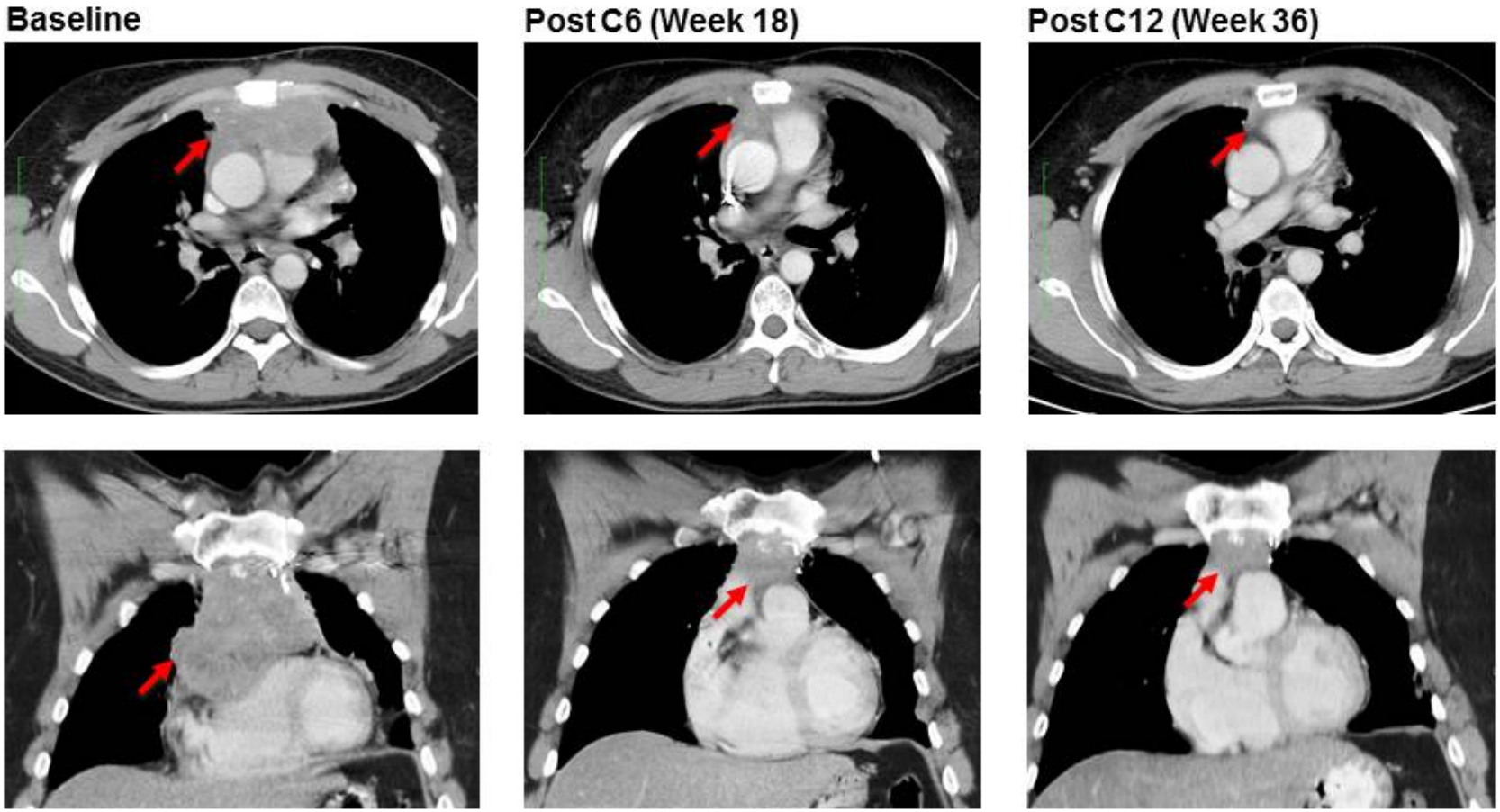
Serial Biopsy in a PD-L1–Positive RCC Patient With a Rapid Response to MPDL3280A



51-year-old male with RCC s/p L nephrectomy, sunitinib, XRT T9, temsirolimus, PD-L1 positive

Presented By John D. Powderly, MD at 2013 ASCO Annual Meeting

Clinical Activity of MPDL3280A in an NSCLC Patient

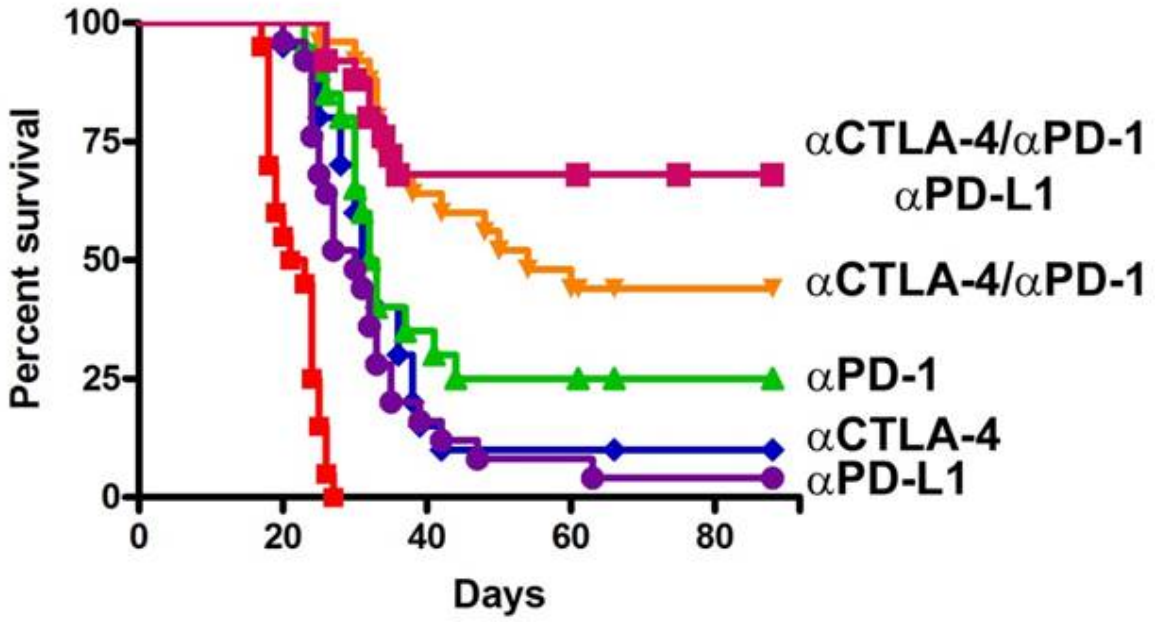


44-year-old male with adenocarcinoma NSCLC, s/p radiotherapy, gemcitabine + cisplatin, temozolomide + docetaxel, pemetrexed, bevacizumab, CDX-1401, PD-L1 negative

Images include data from after Feb 1, 2013.
Yale University (Gettinger/Herbst).

MPDL3280A Phase Ia

Mouse models with improved tumor rejection and survival due to combination therapy: anti-CTLA-4 plus targeting the PD-1/PD-L1 pathway



Curran et al., *PNAS*, 2010

PERSONALIZED CANCER IMMUNETHERAPY

1.- **Pre-existing immune-reactivity**

Evidence; NSCLC, Mel, RCC, CRPC, Other common tumors?

Therapy - **Checkpoint blockade** (CTLA4, PD1, PD-L1) **combination**

- Adoptive T cell enhancement/therapy (TILs)
- Cytokine therapy: HD-IL2, IL21
- Lymphodepletion, anti-Treg therapy

2.- **Absent immune-reactivity**

Therapy - **Adoptive trasfer of genetically engineered T cells**

- Chimeric antigen receptor (CAR)

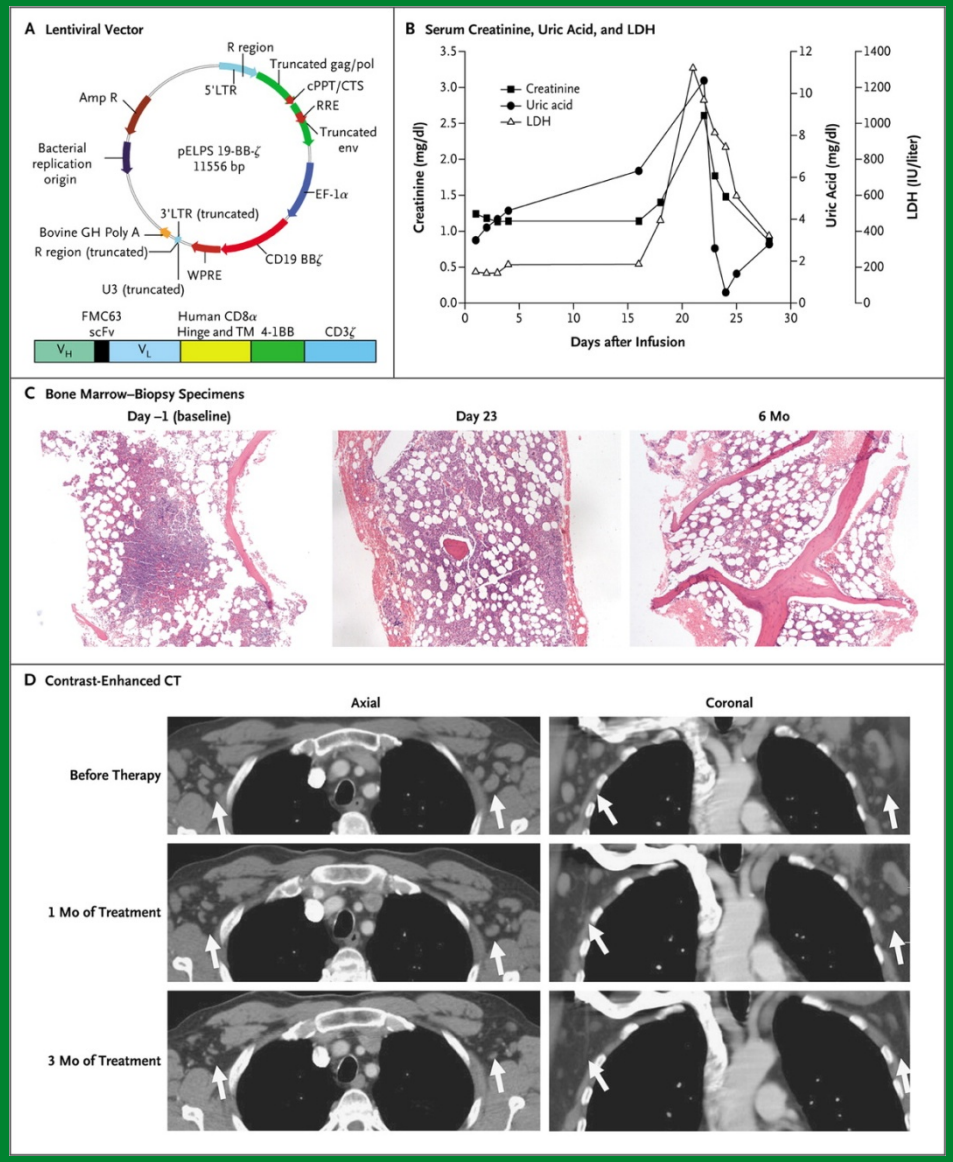
(CD19, VEGFR-2, EGFRvIII, Mesothelin, CSP4)

- Cancer specific TCR

(NYESO-1, MAGE-A3, MART-1, gp 100, CEA, MUC-1, 2G1, SSX2)

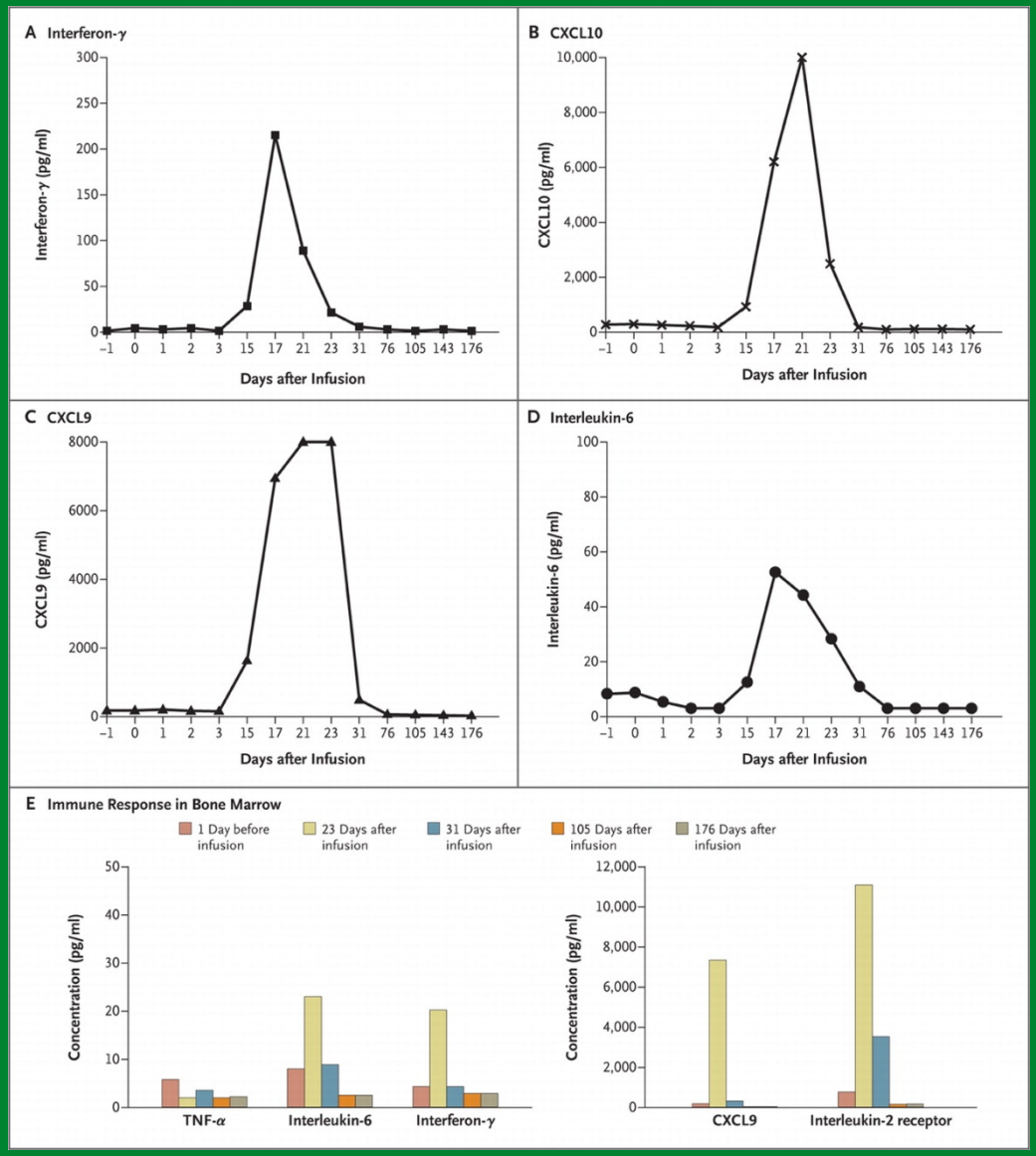
- Non specific stimulation, cytokine therapy (HD-IL2)

Clinical Response in the Patient.



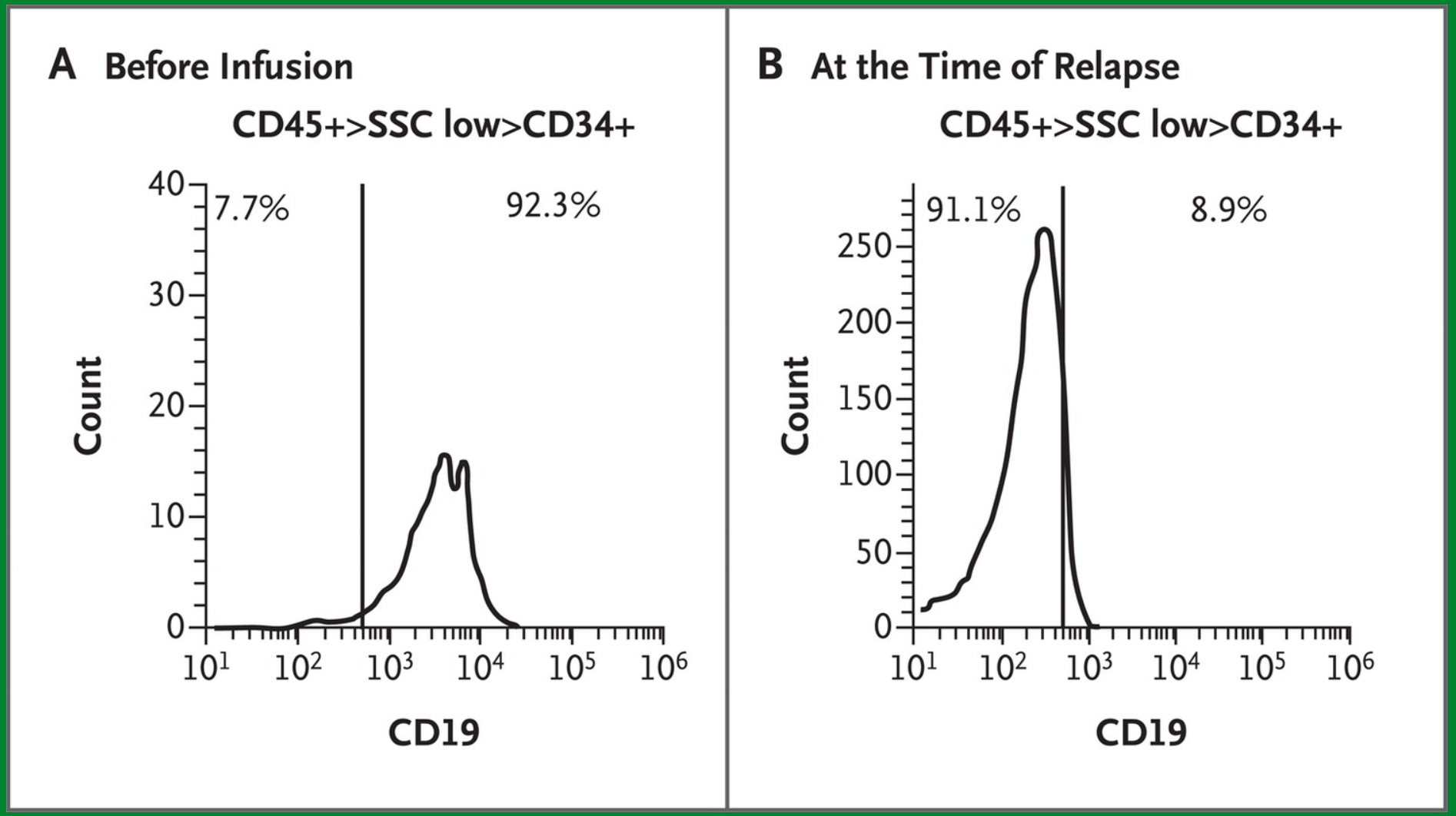
Porter DL et al. N Engl J Med 2011;365:725-733.

Serum and Bone Marrow Cytokines before and after Chimeric Antigen Receptor T-Cell Infusion.



Porter DL et al. N Engl J Med 2011;365:725-733.

CD19 Expression at Baseline and at the Time of Relapse in Patient 2.

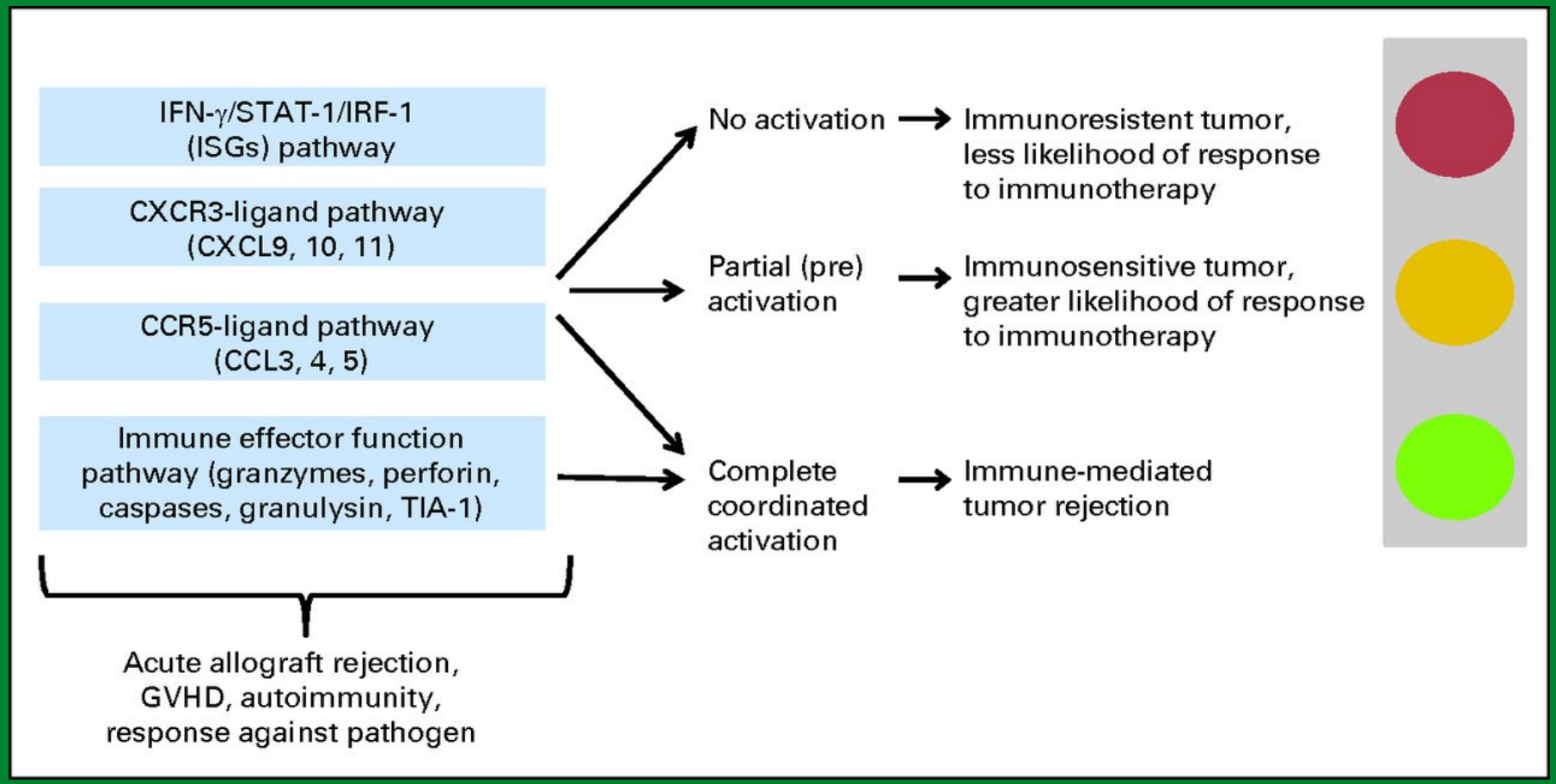


Grupp SA et al. N Engl J Med 2013;368:1509-1518.

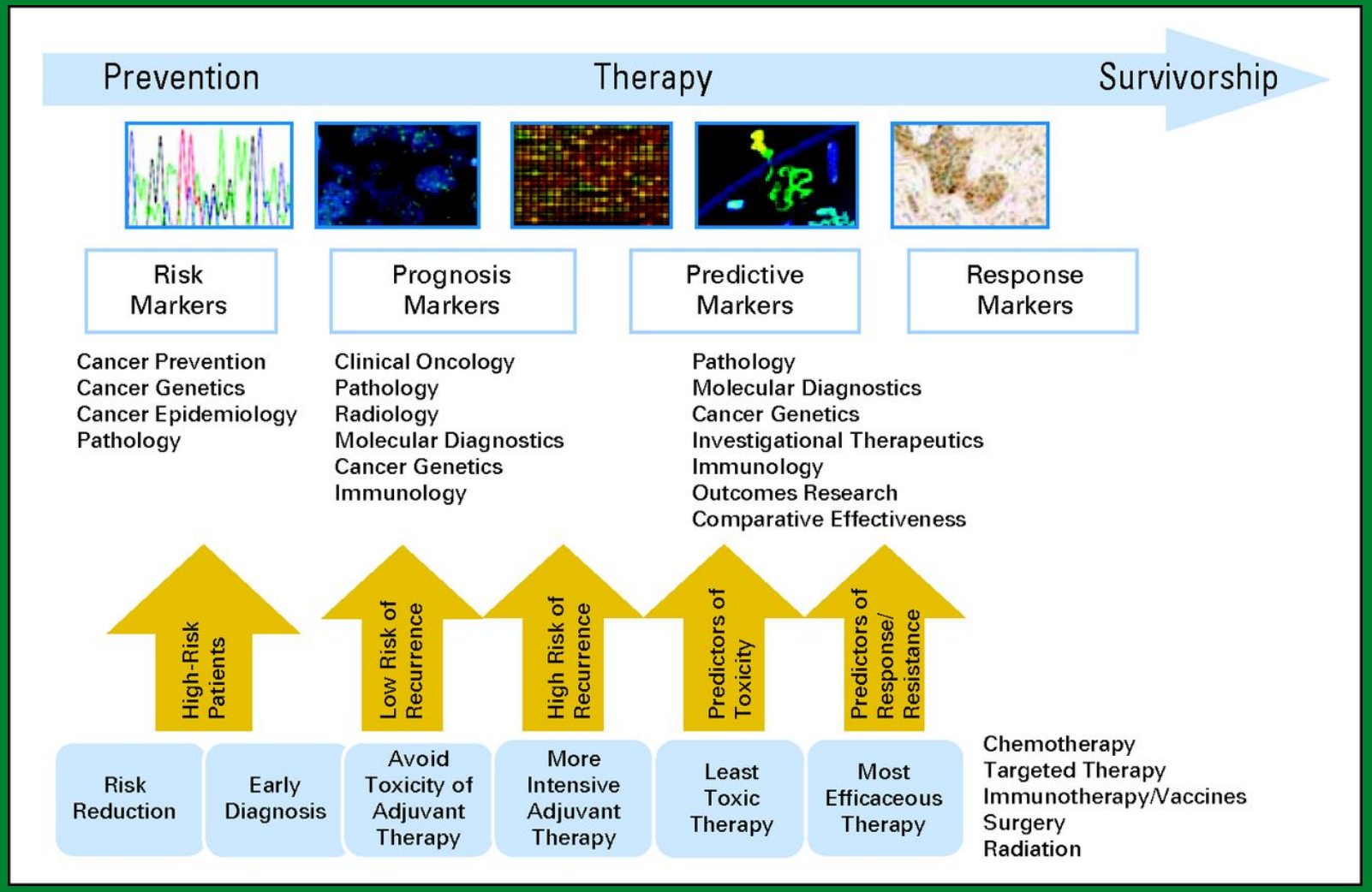
PRE-EXISTING IMMUNE-REACTIVITY

1. Tumor phenotype microarray RNA study before therapy (MAGE-3 or CTLA-4) in different tumors (melanoma, NSCLC and CRC) found a 61/81 gene signature that predicted TSD (Tissue Specific Destruction) designated as ICR (Immunological Constant of Rejection)
2. Cancer phenotype profile associated with immune responsiveness indicated a naturally occurring phenomenon, present in tumor induced immune destruction, acute allograft rejection, GVHD and autoimmune diagnosis

Ulloa Montoya et al. Predictive gene signature in MAGE-A3 Antigen- Specific Cancer Immunotherapy. J Clin Oncol 2013;31:2388-95


IFN-γ/STAT-1/IRF-1 (ISGs) pathway
CXCR3-ligand pathway (CXCL9, 10, 11)
CCR5-ligand pathway (CCL3, 4, 5)
Immune effector function pathway (granzymes, perforin, caspases, granulysin, TIA-1)
Acute allograft rejection, GVHD, autoimmunity, response against pathogen
No activation
Immunoresistent tumor, less likelihood of response to immunotherapy
Partial (pre) activation
Immunosensitive tumor, greater likelihood of response to immunotherapy
Complete coordinated activation
Immune-mediated tumor rejection

The personalized cancer care continuum.



Meric-Bernstam F et al. JCO 2013;31:1849-1857

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JOURNAL OF CLINICAL ONCOLOGY ASCO

Five key lessons for the future of personalized cancer medicine, by Francis Collins *(Nature 2010)*

- Free and open access to genome results to develop & achieve immediate analysis of massive data
- Technology development for sequencing and functional genomics must continue be invested
- Success depend on accurate identification of genetic and environmental risk factors and the ability to use this information in the real world
- New emerging drug targets require new paradigms (N=1)
- Good policy decisions: privacy, education, financing, etc.

PERSONALIZED CANCER MEDICINE

REVIEW THE PRESENT TO ENABLE THE FUTURE:

➔ CURE IS THE BEST COST-SAVING CHOICE

- NECESSARY DEVELOPMENTS: AVAILABILITY OF ROUTINE FACILITIES, RAPID AND EFFECTIVE TRIALS, ACCELERATING CANCER RESEARCH PROGRESS
- COST - EFFECTIVENESS ANALYSIS: GAIN BETTER OUTCOMES OF CARE AND SPEND IN HIGHLY VALUED TREATMENTS.
- LIMITS ON CARE: SUSTAINABILITY AND COST CONTAINMENT
- EQUITABLE PARTICIPATION OF PATIENTS, SCIENTISTS, DOCTORS, AND REPRESENTATIVES OF INDUSTRY, ECONOMY AND HEALTH CARE SYSTEM

PERSONALIZED MULTIDISCIPLINARY CANCER MEDICINE= ONCOLOGY PLATFORM

Development of a new integrated paradigm based in a customized patient care, through a multi-interdisciplinary approach,

Sitting in the confluence of two clinical and genomic analysis: Patient and Tumor,

Require changes in the teaching, organizing, delivering and financing of the health system.