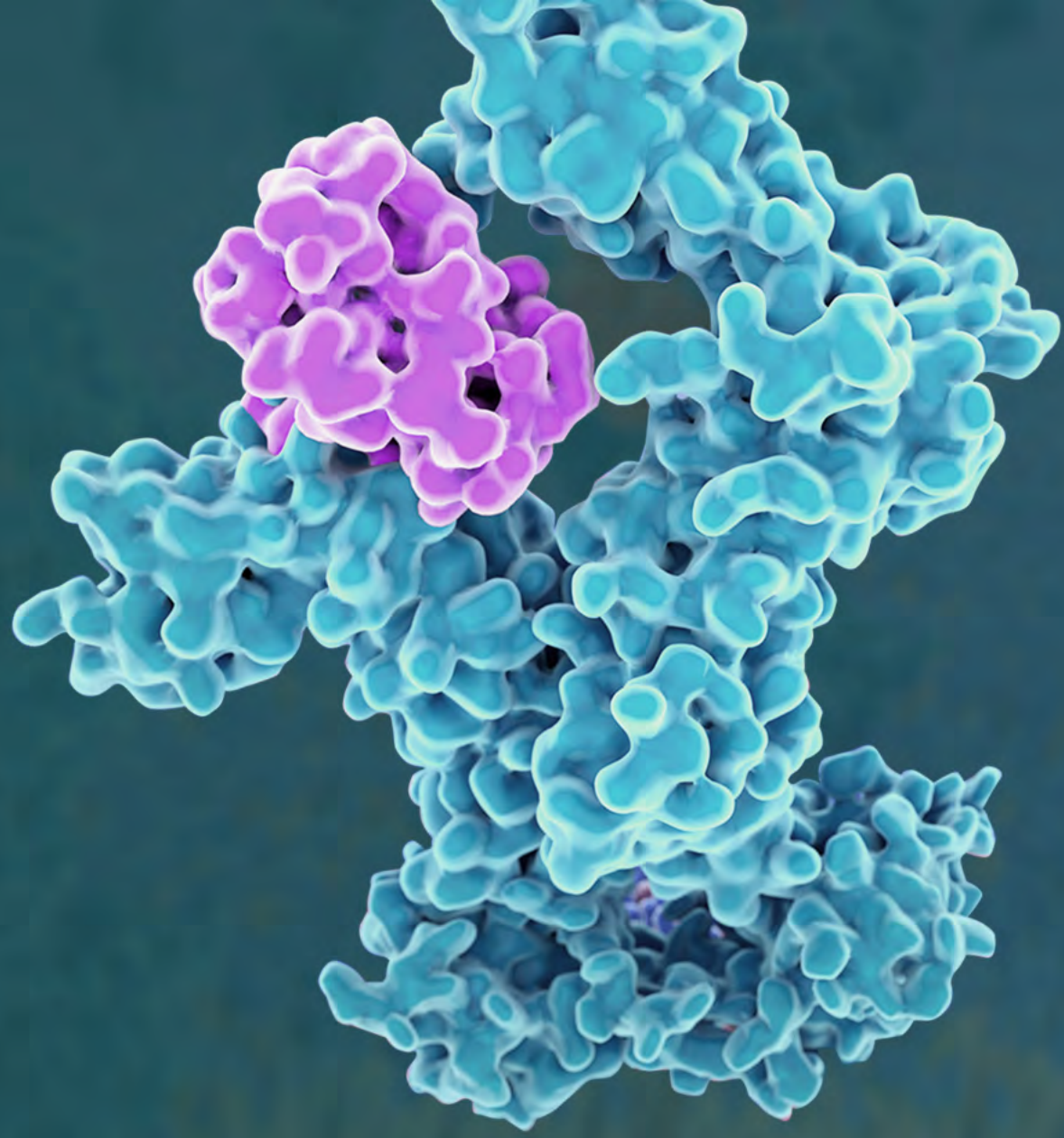
KOL Call on MDNA55 for the Treatment of Recurrent Glioblastoma (rGBM)



December 11, 2020, 11:00am EST

MEDICENNA

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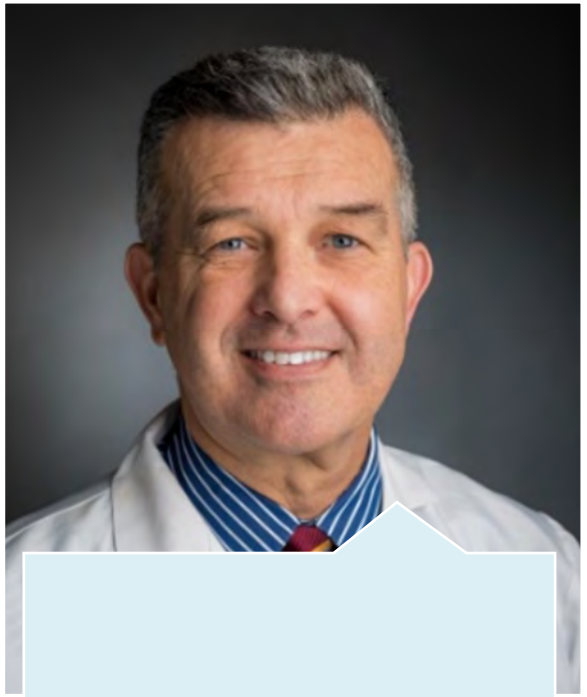
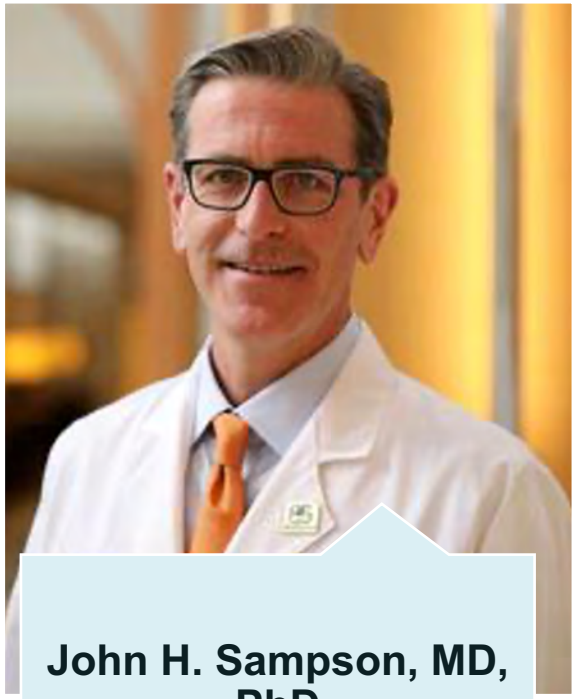
Speaker Panel



David A. Reardon, MD

Professor of Medicine Harvard Medical School, Clinical Director of the Center for Neuro-Oncology, Dana-Farber Cancer Institute



John H. Sampson, MD, PhD

Robert H. and Gloria Wilkins Distinguished Professor Dept of Neurosurgery, Co-leader Duke Cancer Institute Neuro-Oncology program, Duke University School of Medicine

Ruthie Davi, PhD

Vice President, Data Science at Acorn AI, a Medidata company

Amy McKee, M.D.

VP, Regulatory Consulting Services, Parexel

Agenda

- Welcome message and introduction of KOLs
- GBM Background & Need for New Therapies *(David Reardon)*
- Clinical Efficacy of MDNA55 in rGBM *(John Sampson)*
- Benefits of a Propensity Matched External Control Arm (ECA) *(Ruthie Davi)*
- Incorporation of an ECA in a Planned rGBM Registration Trial *(Amy McKee)*
- Medicenna Overview *(Fahar Merchant)*
- Q&A

GBM Background & Need for New Therapies

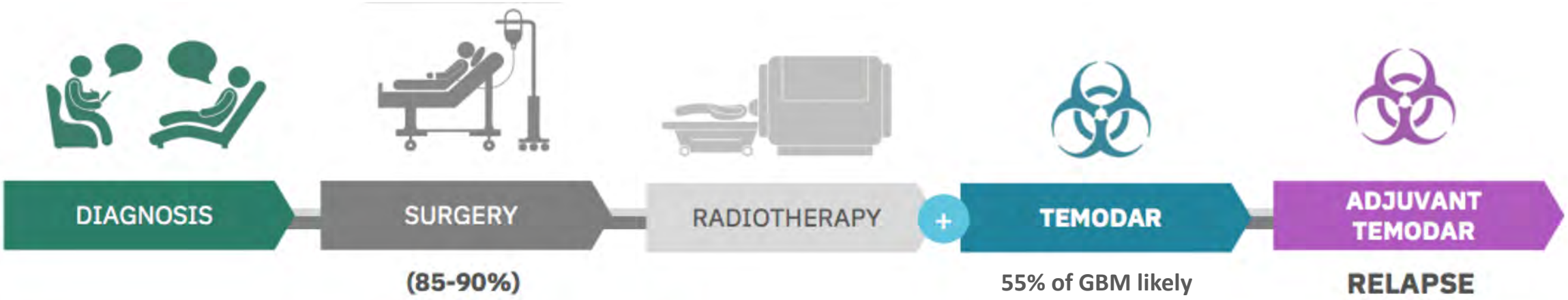
David A. Reardon, MD
Professor of Medicine Harvard Medical School, Clinical Director of the Center for Neuro-Oncology Dana-Farber Cancer Institute

Therapeutic Challenges of GBM

- GBM is the most aggressive primary brain tumor characterized by rapid proliferation of undifferentiated cells, extensive infiltration and a high propensity to recur
- Blood Brain Barrier (BBB) blocks transport of large molecular therapies to the tumor
- Recurrent GBM patients have a compromised immune system following chemo-radiation which is further exacerbated by steroid use
- Tumor microenvironment (TME) comprises a majority of GBM tumor mass; the TME provides an immunosuppressive environment by supplying growth factors and nutrients to support tumor growth and survival[1].
- GBM is heterogeneous with a highly complex tumor biology
    - IDH mutated vs. wild-type
    - MGMT promoter methylated vs. unmethylated

1) Kennedy et al, JCO, 2013

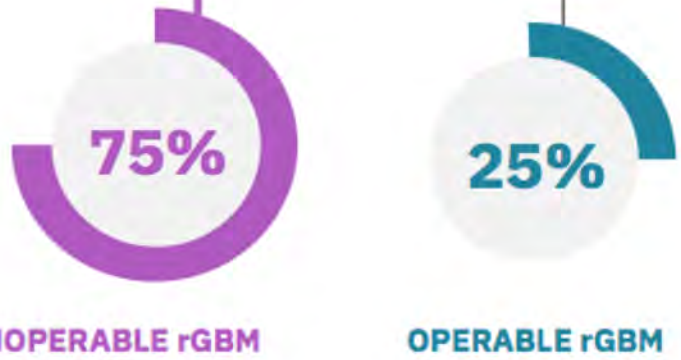
Current Treatment Strategies for GBM are Ineffective



Glioblastoma (GBM) & IL4 Receptor

- Uniformly fatal – virtually all tumors will recur
- New treatment strategies are needed
- IL4 receptor (IL4R) is overexpressed in GBM cells and the tumor microenvironment making it a promising target for GBM treatments

MDNA55 Treatment
Direct infusion into tumor
via convection enhanced delivery (CED)



* Expression of the DNA repair protein O6-methylguanine-DNA methyltransferase (MGMT) is responsible for resistance to Temodar used in GBM treatment.

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Treatments for GBM and rGBM

Very high Unmet Need - No available treatment options for GBM have a meaningful survival benefit

Newly Diagnosed GBM

- Treatments focus on preserving quality of life, neurological function, extending survival
- Standard of care (SOC) consists of:
  - Maximal resection possible
  - Radiotherapy
  - Temozolomide
  - Gliadel
  - Optune

Recurrent GBM*

- Virtually all patients relapse
- No defined SOC
- Therapies include:
  - Avastin
  - Lomustine
  - Gliadel
  - Optune
  - Salvage therapies (radiotherapy, temozolomide)
  - Experimental therapies

* Treatment options following recurrence are very limited and outcome generally unsatisfactory. The median overall survival (OS) is estimated to be 6-10 months with approved therapies

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Key Prognostic Factors for GBM

CLINICAL FACTORS	PROGNOSTIC ASSOCIATION	
	FAVORABLE	POOR
Younger Age	◉	
Older Age		X
Higher KPS	◉	
Lower KPS		X
Tumor resectability	◉	
Not eligible for resection		X

GENETIC FACTORS	PROGNOSTIC ASSOCIATION	
	FAVORABLE	POOR
Secondary GBM	◉	
Primary *de novo* GBM		X
IDH gene Mutation	◉	
IDH gene Wild-type		X
MGMT methylated	◉	
MGMT unmethylated		X
IL4R Low-expression	◉	
IL4R Over-expression		X

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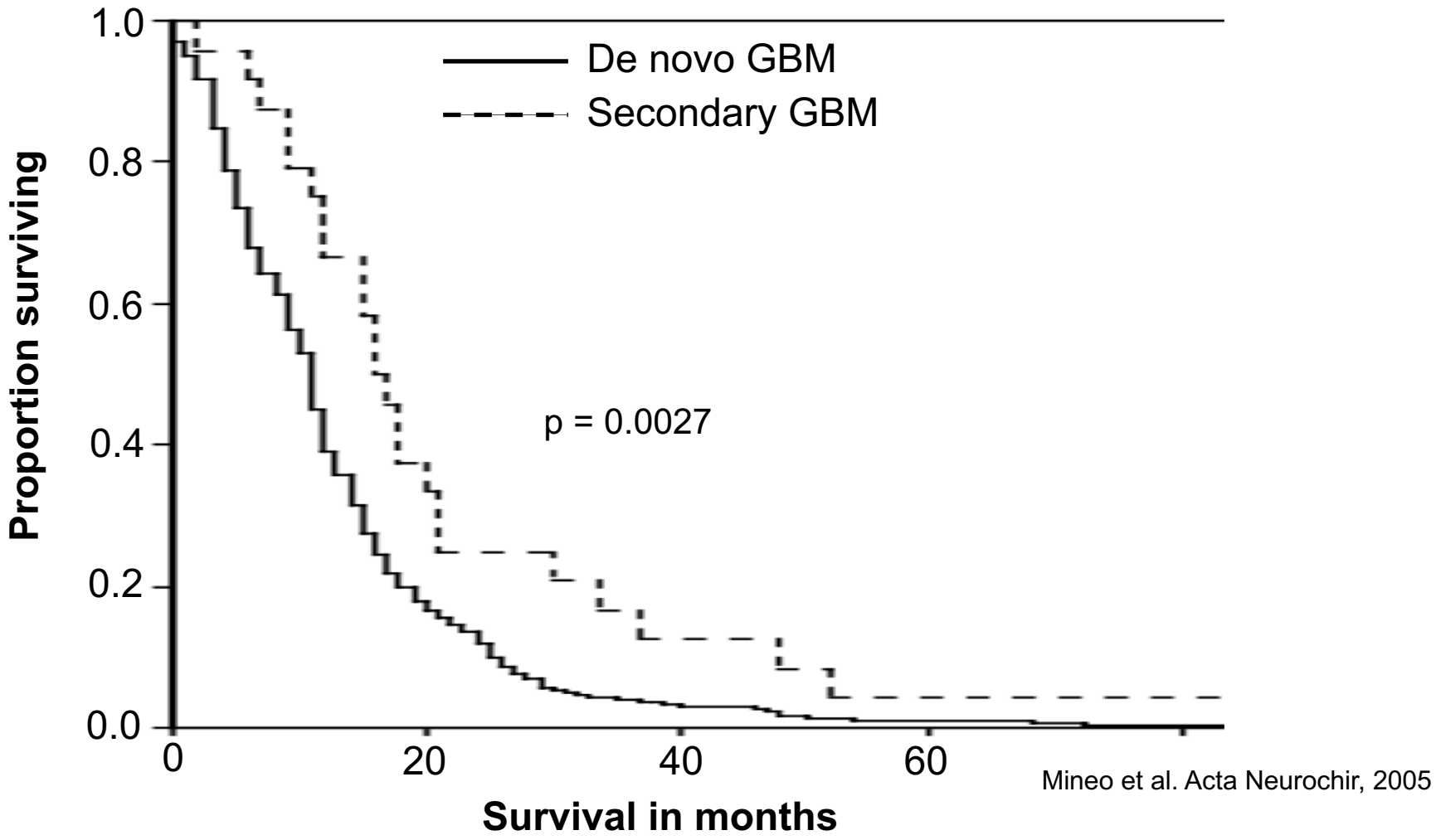
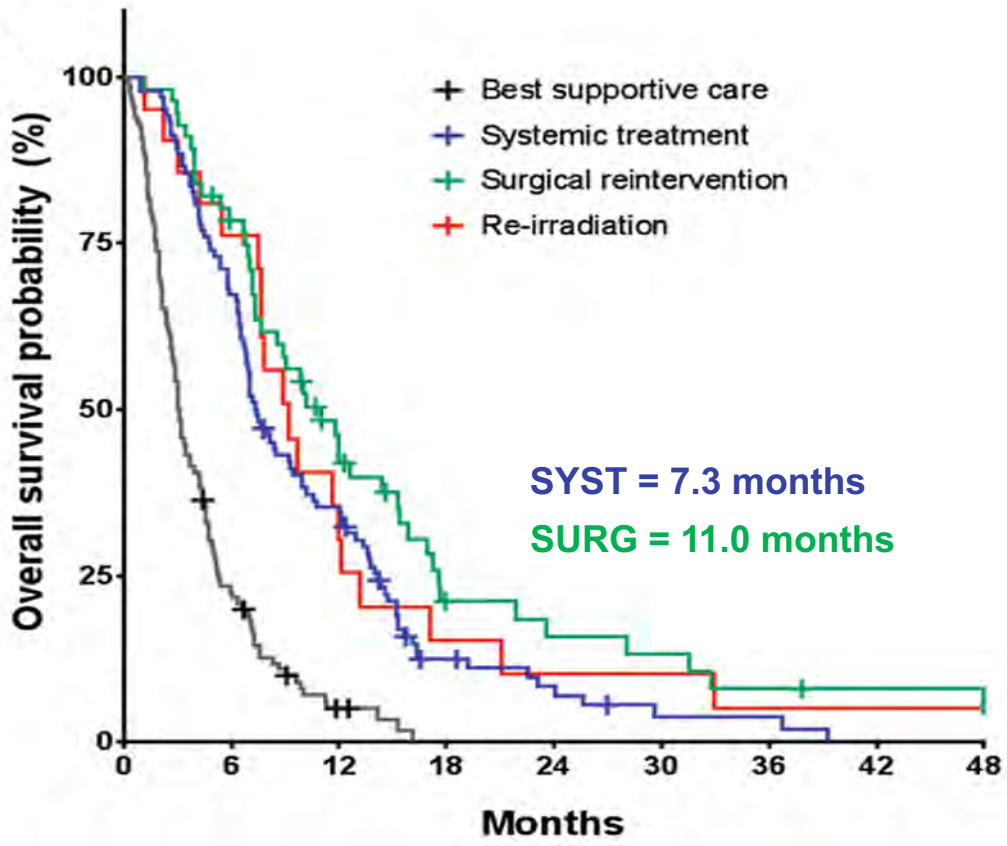
Primary *de novo* GBM is Associated with Poor Survival



Mineo et al. Acta Neurochir, 2005

Data of 340 patients with newly-diagnosed GBM were retrospectively analyzed. GBM type (*de novo* or secondary) was suggested to influence survival by univariate analysis.

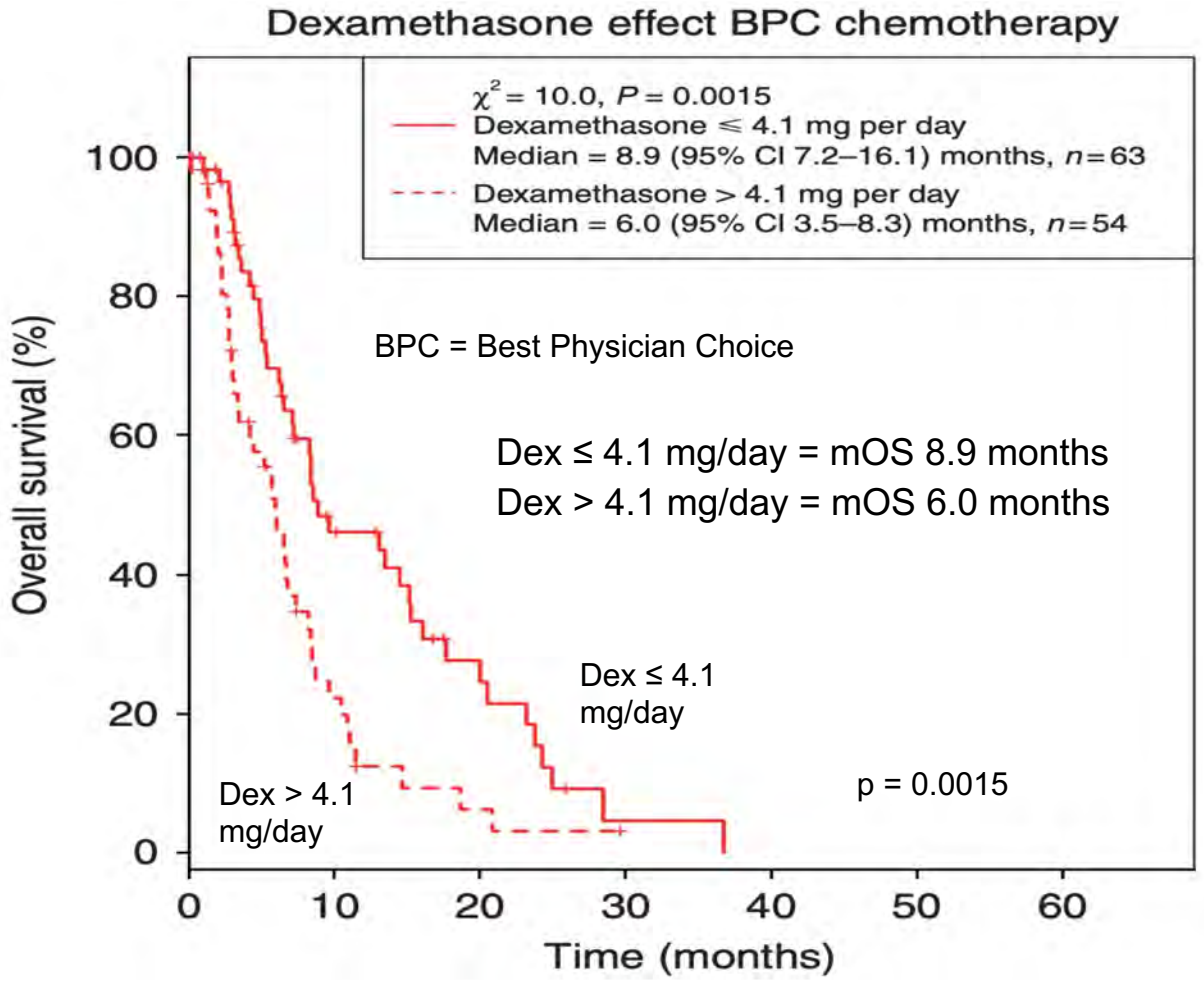
No Surgery at Relapse Lowers Survival



Van Linde et al. J. Neurooncol, 2017

Data of 299 patients recurrent GBM were retrospectively analyzed. Different treatments were suggested to influence survival by univariate analysis.

High Steroid Use Negatively Impacts Survival



Wong et al. BJC, 2015

Overall Survival with respect to dexamethasone requirement from recurrent GBM subjects enrolled in the phase III with Best Standard of Care (BSC) chemotherapy (NCT00379470).

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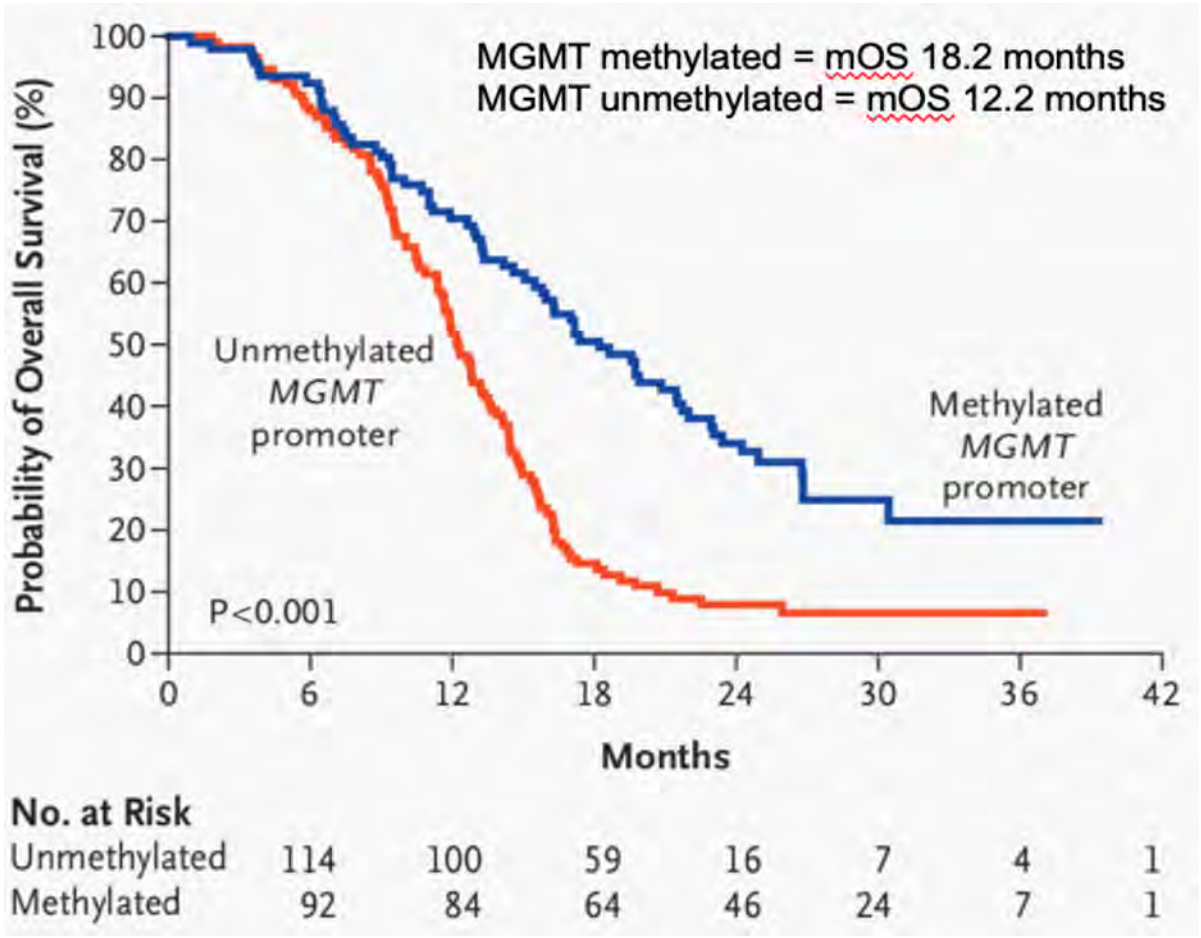
Unmethylated *MGMT* Promoter Associated with Poor Prognosis



Hegi et al. NEJM, 2005

Overall survival of 206 patients with newly diagnosed GBM for whom MGMT status could be evaluated irrespective of treatment assignment (RT or RT/TMZ).

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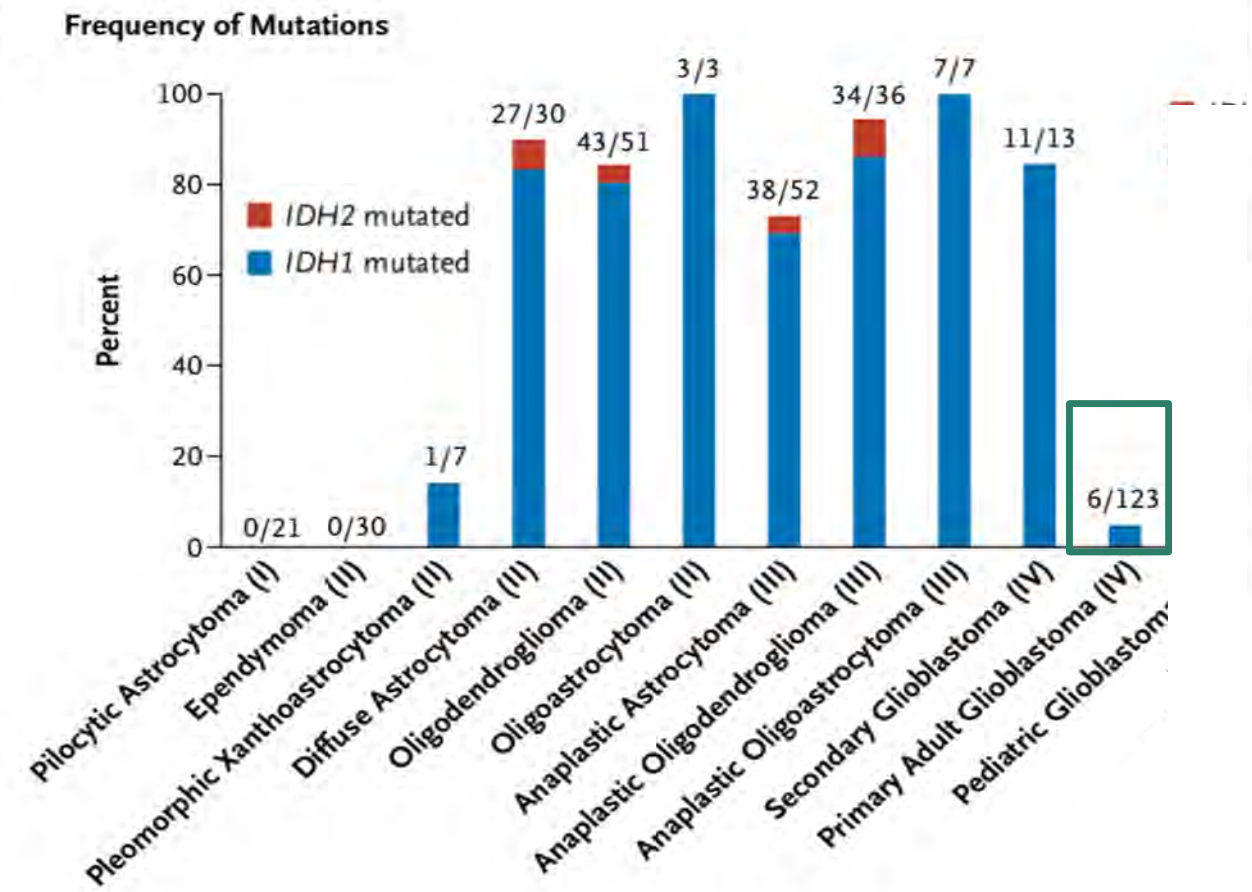
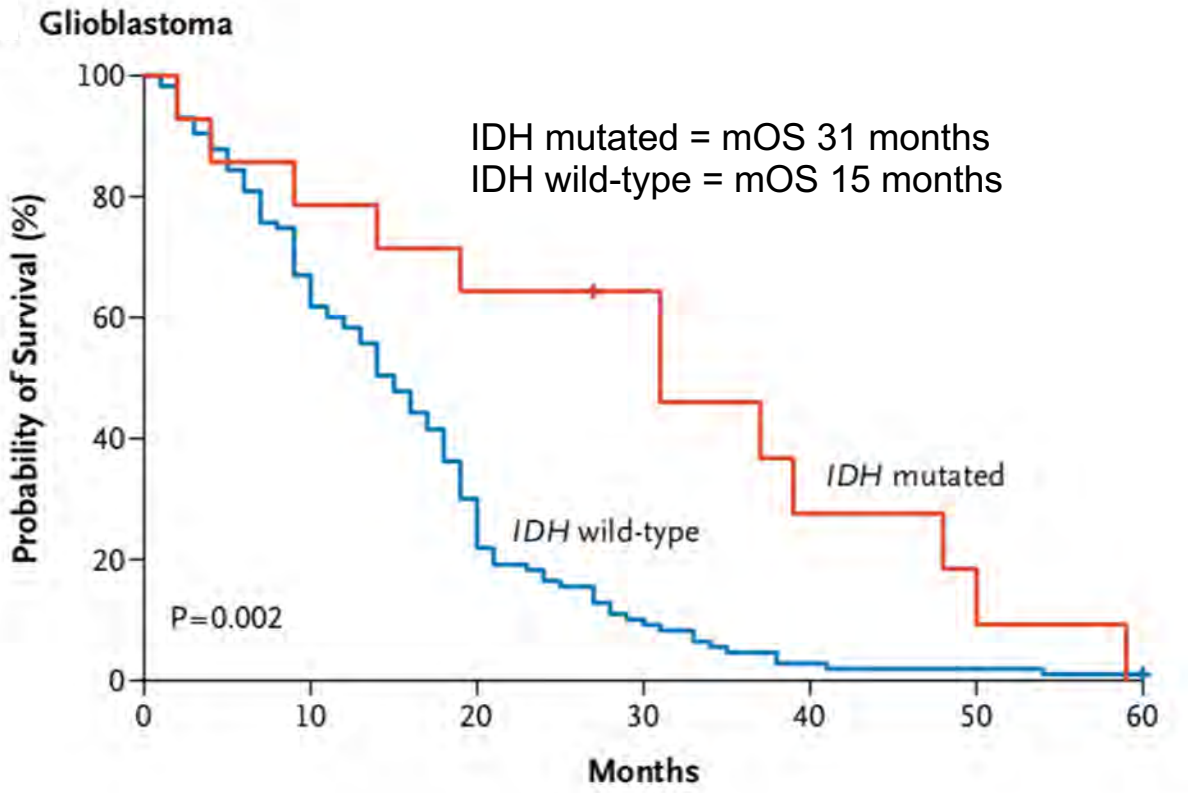
GBM with *IDH* Wild-Type Status Associated with Aggressive GBM



Number and frequency of IDH1 and IDH2 mutations in gliomas and other types of tumors. Roman numerals in parentheses are the tumor grades according to histopathological and clinical criteria established by the World Health Organization.

Yan et al. NEJM, 2009



Survival of adult patients with GBM with or without IDH gene mutations. Median survival was 31 months for the 14 patients with mutated IDH1/2, as compared with 15 months for the 115 patients with wild-type IDH1/2

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IL4R is Expressed in Majority of Brain Tumors, Including GBM

> 300 Patient Biopsies Analyzed Show IL-4R Over-Expression[1-7]

Glioblastoma **76%**	**Mixed Adult Glioma** **>83%**	**Mixed Pediatric Glioma** **76%**	**Pediatric DIPG** **71%**
Medulloblastoma **100%**	**Adult Pituitary Adenoma** **100%**	**Meningioma** **77%**	**Normal Brain Tissue** **0%**

1. Joshi BH, et. al. Cancer Res 2001;61:8058-8061.
2. Puri RK, et. al., Cancer Res 1996;56:5631-5637.
3. Kawakami M, et. al., Cancer. 2004 Sep 1; 101(5):1036-42.
4. Berlow NE, et al. PLoS One. 2018 Apr 5; 13(4):e0193565.
5. Joshi BH, et. al. British J of Cancer (2002) 86, 285 –291.
6. Chen L, et al. Neurosci Lett. 2007 Apr 24; 417 (1):30-5.
7. Puri S, et. al., Cancer. 2005 May 15; 103(10):2132-42.

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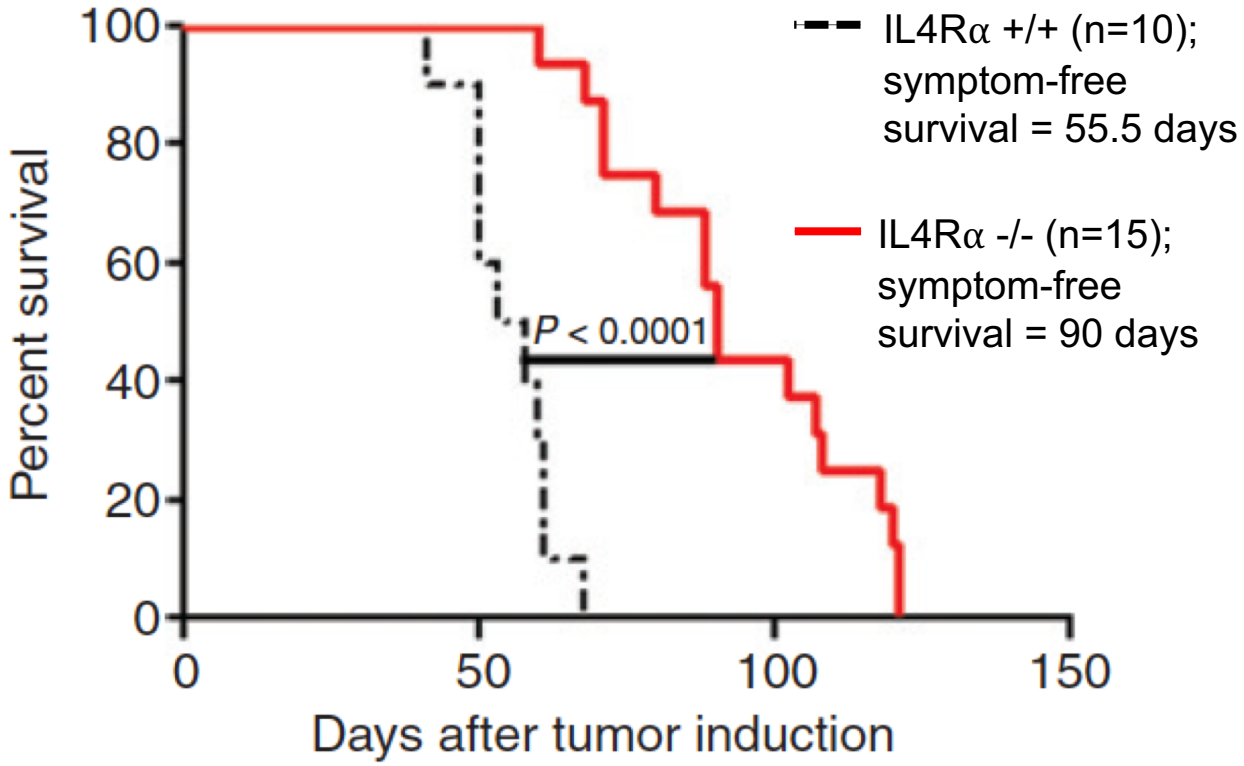
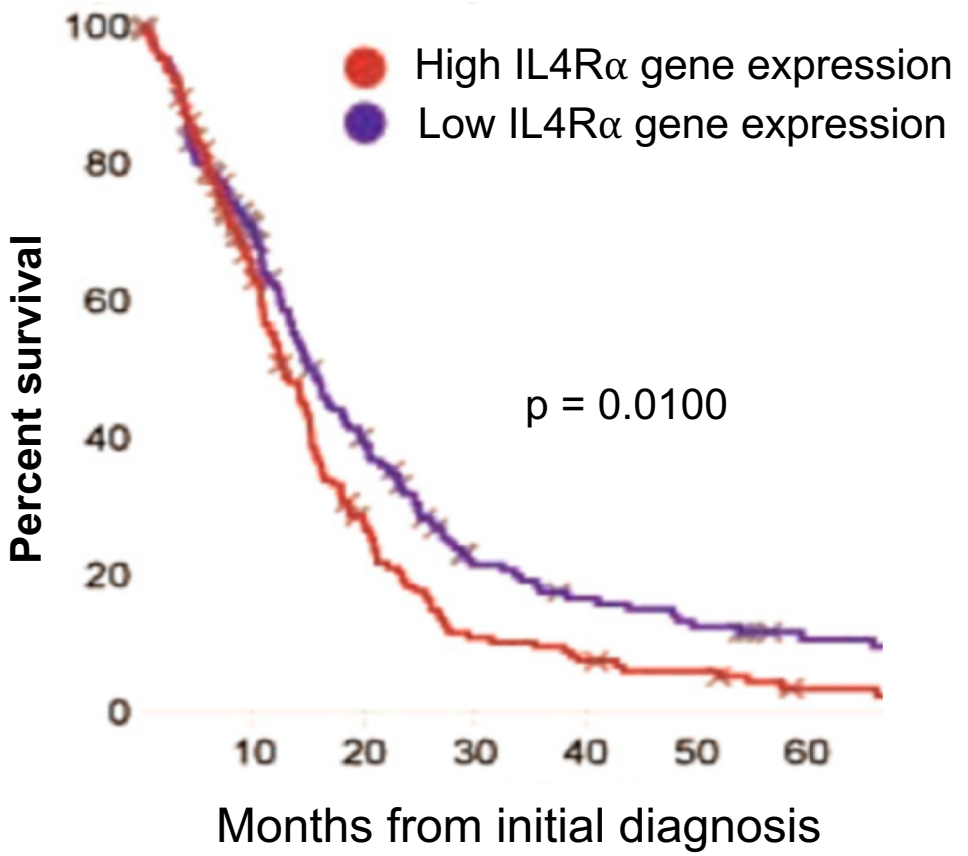
High IL4Rα Expression Predicts Poor Survival in GBM

Survival in BALB/c Glioma Mouse Model



Kohanbash G et al. Cancer Res 2013;73:6413-6423

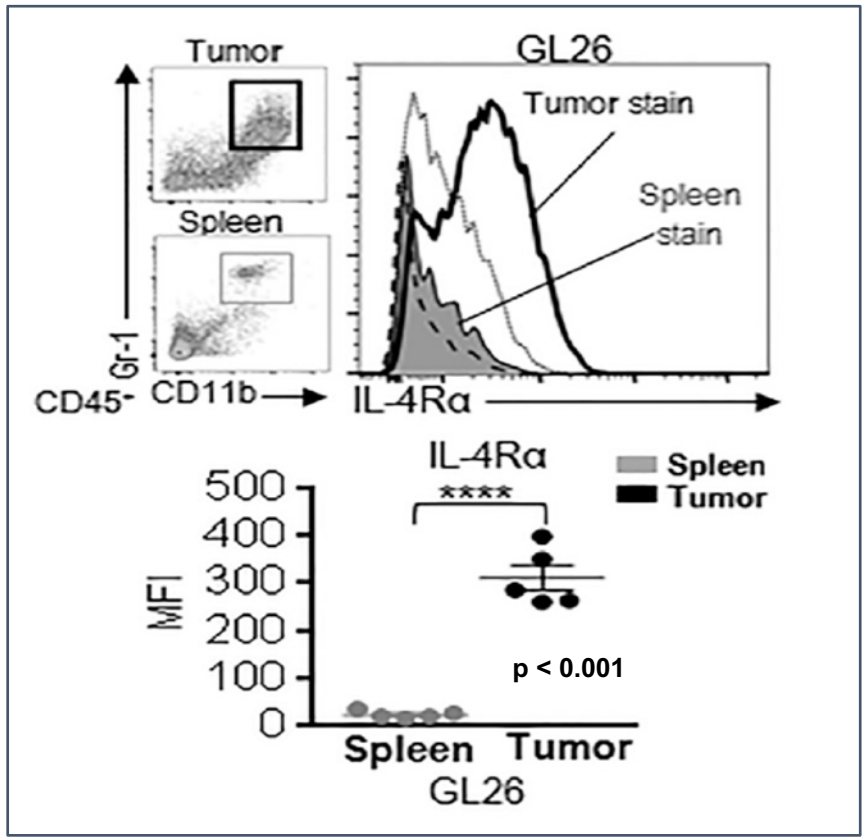
Survival in GBM Patients (N=348) - TCGA



D'Alessandro G, et al. Cancers (Basel). 2019

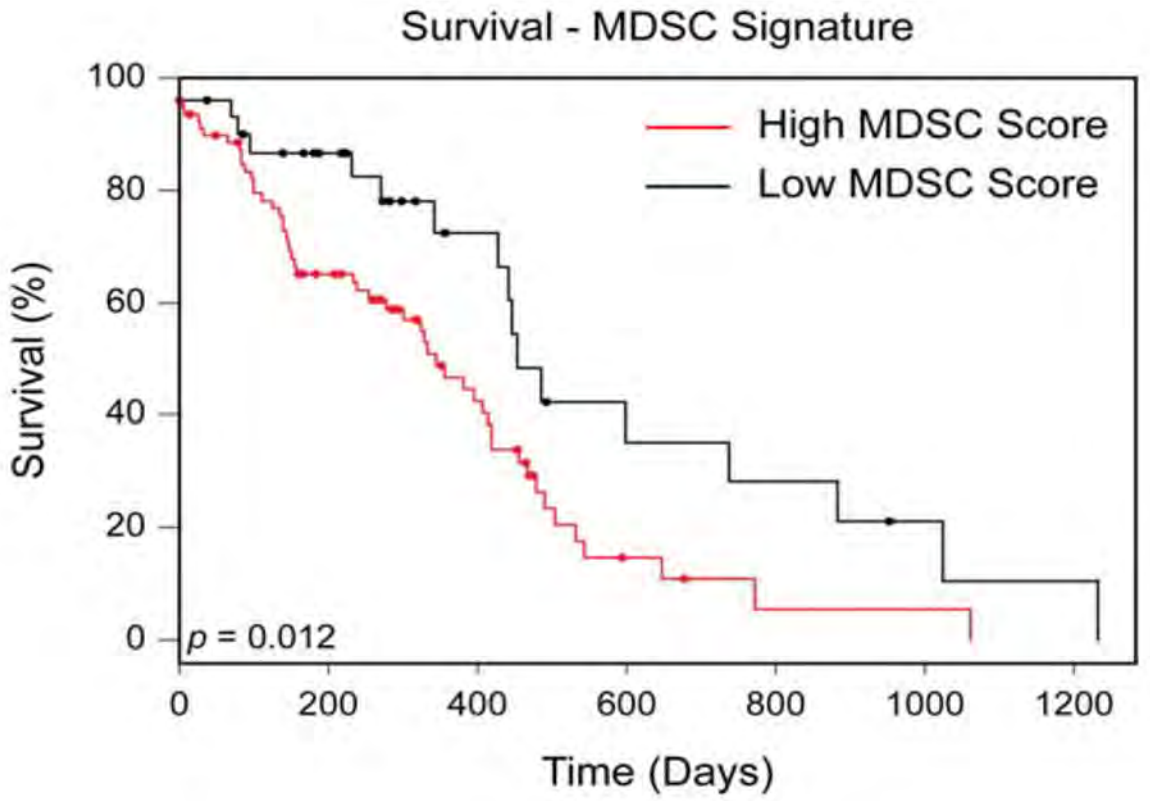
TME-Infiltrating MDSCs Express IL4R and Predict Poor Survival in GBM

TME-MDSCs show 12-fold increase in IL-4Rα expression compared to splenic myeloid cells



Surface expression of IL-4Ra on tumor-infiltrating and splenic CD11b+/Gr-1+ MDSCs from GL26 tumor-bearing mice.

Kamran N, et. al., (2017). Mol Ther 25:232-248



MDSC gene signature (based on the combined positive expression of CD11b, CD33, CD45, CD244, and CXCR2) negatively correlates with GBM patient prognosis. Statistical significance of survival was based on log-rank analysis. (N=112)

Otvos B et. al., (2016). Stem Cells 34:2026–2039

Prior Ph 3 Trials in GBM and rGBM

Failed Phase 3 Trials in rGBM with OS as Primary Endpoint (conducted between 2003 – 2019)

Agent (Sponsor)	Target/Class	Study Design	Control Arm	Total Subjects Enrolled
Edotecarin (Pfizer)	Topoisomerase I inhibitor	1:1 randomization	TMZ, Camustine, or LOM	118 (59 in SOC)
IL13-PE38QQR (INSYS Therapeutics)	IL13R-targeted toxin	2:1 randomization	Gliadel	296 (104 in SOC)
Bevacizumab (EORTC)	VEGF inhibitor	2:1 randomization	LOM	437 (149 in SOC)
Tumor Treating Fields (Novocure)	Device	1:1 randomization	Best active chemotherapy	237 (117 in SOC)
Toca 511 + Toca FC (Tocagen)	Retroviral vector	1:1 randomization	TMZ, LOM, or BEV	403 (202 in SOC)
VB-111 (VBL Therapeutics)	Angiogenesis inhibitor	1:1 randomization	BEV	256 (128 in SOC)
Nivolumab (BMS)	PD-1 inhibitor	1:1 randomization	BEV	369 (185 in SOC)

Overcoming the Pitfalls of Prior GBM Ph 3 Clinical Studies

- Ph 3 studies have less restrictive inclusion/exclusion criteria compared to Ph 2 due to need for faster enrolment
- False efficacy signal in Ph 2 (especially single arm studies) leading to Ph 3 efficacy failure
    - For locally administered drugs, there was no method to ensure efficient drug delivery
    - For orally or systemically administered drugs, BBB blocks transport of therapy to tumor
    - Absence of rational biomarkers to predict benefit
    - Inadequacy to recognize importance of the TME; need therapy to target both TME and the tumor
- A major contributor to the high failure rate is inadequate Ph 2 program that provides sub-optimal information for the “go/no go” decision to move to Ph 3 and the design of the Ph 3 trial

Clinical Efficacy of MDNA55 in rGBM

John H. Sampson, MD, PhD
Robert H. and Gloria Wilkins Distinguished Professor Dept of Neurosurgery and
President of the Private Diagnostic Clinic
Duke University School of Medicine

Duke University
School of Medicine

MDNA55: A Targeted Immunotherapy for GBM

MDNA55

Targets the IL4R, which is expressed in brain tumors and in the tumor microenvironment (TME), but not the healthy brain

Highly Selective

Avoids off-target toxicity

Disrupts the TME

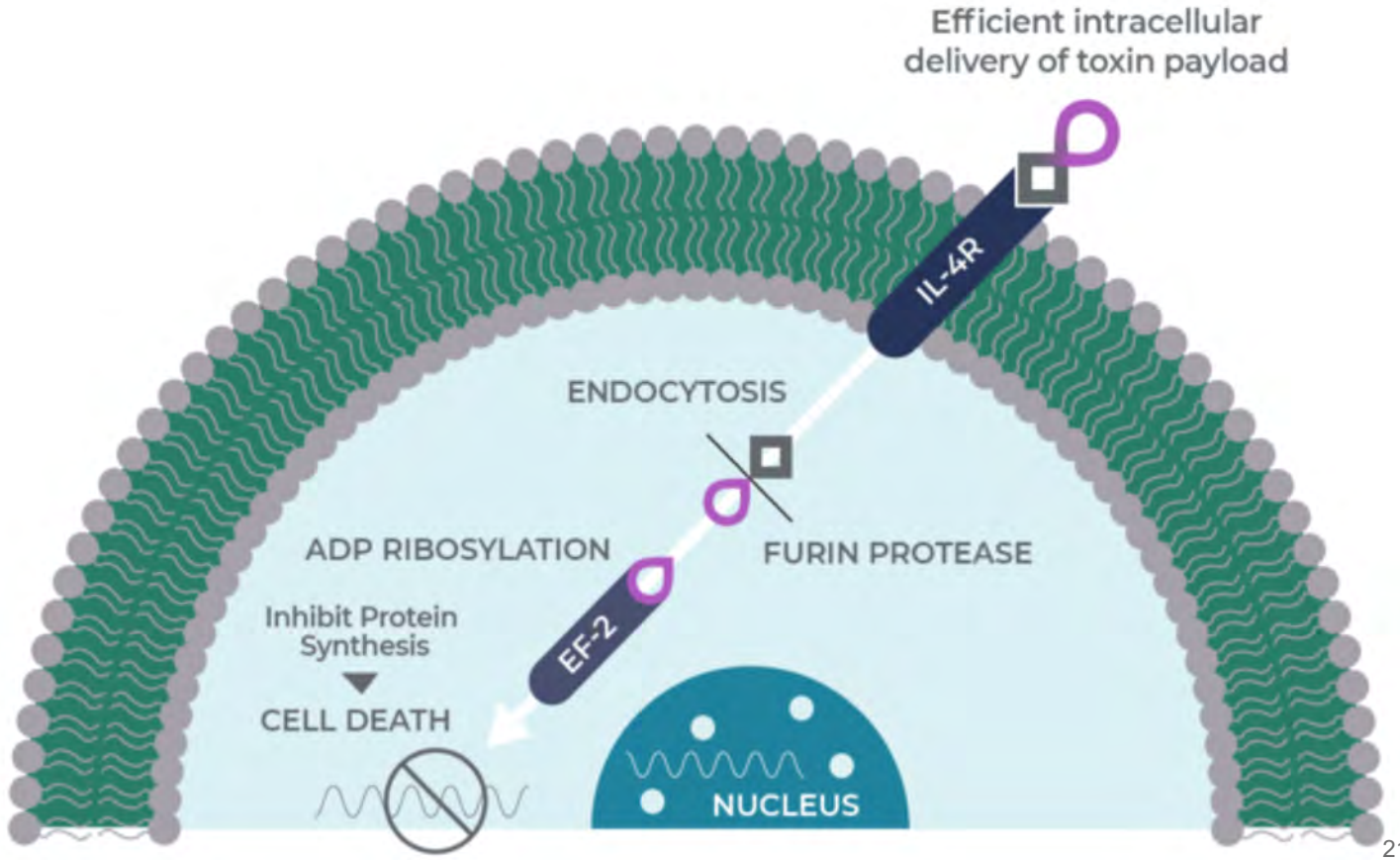
By targeting IL4R positive cells found throughout the TME, MDNA55 unblinds the tumor to the body's immune system

Sustained Immune Memory Response

Anti-tumor immunity is initiated and remains active after MDNA55 is cleared

Targeting Domain
Circularly Permuted Interleukin-4 (cpIL-4)

Lethal Payload
Catalytic domain of *Pseudomonas* Exotoxin A (FDA approved Moxetumomab pasudotox)



MDNA55-05 Phase 2b Study Design

Open-Label Single Arm Study in Recurrent GBM Patients (n=47) (NCT02858895)

ELIGIBILITY

- Adults ≥ 18 yrs
- De novo GBM
- 1st or 2nd relapse
- No resection
- KPS ≥ 70
- IDH wild-type only
- Retrospective IL4R analysis from initial Dx

PLANNING

- MRI - tumor size and location
- Optimal catheter trajectory

TREATMENT

- Image-guided catheter placement
- Monitor real-time drug distribution with co-infusion of Magnevist ®
- Single infusion (median 26.5 hrs.)
- Conc. range: 1.5-9.0 µg/mL
- Volume range: 12-66 mL
- Total Dose range: 18-240µg
- Transient low-dose BEV allowed for symptom control and/or steroid sparing (6 and 9 µg/mL cohorts only)

ENDPOINTS

1º Endpoint

- OS

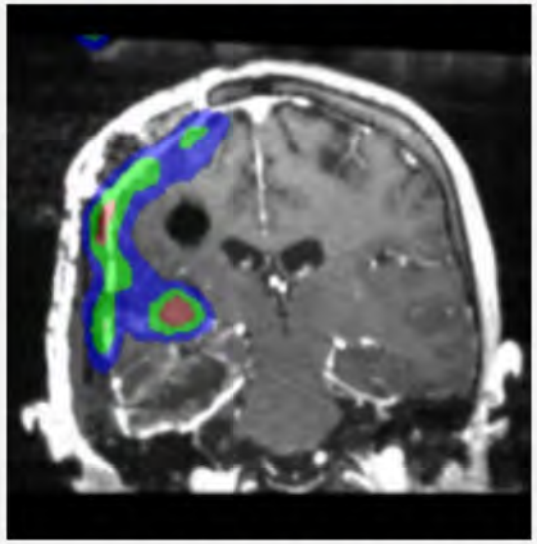
2º Endpoint

- ORR
- PFS
- OS vs. IL4R expression
- Safety

High-Flow Image Guided CED Improves Distribution

PAST STUDIES

1st Generation CED



Inaccurate catheter placement

Drug leakage due to backflow

Inadequate tumor coverage

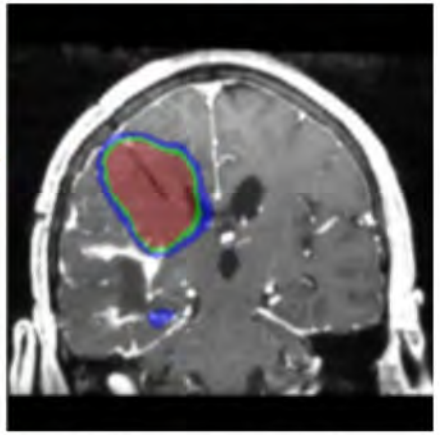
CURRENT STUDIES

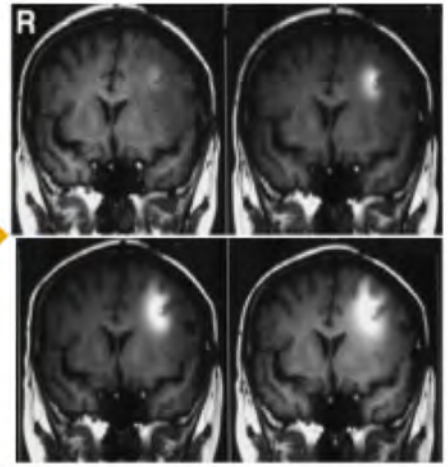
2nd Generation High-flow CED

Image-guided catheter placement

New catheters prevent backflow

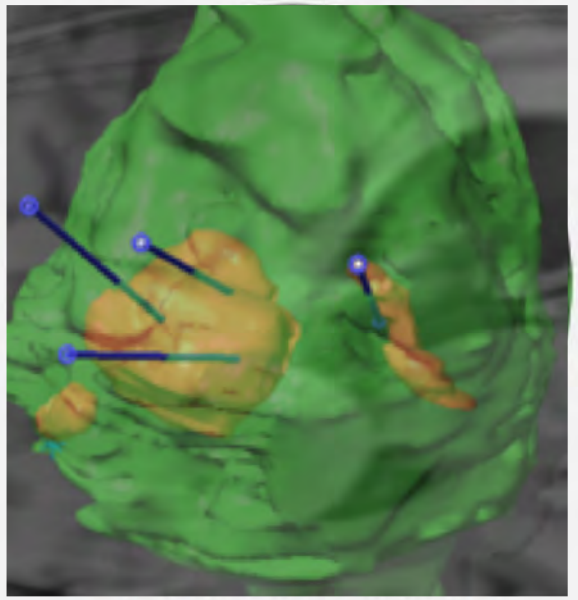
Real-time monitoring ensures tumor coverage





Saito and Tominaga (2012), Neurol Med Chir (Tokyo) 52, 531

3D IMAGE FROM PATIENT IN CURRENT CLINICAL STUDY



Tumor Drug Coverage

MDNA55-05 Demographics and Safety

Patient Demographics	N=44
Age (median, range)	55 years (34 – 77)
Sex (Male)	27 / 44 (61%)
KPS at Enrolment: 70, 80 90, 100	22 / 44 (50%) 22 / 44 (50%)
De novo GBM	44 / 44 (100%)
Poor candidates for repeat surgery	44 / 44 (100%)
IDH Wild-type	37 / 37 (100%)
Unmethylated *MGMT*	23 / 40 (58%)
IL4R over-expression	21 / 40 (53%)
Steroid use > 4mg/day	23 / 44 (52%)
Max Tumor Diameter*	29.6 mm (8 – 59)
# Prior Relapse: 1 , 2	35 (80%) , 9 (20%)

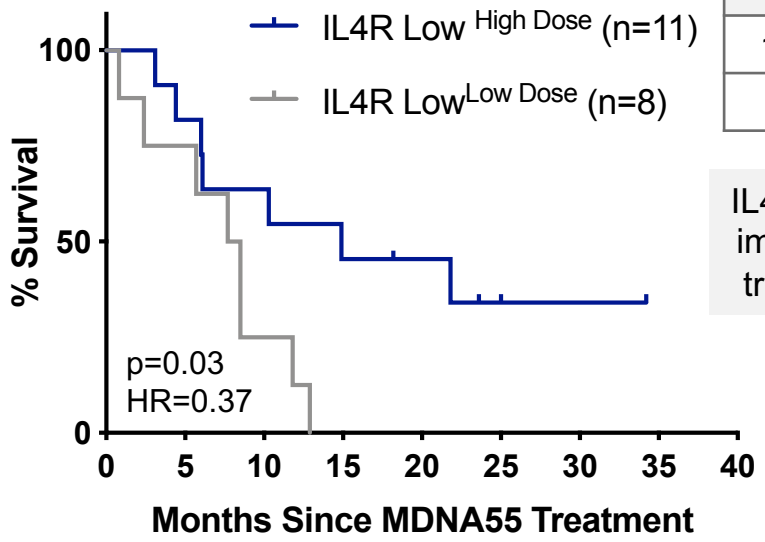
*Based on central tumor assessments

MDNA55-05 Safety Profile

- No systemic toxicities
- No clinically significant laboratory abnormalities
- Drug-related AEs were primarily neurological/aggravation of pre-existing neurological deficits characteristic with GBM; manageable with standard measures.

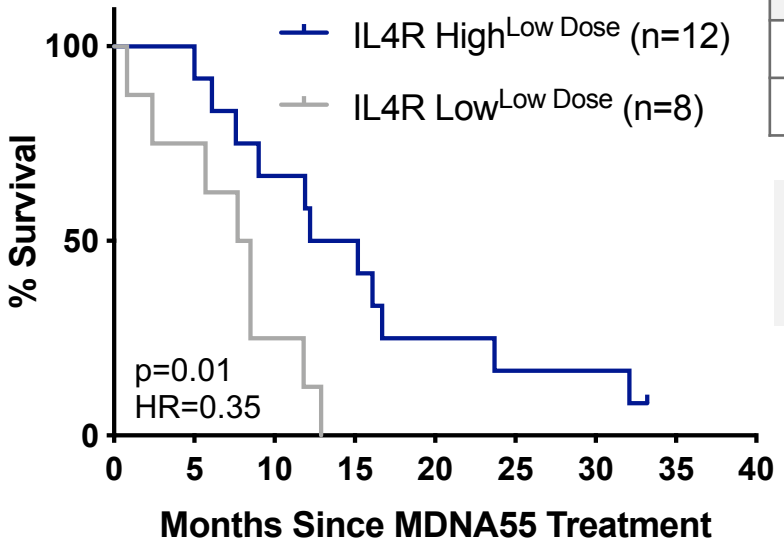
Related AEs ≥ Grade 3 Occurring in ≥ 5% Subjects	Total N=47 [n (%)]
# of Subjects	10 (21.3)
Nervous system disorders	10 (21.3)
Brain Edema / Hydrocephalus	4 (8.5)
Hemiparesis	3 (6.3)
Seizure	3 (6.3)

Effect of MDNA55 Dose and IL4R Expression on Survival



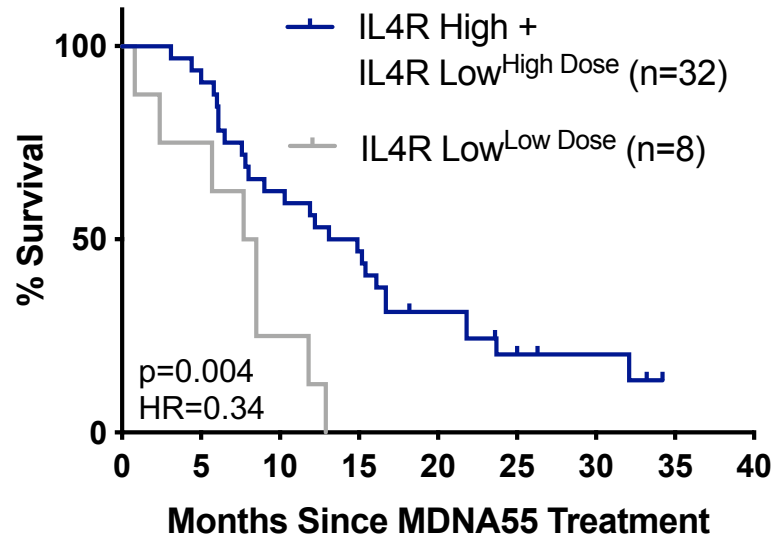
mOS	OS-12	OS-24
14.9	55%	34%
8.1	13%	0%

IL4R Low group showed improved survival when treated with high dose.



mOS	OS-12	OS-24
13.7	58%	17%
8.1	13%	0%

At low dose, only High IL4R expression improved survival.



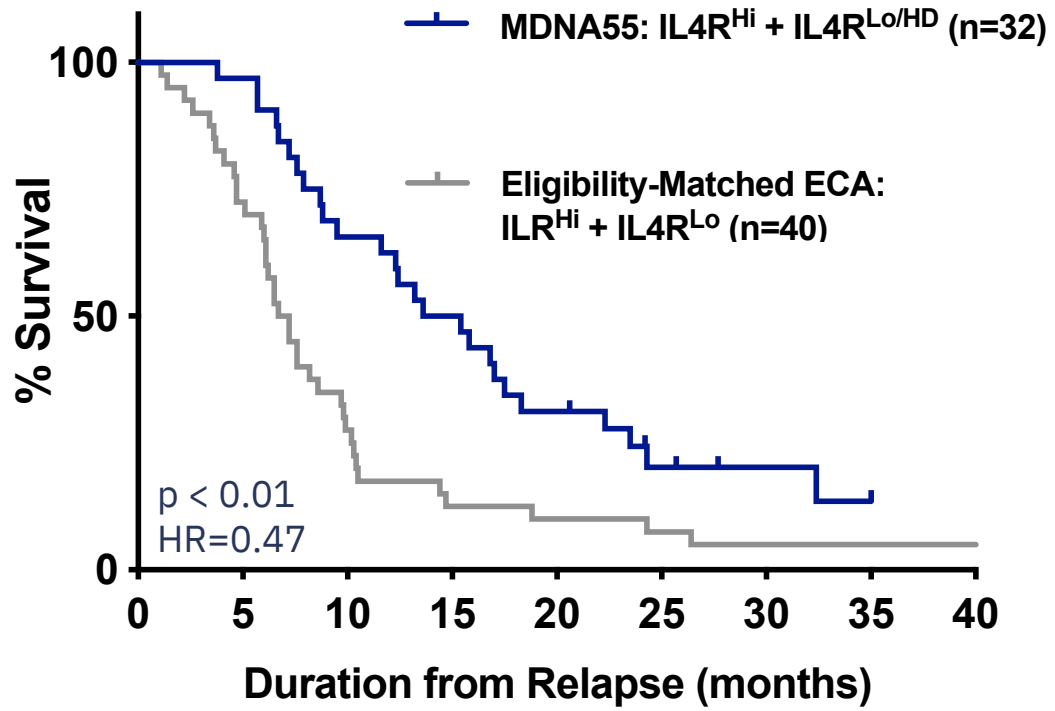
mOS	OS-12	OS-24
14.0	56%	20%
8.1	13%	0%

IL4R High (irrespective of dose) and IL4R Low patients receiving high dose were identified to benefit most from single treatment of MDNA55.

Duke University School of Medicine

MDNA55 Prolongs Survival Vs Eligibility-Matched External Control Arm (ECA)

2-Year Survival Rate > 20% in IL4RHi + IL4R$^{Lo/HD}$ Subgroup



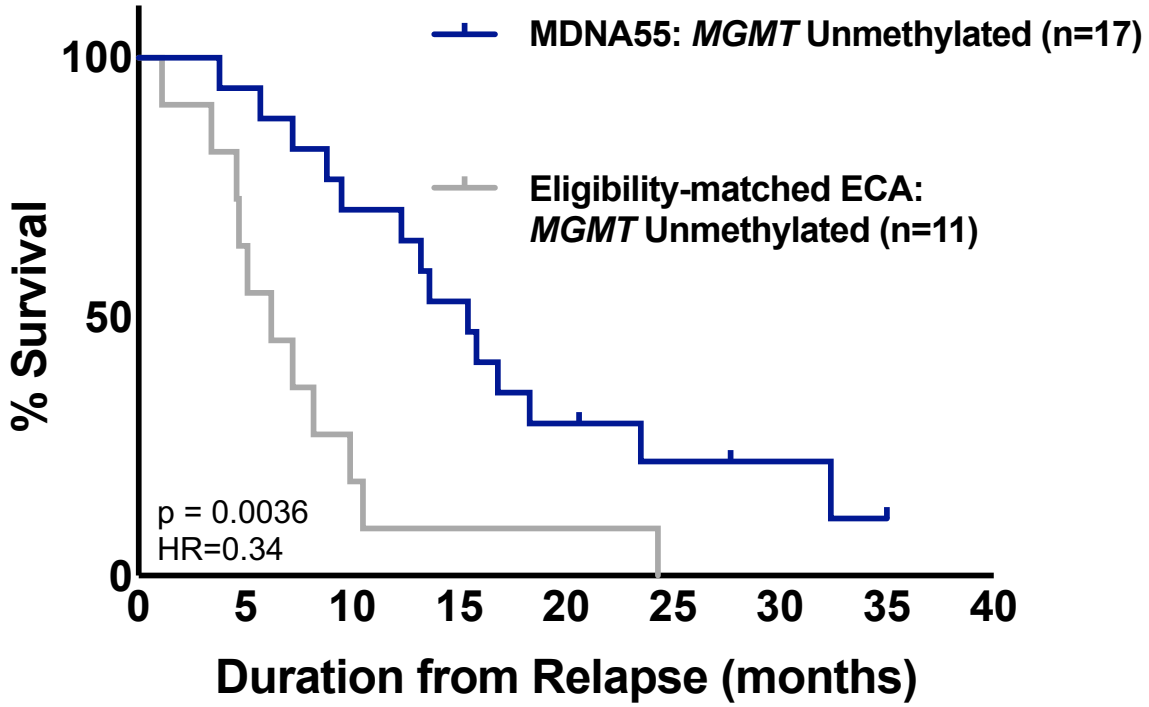
GROUP	N	mOS	OS-12	OS-24
MDNA55	32	14.5*	63%	24%
ECA*	40	7.0	18%	10%

* Survival calculated from date of relapse.
Median OS from time of MDNA55 treatment is 14.0 months;
OS-12 = 56%; OS-24 = 20%

- ECA comprised of patients meeting the same eligibility criteria of the Phase 2b study (≥ 18 yrs old, de novo GBM, 1st or 2nd relapse, not indicated for resection, KPS ≥ 70, IDH wild-type, Tumor size ≥1cm x ≤ 4cm, archived tissue from initial Dx) and received treatment at eligible relapse that included approved therapies (monotherapy or combination) for rGBM

MDNA55 is Effective in *MGMT* Promoter Unmethylated rGBM

MDNA55 is Potent in a Temozolomide-Resistant Population

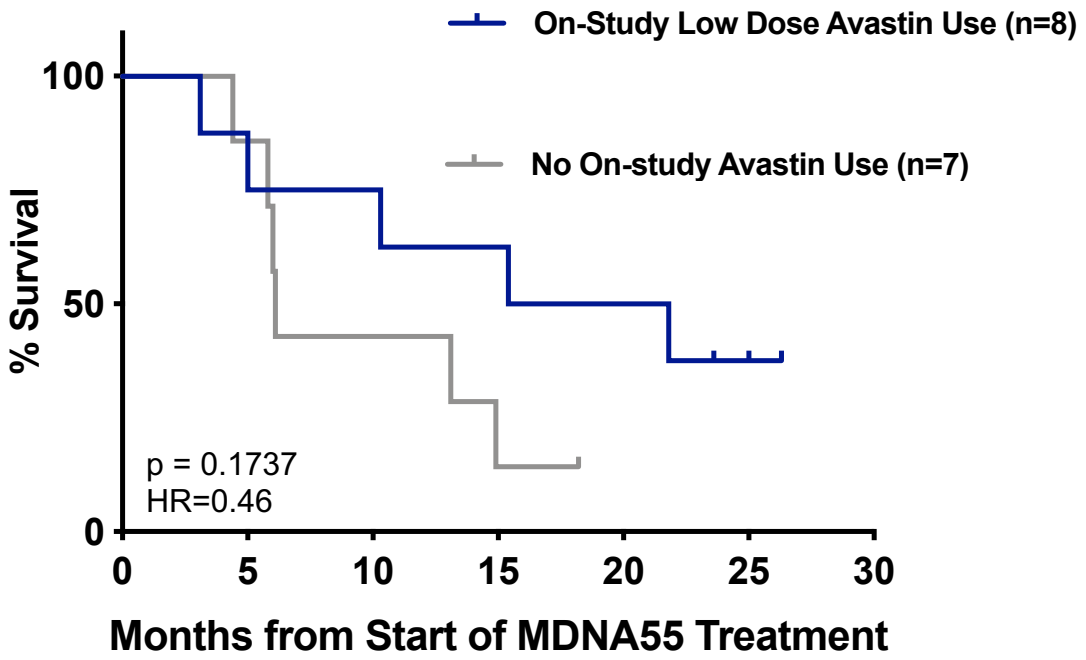


GROUP	N	mOS	OS-12	OS-24
MDNA55 – *MGMT* Unmethyl	17	15.4*	71%	22%
ECA* – *MGMT* Unmethyl	11	6.2	9%	9%

* Survival calculated from date of relapse.
Median OS from time of MDNA55 treatment is 14.9 months; OS-12 = 65%; OS-24 = 22%

- ECA comprised of patients meeting the same eligibility criteria of the Phase 2b study (≥ 18 yrs old, de novo GBM, 1st or 2nd relapse, not indicated for resection, KPS ≥ 70, IDH wild-type, Tumor size ≥1cm x ≤ 4cm, archived tissue from initial Dx) and received treatment at eligible relapse that included approved therapies (monotherapy or combination) for rGBM

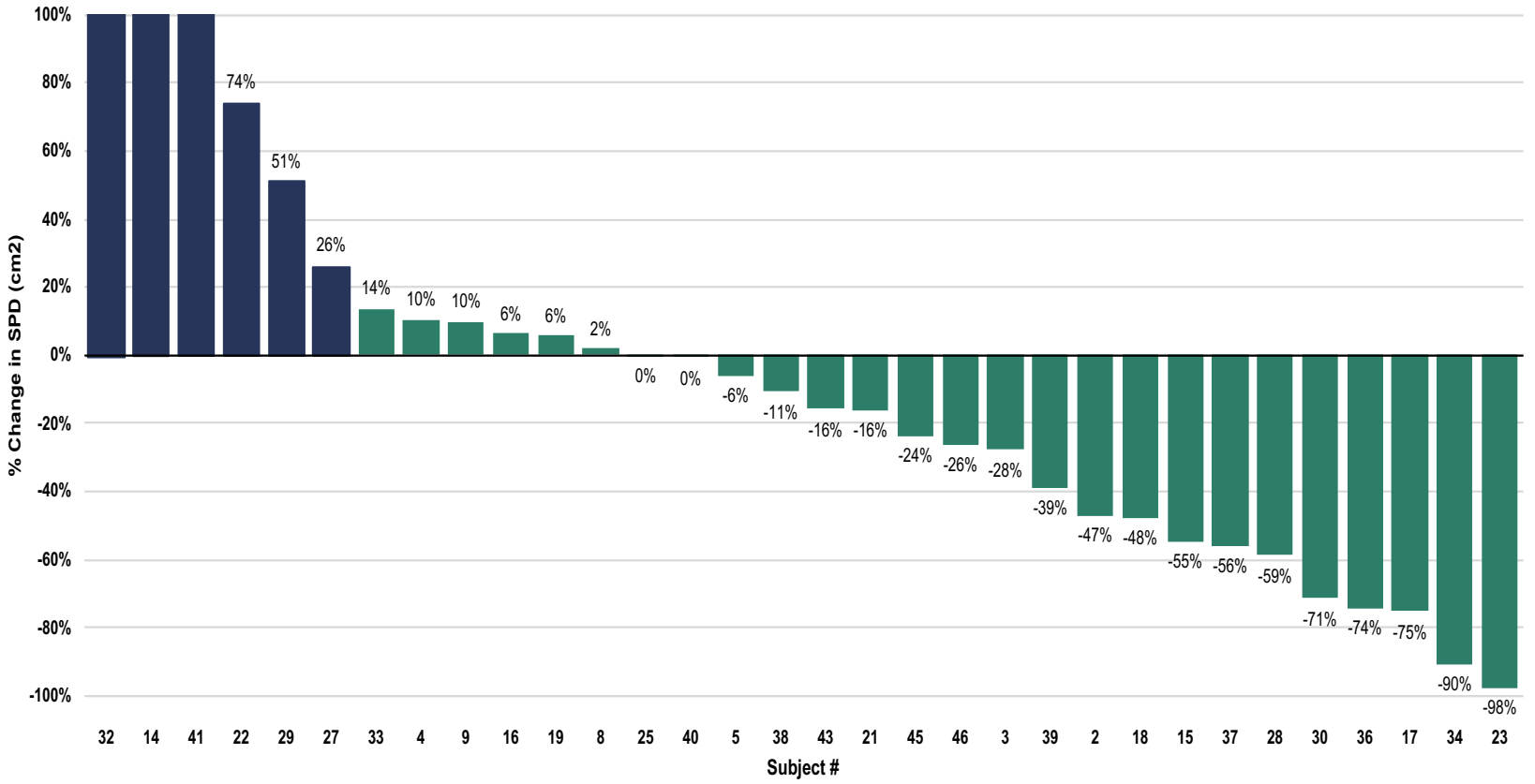
Low-Dose Transient Avastin Following MDNA55 Treatment Extends Survival in IL4R^{Hi} + IL4R$^{Lo/HD}$ Subgroup



GROUP	N	mOS	OS-12	OS-24
MDNA55 – *On-study Low Dose Avastin Use*	8	18.6	63%	38%
ECA – *No On-study Avastin Use*	7	6.1	43%	NE

- In the higher concentration cohorts (6 and 9 µg/mL; n=17), transient use of low-dose Avastin (5 mg/kg q2w or 7.5 mg/kg q3w) was allowed for management of symptom control and/or steroid sparing.
- Median number of cycles of Avastin was 3 cycles in both groups.
- In the higher concentration cohorts, 10 patients had Low IL4R, 5 patients had High IL4R, and 2 patients were unknown.

Tumor Control Following Pseudo-Progression: IL4R^{Hi} + IL4R$^{Lo/HD}$ Subgroup



Tumor control rate = 81%
(26/32 evaluable subjects)

Shown are tumor responses assessed from nadir based on radiologic imaging only

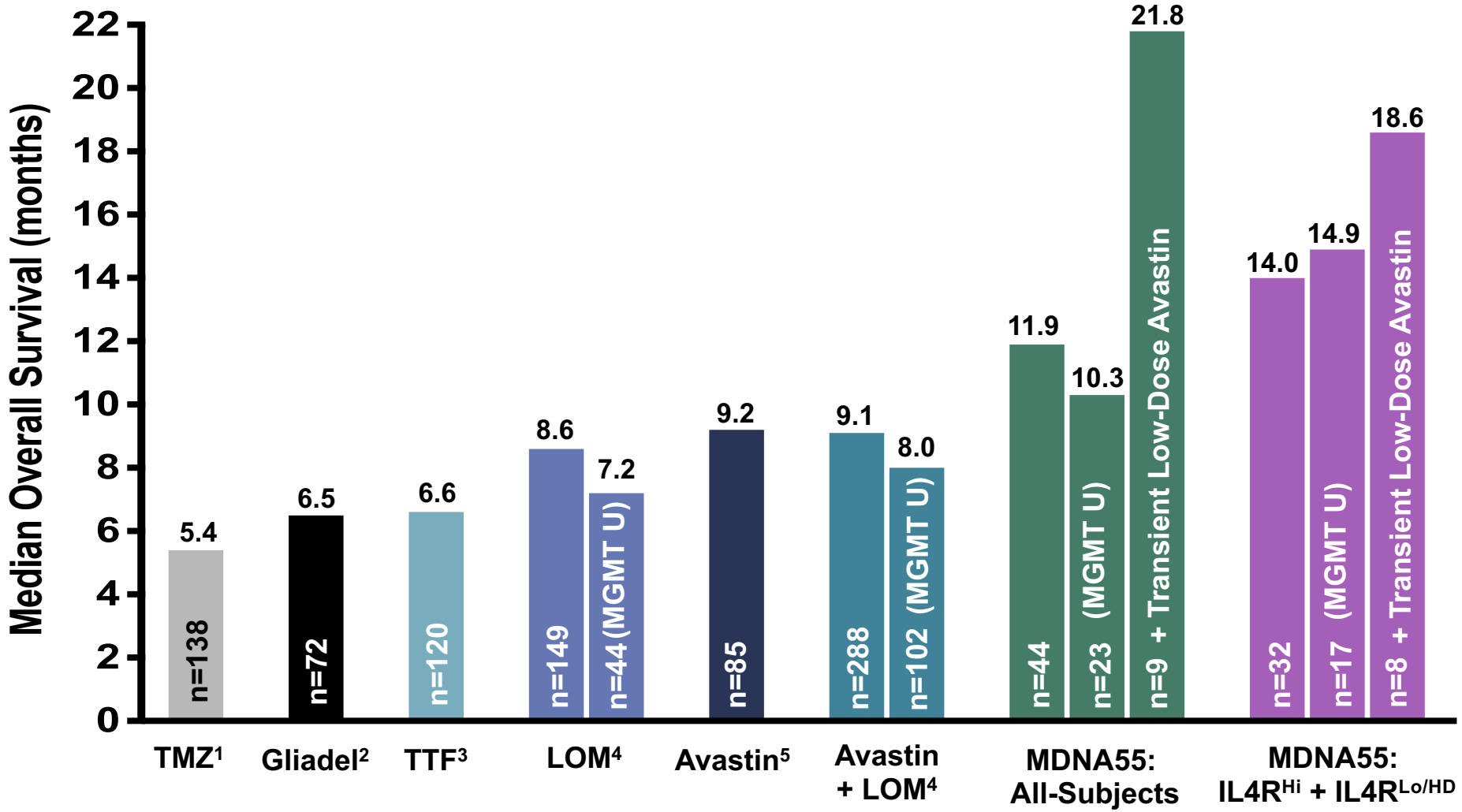
Prolonged Progression-Free Survival After MDNA55 Treatment

Increase of > 100% in PFS-12 Compared to Standard Therapies

Therapy	N	mPFS	PFS-12
MDNA55 Groups			
All Subjects	41	3.6*	27%
$IL4R^{Hi}$ + $IL4R^{Lo/HD}$	32	3.0*	24%
Approved Therapies			
Avastin[1]	85	4.2	10%**
Avastin[2]	48	4.0	10%**
Lomustine[3]	149	1.5	2%**
Avastin + Lomustine[3]	288	4.2	10%**

* Assessed by mRANO criteria using radiologic data only
** Approximations based on Kaplan-Meier curve.
1) Friedman et al., 2009; 2) Kreisl et al. 2008, 3) Wick 2017

Encouraging Survival Results Compared to Approved Therapies



TTF = Tumor Treating Fields;

LOM = Lomustine;

MGMT U = MGMT unmethylated promoter

References:

1=Brada et al., 2001; 2=Gliadel FDA Label 2018; 3=Stupp et al., 2012; 4=Wick et al., 2017; 5=Friedman et al., 2009

Benefits of a Propensity Matched External Control Arm (ECA)

Ruthie Davi, PhD
Vice President, Data Science at Acorn AI,
a Medidata company

Retrospective Matched-External Control Arm Study

For Comparison of Survival Against MDNA55-05 Study

ELIGIBILITY

- Adults ≥ 18 yrs
- De novo GBM
- 1st or 2nd relapse
- Not candidates for resection
- KPS ≥ 70
- IDH wild-type only
- Tumor size ≥1cm x ≤ 4cm
- Archive tissue from initial Dx if available

SOURCE

- Patient registries at:
  - University of California, San Francisco (UCSF)
  - St. Michael's Hospital (Toronto, Canada)
- Study conducted under IRB-approved protocols
- Investigators and Medicenna blinded to survival outcome
- IL4R analysis used same IHC assay as MDNA55-05 study

TREATMENT

Types of therapies received in the ECA (n=81):

- Avastin (26%)
- Lomustine (25%)
- Temozolmide (14%)
- Experimental Therapy (20%)
- Irinotecan (7%)
- Avastin + Lomustine (5%)
- Radiotherapy (2%)
- Avastin + Radiotherapy (1%)

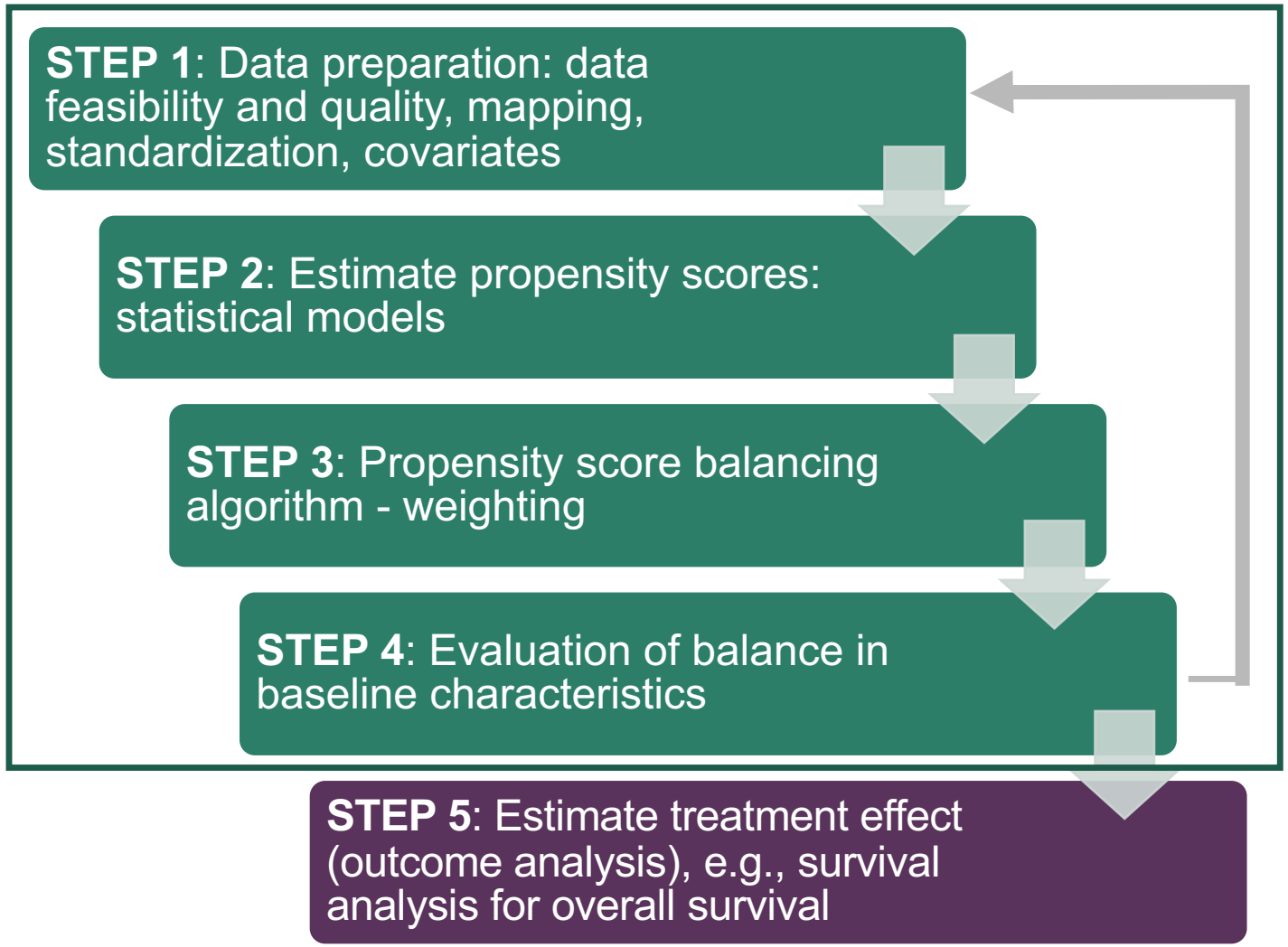
ANALYSIS

- Propensity score methodology was used to balance groups on key prognostic factors; performed prior to unblinding survival data
- Survival time was computed using a common index date (i.e., date of relapse)
- KM curves and HRs were calculated accounting for propensity score weights

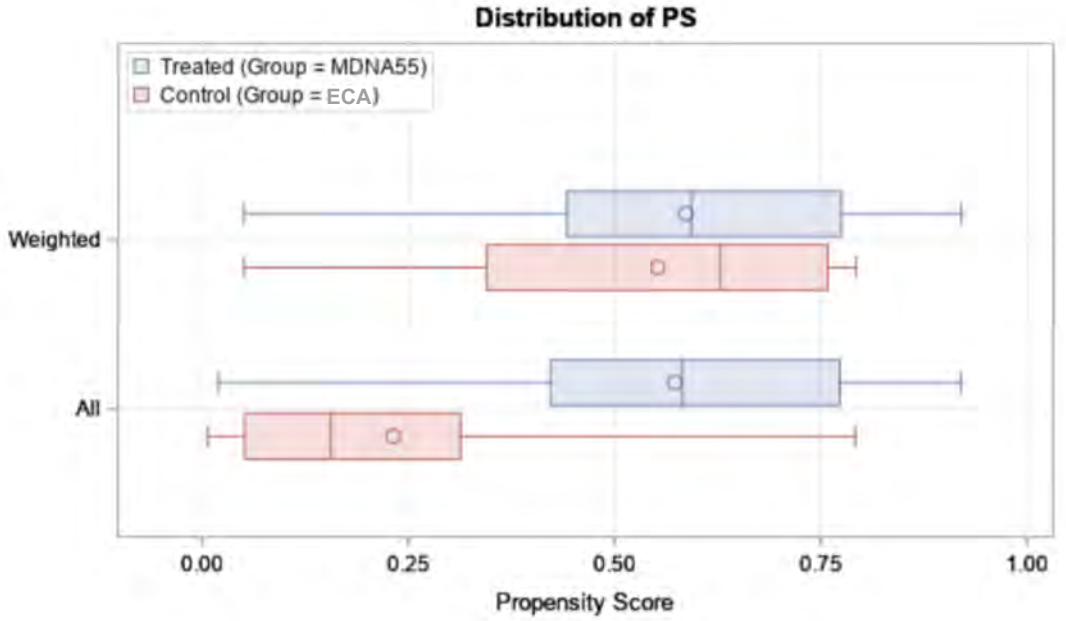
Construction of the ECA

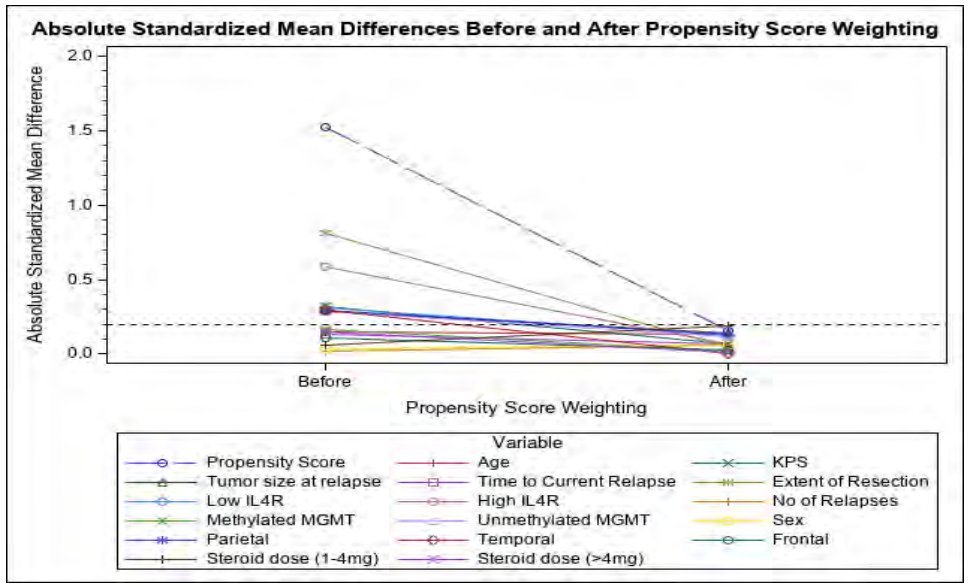
Baseline Characteristics used for Propensity Matching

- Age
- Sex
- KPS
- MGMT methylation status
- IL4R expression level
- Time from initial diagnosis to relapse
- Number of prior relapses
- Extent of resection at initial diagnosis
- Tumor size at relapse
- Tumor location at relapse
- Steroid use prior to treatment

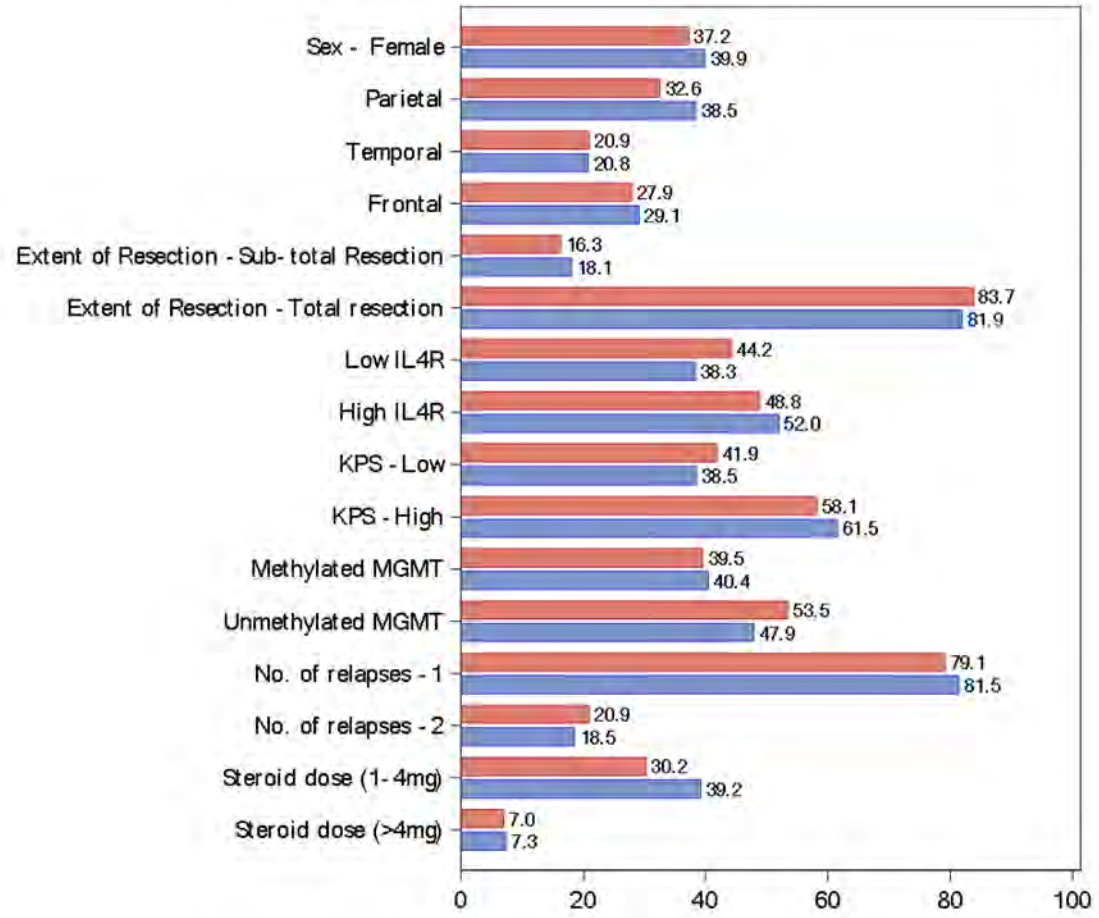


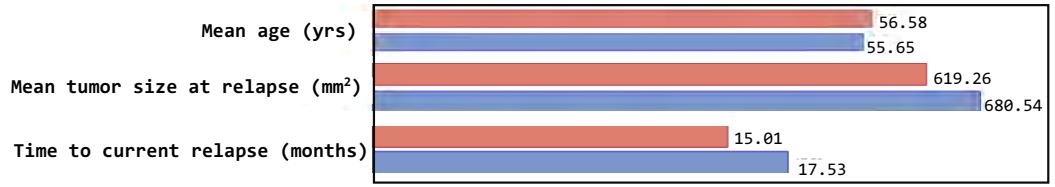
Weighted Baseline Characteristics are Well Matched





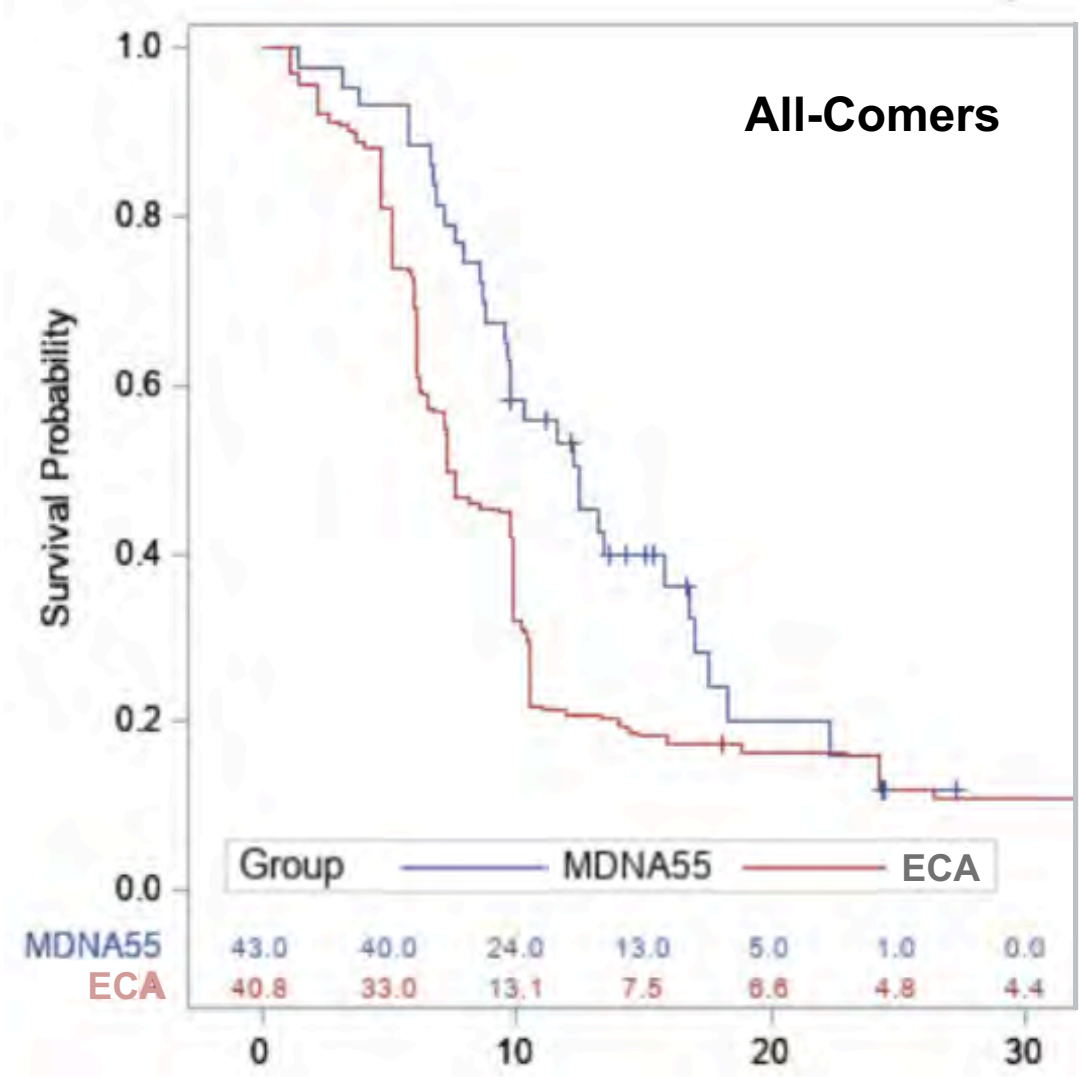
Baseline Demographic and Disease Characteristics





Weighted Survival Analysis: All-Comers

Adjusted Product-Limit Survival Estimates



Propensity score weighted estimates:

Group	Median (months)	Log-rank test p-value
MDNA55 (n=43)	12.4	0.1426
ECA (n=40.8)	7.2	

Comparison	Hazard Ratio	95% Confidence Limits	
MDNA55 vs ECA	0.634	0.392	1.026

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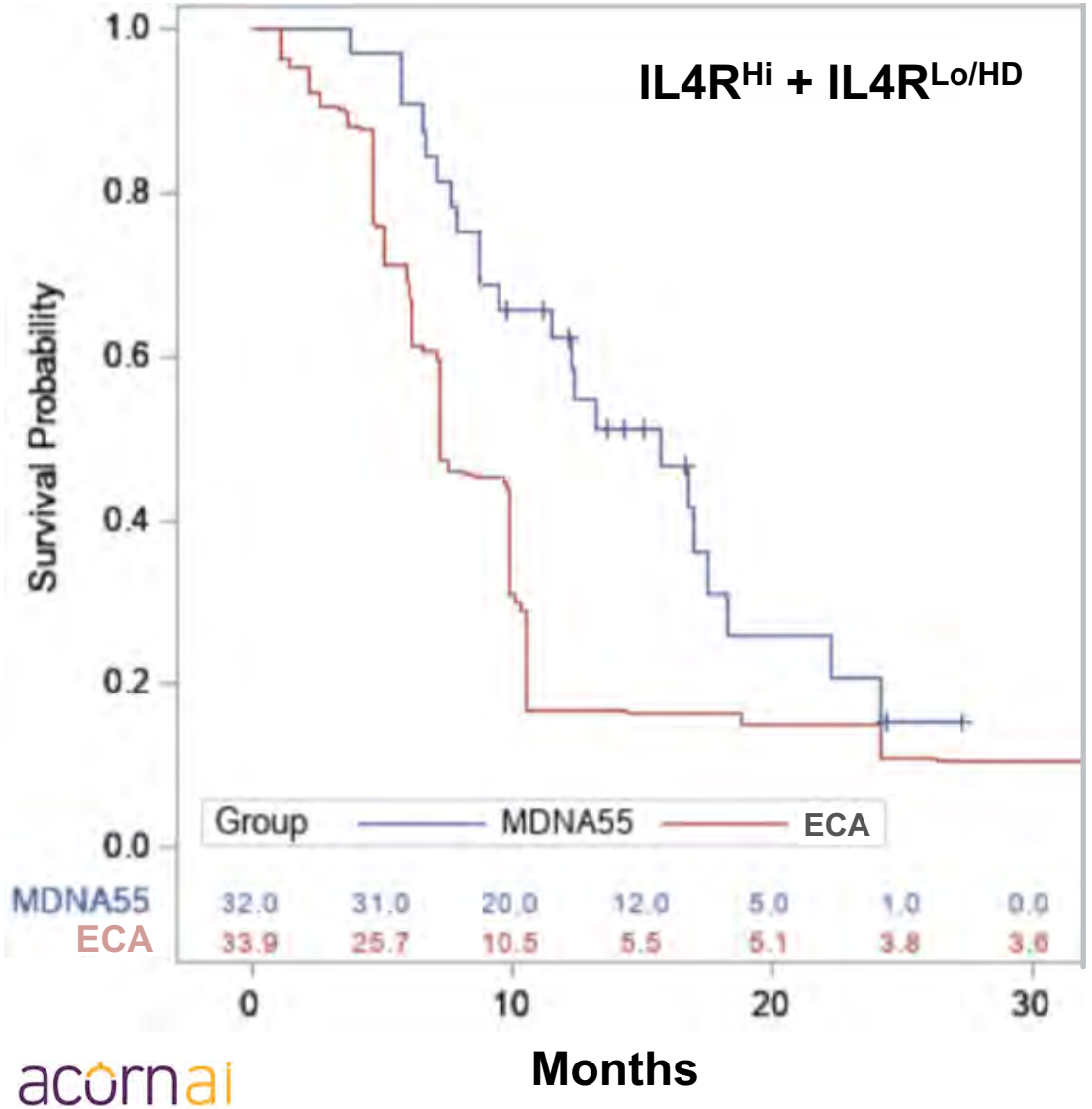
Weighted Survival Analysis: IL4R^{Hi} + IL4R$^{Lo/HD}$ Population

Adjusted Product-Limit Survival Estimates



Propensity score weighted estimates:

Group	Median (months)	Log-rank test p-value
MDNA55 (n=32)	15.7	0.1177
ECA (n=33.86)	7.2	

Comparison	Hazard Ratio	95% Confidence Limits	
MDNA55 vs ECA	0.523	0.300	0.913

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Incorporation of an ECA in a Planned rGBM Registration Trial

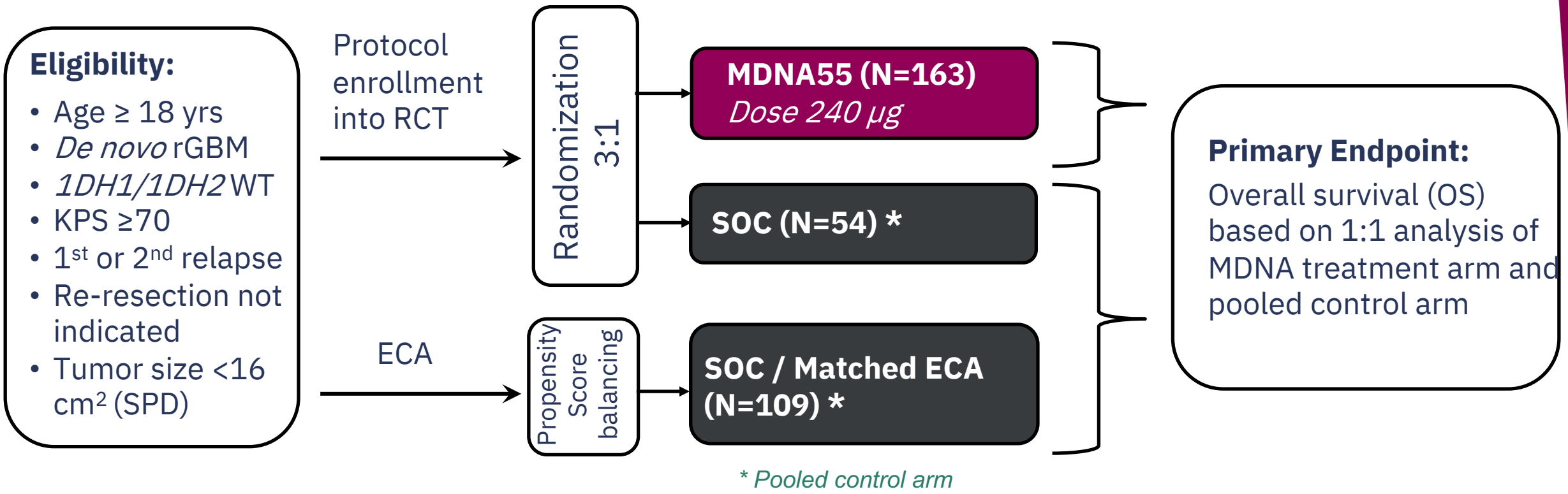
Amy McKee, M.D.
VP, Regulatory Consulting Services

parexel BIOTECH

Challenges Associated with a Traditional Randomized Controlled Trial (RCT) in rGBM

- Current NCCN guidelines specify “efficacy of SOC for rGBM is suboptimal and consideration of clinical trials is highly encouraged”
- Very high unmet need and dismal prognosis result in patients seeking experimental therapy in a trial where there is no risk of randomization to a control SOC arm
- Blinding may be unfeasible (i.e. due to method of administration) – inability to blind undermines the purpose of randomization
- Withdrawal prior to study therapy initiation of a significant percentage of participants randomized to the control arm may jeopardize the validity of the control arm experience and thereby undermine the value of a randomized trial design for the trial in question.
- Disproportionate discontinuation from SOC arm has been reported as a cause of study failure in GBM studies

Planned MDNA55 Phase 3 Trial – Hybrid Design with ECA



SOC therapies allowed:

- Bevacizumab (Avastin®)
- Lomustine (CCNU, CeeNU®, Gleostine™)
- Temozolomide (Temodar®)
- Tumor Treating Fields (Optune®)
- Radiation Therapy

Planned MDNA55 Phase 3 Trial (cont.)

ECA Arm Details

- Subjects for ECA will be identified at same sites enrolling in MDNA55 treatment arm to reduce variability.
- ECA subjects will be required to have been treated for recurrence within 5 yrs to ensure contemporaneity.
- Subject will not be eligible for ECA unless all data capture requirements are met to mitigate risk of missing data.
- All efficacy endpoints including survival for the ECA will remain blinded until all data standardization and propensity score balancing has been completed.

Study Assumptions

- 90% power
- HR of MDNA55 vs. pooled control = 0.65
- 2-sided alpha = 0.05
- Effect size = 4.6 months in mOS time
- Drop-out rate = approximately 5%

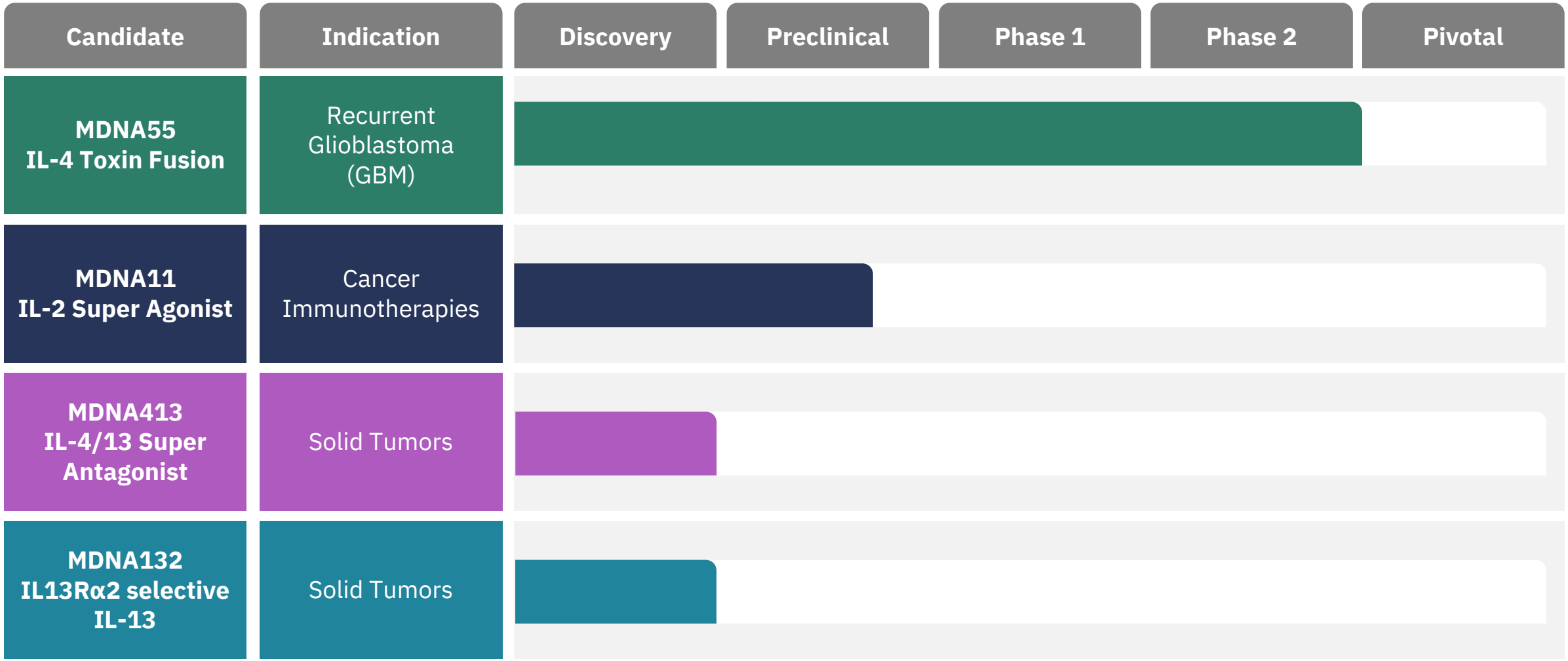
Summary

- First randomized hybrid control arm with an ECA component for a registration trial in oncology
- Trial design retains many elements preferred by FDA for a registration trial
  - Large proportion of patients randomized
  - OS endpoint
  - All data elements required for ECA
- Keys to FDA's acceptance of trial design
  - Significant unmet medical need
  - No substantive change in SOC for rGBM over the time period covered in the ECA
  - Near-contemporaneous ECA by limiting to last 5 years
  - Large effect size demonstrated in Phase 2b study
  - **Buy-in and, in fact, encouragement from FDA statistical review group**

Medicenna Overview

Fahar Merchant, Ph.D.
President & CEO, Medicenna Therapeutics

Expanding Pipeline Anchored by MDNA55 and MDNA11



MDNA55 Trial Design and Market Size Bolster Partnership Strategy

Market Size Estimated at $2 Billion Annually

Tumor Type	Annual Incidence[1]	Projected Market[2]
Recurrent Glioblastoma (rGBM)	33,300	$650M
Metastatic Brain Cancer[3]	91,500	$1.30B
Pediatric Glioma	3,800	$50M
Total	**133,500**	**$2.0B**

Brain Cancer Next Steps

Pursue Partnership Strategy for Further Development

1. GLOBOCAN 2012 http://globocan.iarc.fr/Default.aspx
2. U.S., Europe and Japan
3. Metastatic Brain Cancer numbers from colon, breast and kidney cancer only

MDNA11: IL-2 Super Agonist for Cancer Immunotherapy

Next Steps

MDNA11 Next Steps

- Pre-CTA meeting **(Complete)**
- Initiate Phase 1 clinical trial **(Mid 2021)**
- Report safety, PK/PD and biomarker results from Phase 1 monotherapy study **(End 2021)**

Advantages of Initiating Phase 1 in the UK

- Dose escalation studies can begin at a higher initial dose
- Increased prevalence of immune checkpoint inhibitor naïve patients
- Trial can expand into the United States after completion of the dose escalation portion

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.....And most of all, to the patients & their families

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CANCER PREVENTION & RESEARCH INSTITUTE OF TEXAS

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Fahar Merchant, PhD
President and CEO

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