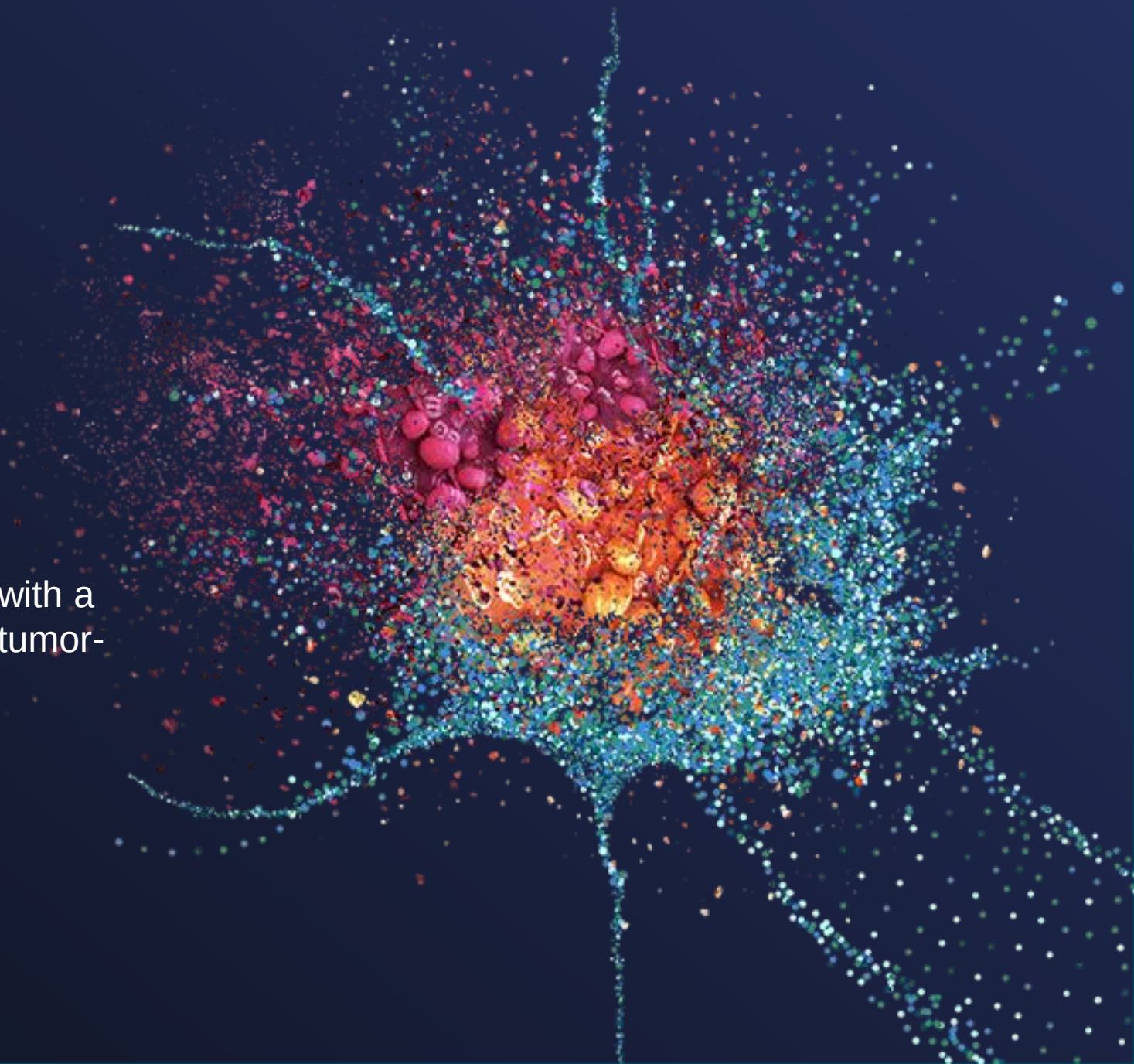
Imvax™

Imvax Corporate Overview

A clinical-stage biotechnology company with a unique platform for personalized, whole tumor-derived immunotherapies



January 2023

A compelling immuno-oncology platform opportunity

- The Imvax Goldspire™ platform harnesses decades of research and multiple validated technologies to create a pipeline of personalized, whole tumor-derived treatments targeting intractable solid tumors
- Lead asset in GBM, with Phase 2b study to commence in Q1 2023
- Additional INDs planned for other solid tumors in 2023
- Established manufacturing capabilities at Imvax with redundancy established at CDMO
- Projected up to $6B annual revenue in addressable markets, largely underserved therapeutic areas (on a non-risk adjusted basis)
    - $2B - $3B global peak sales in ndGBM alone
- Seasoned leadership with deep biopharma development and commercialization experience

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Team that's been directly involved in development, approval and commercialization of multiple biological modalities

John Furey
CEO
- COO, Spark Tx
- SVP Global Operations, Baxalta
- VP Baxter & Pfizer

David Andrews
CMO, Founder
- Chief, Tumor Division, Dept of Neurosurgery, TJU
- Trained at NY Presbyterian & Memorial Sloan Kettering

Mark Exley
CSO
- VP, AgenTus (now MiNK)
- Co-Founder, NKT Tx
- Harvard & Manchester Faculty
- Current Honorary Academics

Sean Hemingway
COO
- SVP, Global Head of BioLife Plasma Services, Takeda
- SVP, Takeda Manufacturing

John Limongelli
CLO
- SVP, GC and CS, Neos Therapeutics
- SVP, GC, Trevena

Diana Martine
Head of HR
- HR Director, Aramark
- Head of HR, Biocoat

Josh Muntner
CFO
- CFO, Mesoblast
- SVP, Bus Dev, ContraFect
- +15 yrs investment banking

Tyler Curiel
Scientific Advisor
- Prof of Med, Microbiology and Immunology at the Dartmouth Geisel School of Medicine

Shefali Agarwal
Scientific Advisor
- Pres & CEO, Onxeo
- Prev. EVP, CMDO Epizyme

Management
Non-Management

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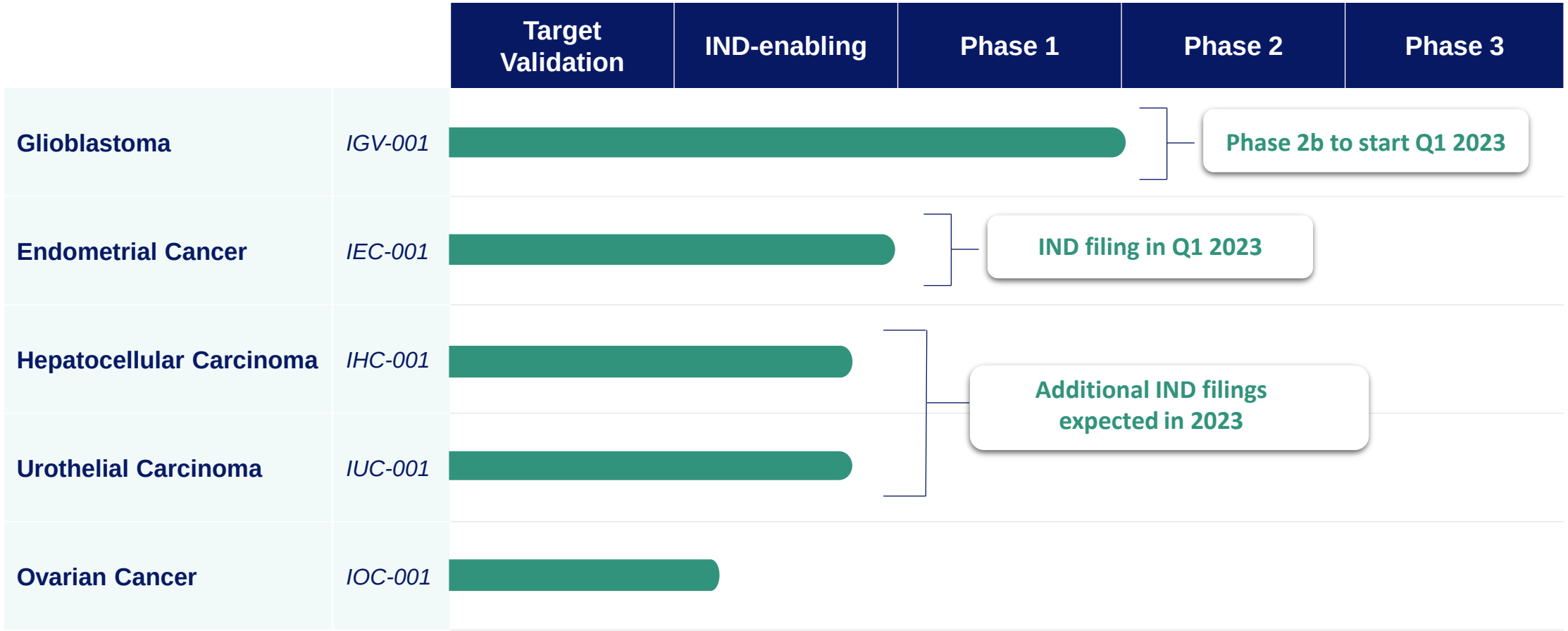
Imvax pipeline: Focused on solid tumor types



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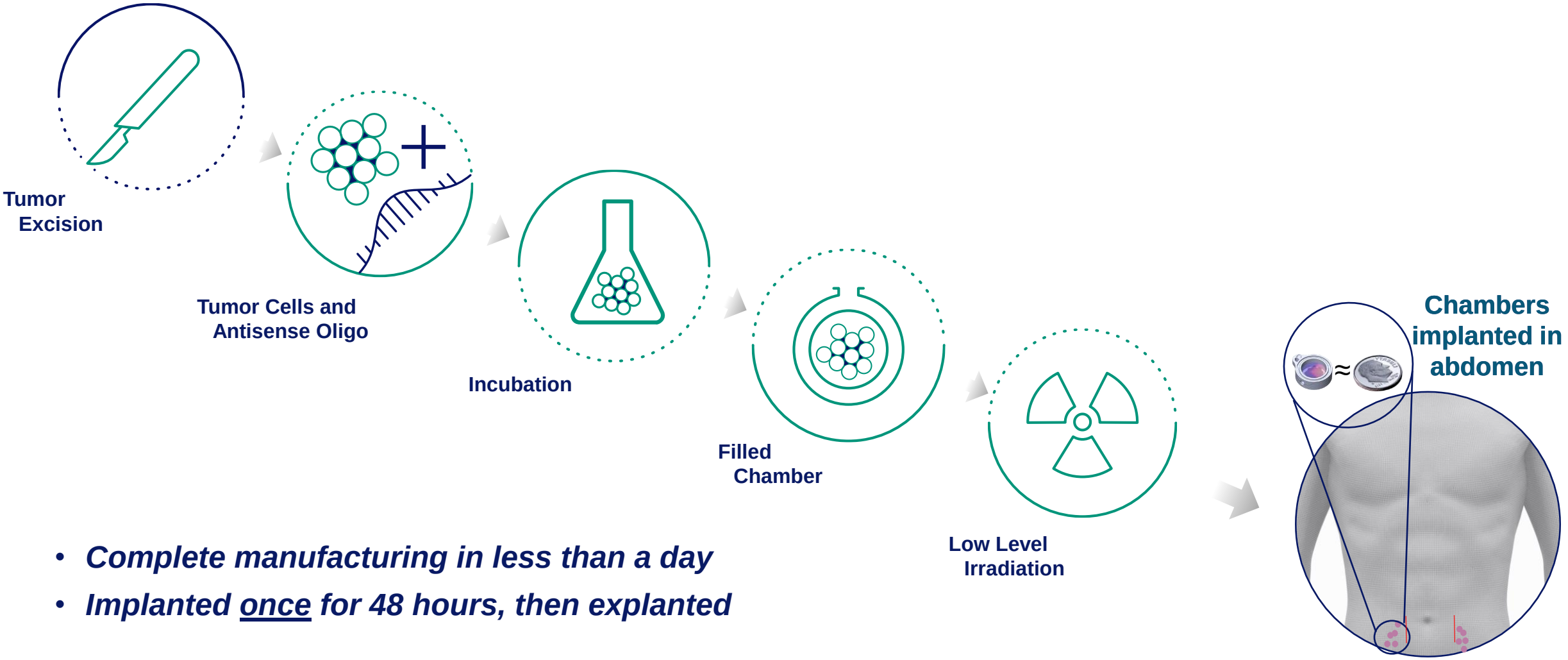
Imvax’s Goldspire Platform

A Powerful Approach to the Complexity of Solid Tumors

Imvax's Goldspire process



- ***Complete manufacturing in less than a day***
- ***Implanted <u>once</u> for 48 hours, then explanted***

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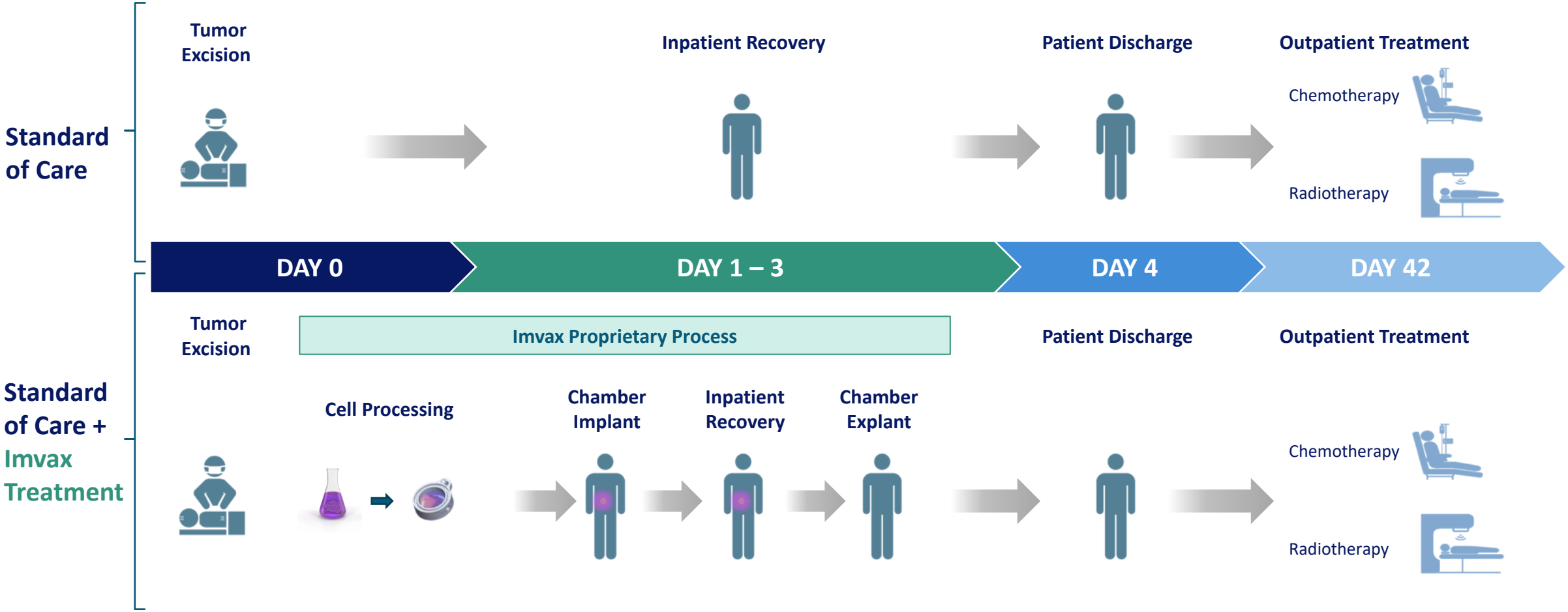
Goldspire fits seamlessly into GBM standard of care

Entering Phase 2 for Newly-Diagnosed GBM



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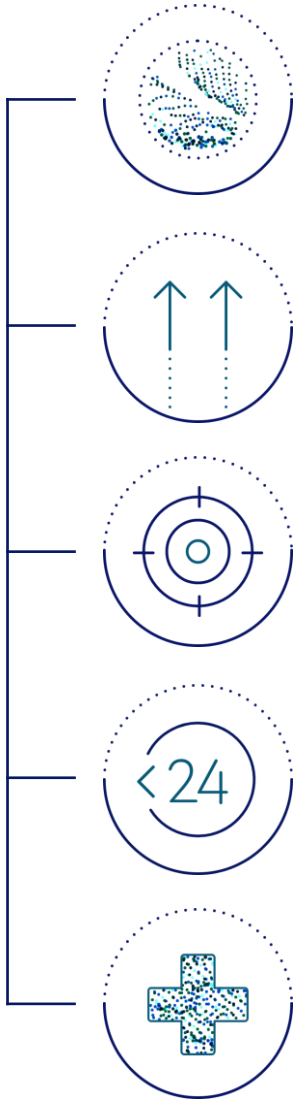
Goldspire platform has multiple advantages



Full antigenic signature capture

Broad spectrum immune activation

No ‘off-target’ effects

Overnight tissue processing

Integrated into the ‘standard of care’

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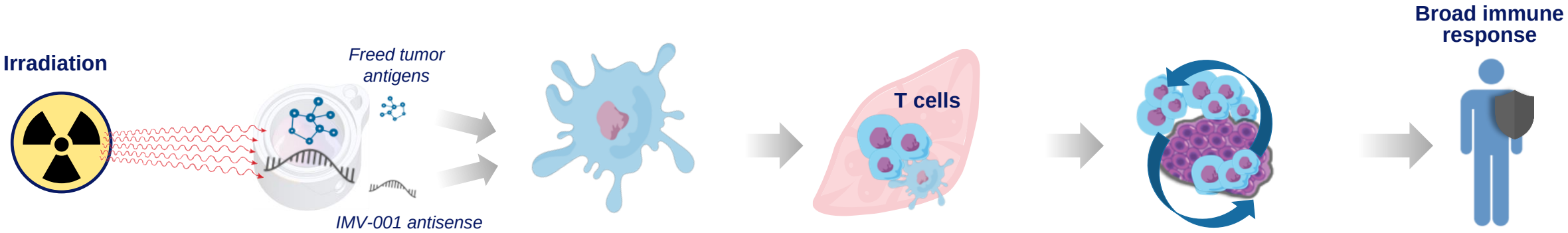
Immune response effectively attacks tumors on multiple fronts



1 Immunogenic cell death

IGF1R is overexpressed in malignant cells, where it promotes cell survival and tumor progression. The use of IMV-001 (antisense oligonucleotide against IGF1R), radiation and implantation within small specialized chambers induces immunogenic cell death during the short implantation in the body.

2 APCs activated

The full tumor antigen signature diffuses out of the chamber's micropores and is picked up by local 'antigen presenting cells' (APC).

Free IMV-001 also diffuses out of the chambers and serves as a second layer of immune stimulation for local APCs.

3 T cells primed

Activated APCs migrate to local draining lymph nodes, where they prime T cells against the tumor antigens. Implanting the chambers remote from immuno-suppressed tumor-draining lymph nodes elicits an optimal immune response

4 T cells impair proliferation and kill tumors

Resulting antigen-specific T cells migrate to tumor site and provide anti-tumor cytotoxicity. These T cells also produce IFNγ, which inhibits tumor cell proliferation and activates other immune cells.

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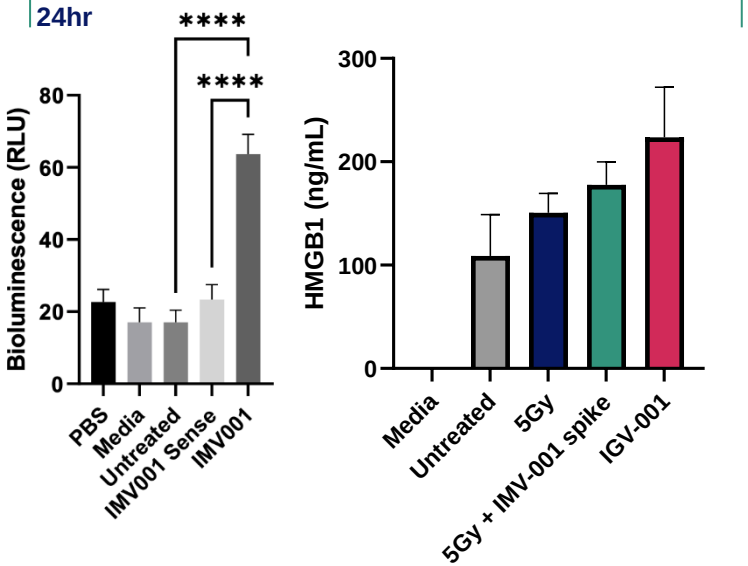
Preclinical data support multi-component mechanism of action

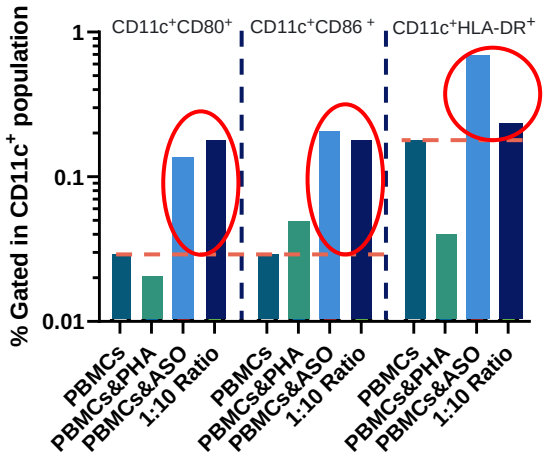
1 Immunogenic cell death

Tumor immunogenic cell death facilitates the induction of anti-tumor cellular immunity, in part due to the release of ATP (see below). Immunogenic cell death promotes the recruitment of APC and subsequent tumor antigen uptake.



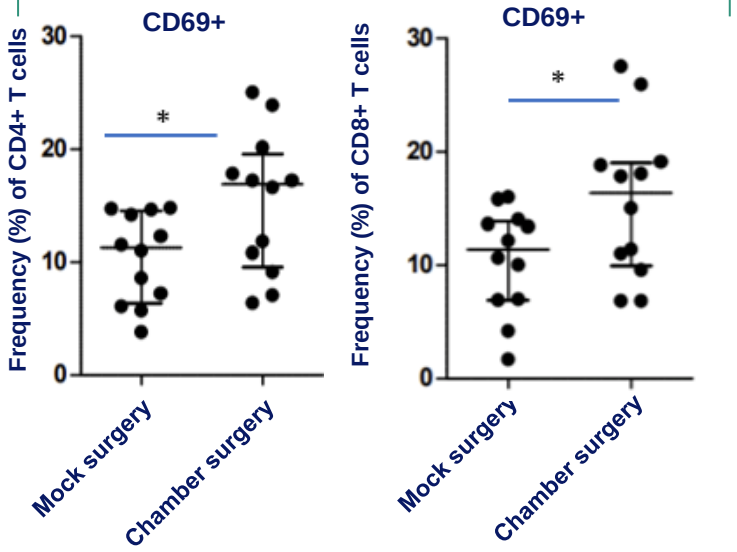
2 APCs activated

Increase in CD11c+ APC Dendritic-type cell activation and maturation induced by antisense.



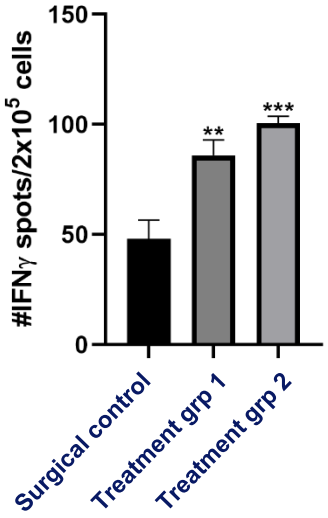
3 T cells primed

Increase in activated CD4 and CD8 T cells seen in treated mice.



4 T cells impair proliferation and kill tumors

Tumor antigen-specific IFNγ levels in PBMCs show significant rise in treated animals vs. controls. Treated mice show tumor shrinkage and extended survival.



* p < 0.05 ** p < 0.01 *** p < 0.001 **** p < 0.0001

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Lead Program: GBM

GBM Overview

- Glioblastoma multiforme (GBM) is the most common malignant tumor of the brain and CNS
- Newly diagnosed GBM patients have median overall survival (OS) or life expectancy of approximately 16 months, and less than 7% of GBM patients survive to five years after diagnosis
- Standard of care treatment is surgery followed by radiotherapy and chemotherapy
- More than 20% of all brain tumor illness is attributable to GBM
- Global market expected to grow with a CAGR of 8.14% to $1.5B by 2027[1]

1. https://www.marketdataforecast.com/market-reports/glioblastoma-market
2. https://pubmed.ncbi.nlm.nih.gov/30790064/

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GBM Clinical Program Overview

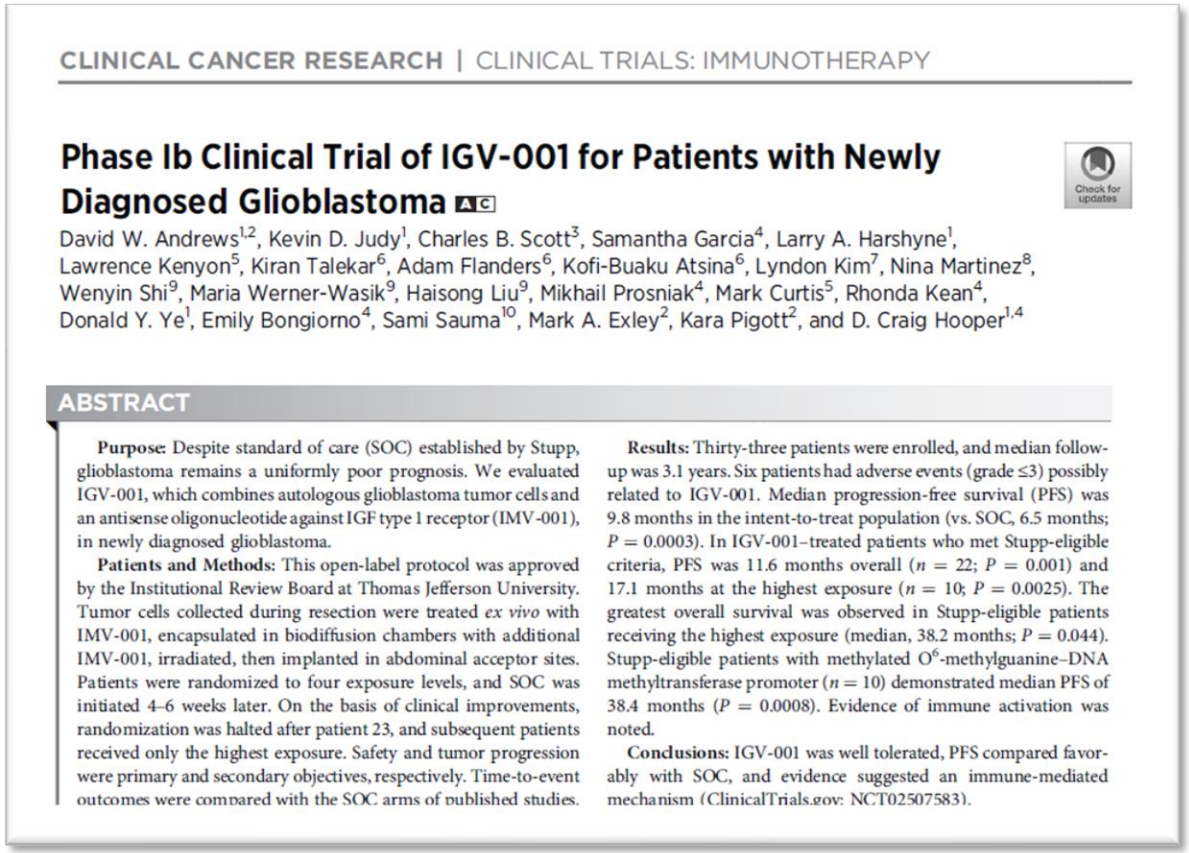
- Imvax's GBM program seeks to build on groundbreaking Phase 1b data
- Strong patent position exclusively licensed from Thomas Jefferson University
- In-house GMP manufacturing in Philadelphia and through 3rd-party CDMO
- Orphan Drug Designation granted by FDA; less than 14,000 newly diagnosed patients in the US annually
    - Underserved market with no recent innovation

Phase 1b results published in peer-reviewed *Clinical Cancer Research* in April 2021

- Ph1b trial of IGV-001 in patients with newly diagnosed glioblastoma
- Primary and secondary objectives were safety and tumor progression
- Broad inclusion criteria resulted in enrollment of difficult to treat patients
- 33 patients randomized to receive varying number of chambers and/or differing lengths of exposure

Number of chambers and length of implantation		
	24 hours	**48 hours**
10 chambers	N = 6	N = 5
20 chambers	N = 5	N = 17*

* 15 patients if two patients with bihemispheric/multicentric disease excluded



CLINICAL CANCER RESEARCH | CLINICAL TRIALS: IMMUNOTHERAPY

Phase Ib Clinical Trial of IGV-001 for Patients with Newly Diagnosed Glioblastoma

David W. Andrews[1,2], Kevin D. Judy[1], Charles B. Scott[3], Samantha Garcia[4], Larry A. Harshyne[1], Lawrence Kenyon[5], Kiran Talekar[6], Adam Flanders[6], Kofi-Buaku Atsina[6], Lyndon Kim[7], Nina Martinez[8], Wenyin Shi[9], Maria Werner-Wasik[9], Haisong Liu[9], Mikhail Prosniak[4], Mark Curtis[5], Rhonda Kean[4], Donald Y. Ye[1], Emily Bongiorno[4], Sami Sauma[10], Mark A. Exley[2], Kara Pigott[2], and D. Craig Hooper[1,4]

ABSTRACT

Purpose: Despite standard of care (SOC) established by Stupp, glioblastoma remains a uniformly poor prognosis. We evaluated IGV-001, which combines autologous glioblastoma tumor cells and an antisense oligonucleotide against IGF type 1 receptor (IMV-001), in newly diagnosed glioblastoma.

Patients and Methods: This open-label protocol was approved by the Institutional Review Board at Thomas Jefferson University. Tumor cells collected during resection were treated *ex vivo* with IMV-001, encapsulated in biodiffusion chambers with additional IMV-001, irradiated, then implanted in abdominal acceptor sites. Patients were randomized to four exposure levels, and SOC was initiated 4–6 weeks later. On the basis of clinical improvements, randomization was halted after patient 23, and subsequent patients received only the highest exposure. Safety and tumor progression were primary and secondary objectives, respectively. Time-to-event outcomes were compared with the SOC arms of published studies.

Results: Thirty-three patients were enrolled, and median follow-up was 3.1 years. Six patients had adverse events (grade ≤3) possibly related to IGV-001. Median progression-free survival (PFS) was 9.8 months in the intent-to-treat population (vs. SOC, 6.5 months; $P = 0.0003$). In IGV-001–treated patients who met Stupp-eligible criteria, PFS was 11.6 months overall ($n = 22$; $P = 0.001$) and 17.1 months at the highest exposure ($n = 10$; $P = 0.0025$). The greatest overall survival was observed in Stupp-eligible patients receiving the highest exposure (median, 38.2 months; $P = 0.044$). Stupp-eligible patients with methylated O^6-methylguanine–DNA methyltransferase promoter ($n = 10$) demonstrated median PFS of 38.4 months ($P = 0.0008$). Evidence of immune activation was noted.

Conclusions: IGV-001 was well tolerated, PFS compared favorably with SOC, and evidence suggested an immune-mediated mechanism (ClinicalTrials.gov: NCT02507583).

https://clincancerres.aacrjournals.org/content/27/7/1912

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Ph1b study met safety endpoints

Efficacy data compelling across broad spectrum of patients

Patients with Newly Diagnosed Glioblastoma				
	IGV-001 102 Phase 1b Study			Standard of Care[1]
Groups	Total ITT	Highest Dose Cohort ITT	Stupp-Eligible[2] Highest Dose Cohort	
Patients (n)	(n=33)	(n=15)	(n=10)	(n=1,059)
mOS	17.3 months	25.3 months	**38.2** months**	**16.2 months**
OS24†	39%	50%	60%	30%
PFS6	86%	85%	90%	56%
mPFS	9.8* months	17.3** months	**17.1** months**	**6.5 months**

- No concerns regarding **safety** profile of product; AEs largely procedural related and addressed during the trial
- ***Statistically significant*** **PFS and survival benefits** compared to SOC seen particularly at higher exposures ITT and when the Stupp Inclusion/Exclusion criteria are applied and even more significant in the MGMT+ subgroup of patients

Note: figures based on May 1, 2020 data cut-off.
1 SOC data only available for age cut < vs.> 60 in Stupp; Stupp et. al. Lancet Oncology 10: 459-466 (2009)
2 Stupp-Eligible excludes >70 yrs old and extensive intracranial disease in both hemispheres or multi-centric disease

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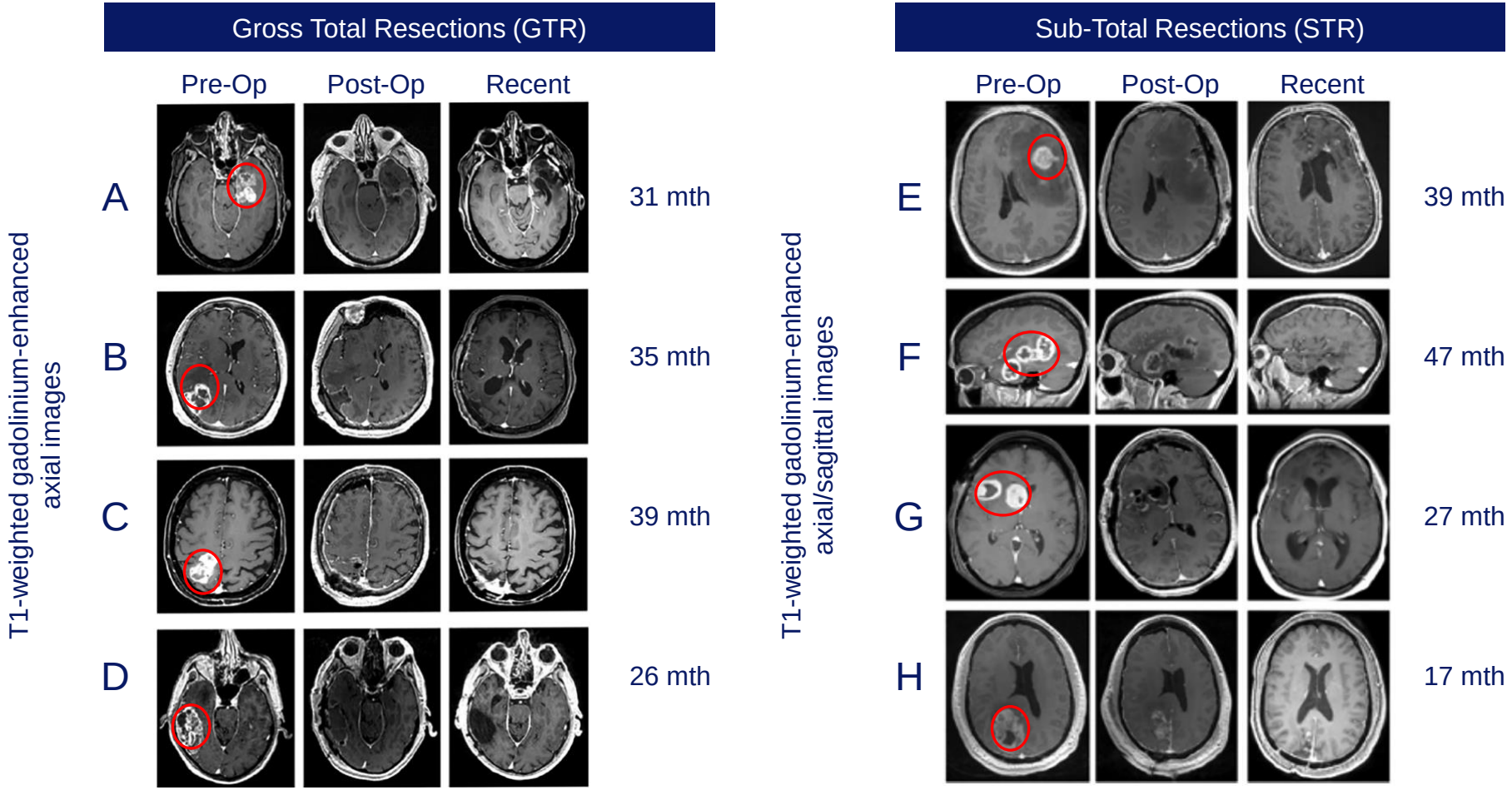
Radiographic responses in Phase 1b study show meaningful delay to disease progression



Median time to progression:
GTR is 8.9mth[1]; these patients = not reached

Median time to progression:
STR is 5.9mth[2]; these patients = 32.7mth

[1] Chaichana KL et. al. (2014) World Neurosurgery 82: 257-265
[2] Fukui A et. al. (2017) World Neurosurgery 98: 73-80

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Phase 2b clinical design

- Randomized, multicenter, double-blind, placebo-controlled, Phase 2b study to assess IGV-001 in newly diagnosed patients with glioblastoma post-craniotomy
- Compares efficacy of a one-time treatment with IGV-001 plus standard-of-care (SOC) GBM therapy (RT + TMZ) with SOC GBM therapy alone
- Study population limited to Stupp-eligible patients
  - Excludes patients > 70 yrs old, with extensive intracranial disease in both hemispheres or multi-centric disease
- 93 patients randomized 2:1 (IGV-001 + SOC vs SOC)
- Conducted at up to 25 US sites
- Primary efficacy endpoint: progression free survival (PFS)
- Overall survival (OS) is the key secondary efficacy endpoint
- Potential PFS data in 2Q 2025 and then OS follow-up in 2Q/3Q 2026

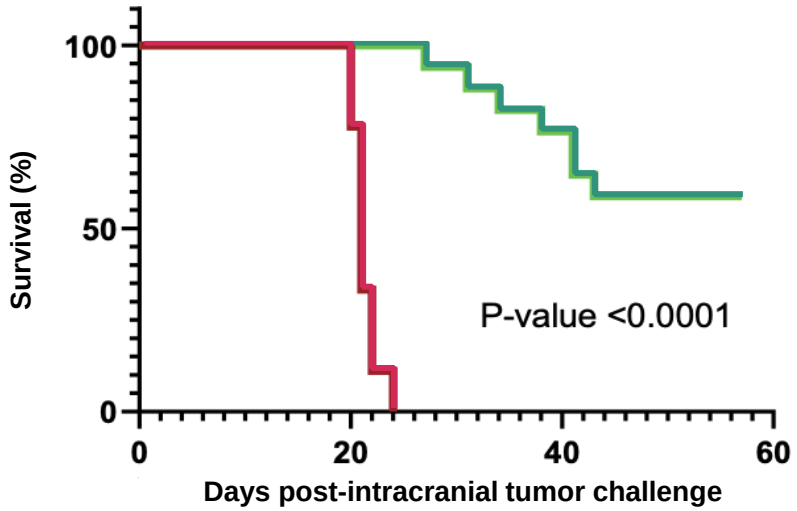
Pipeline in Solid Tumors

Preclinical data supports potential in broad range of solid tumors

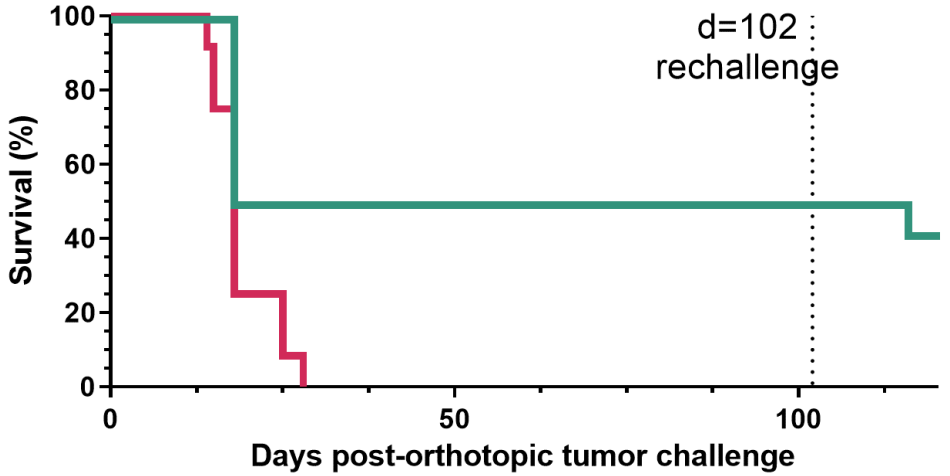
Tumor	Model Status
GBM	Compelling mono-activity, *additional* combo. activity
Ovarian	Compelling durable mono-activity
HCC	Compelling durable mono-activity, rechallenge data
Bladder	Compelling durable mono-activity
PDAC	Modest statistical benefit to date
CRC	Modest statistical benefit to date

In vivo data demonstrating extended survival in multiple animal models

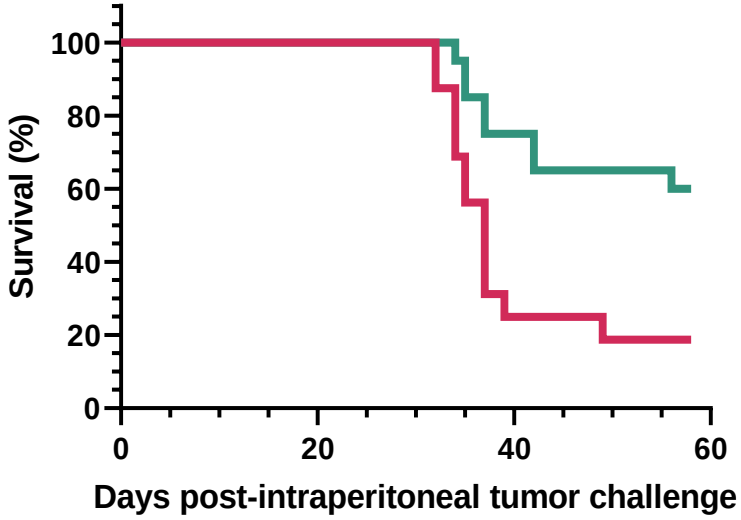
Glioblastoma (GBM)



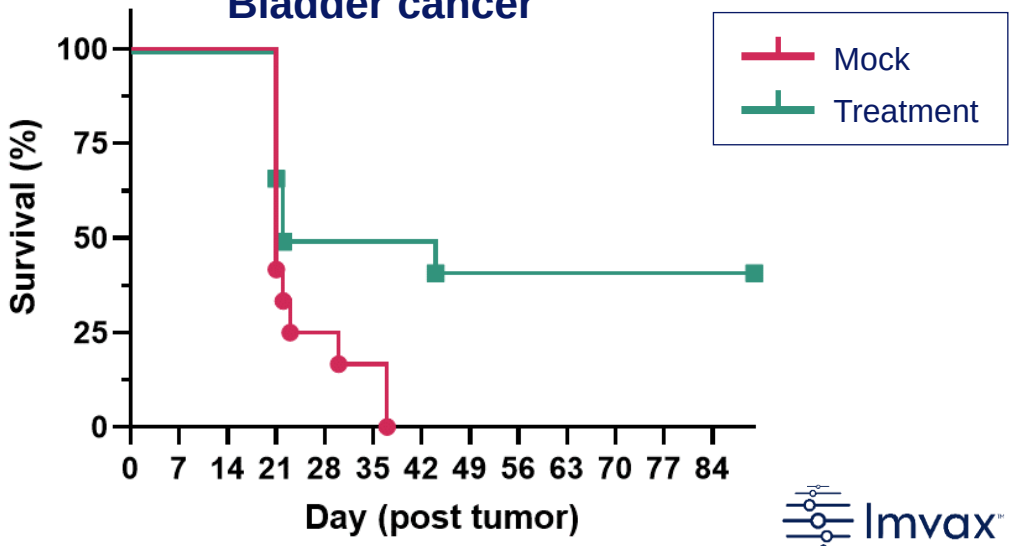
Hepatocellular carcinoma



Ovarian cancer



Bladder cancer



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Preclinical data provides MOA support and shows promise in multiple solid tumors

- Powerful anti-tumor effects in multiple early/primary tumor models (GBM, HCC, UCA and ovarian) and modest benefit in PDAC
- Mouse models use same chamber and antisense as human studies
- Well-tolerated, as in the clinic
- Immune response correlates across indications, including tumor antigen-specific IFNγ
- Increased activation markers in PBMCs exposed to IGV-001:
  - Increased Dendritic (antigen presenting) Cell activation and T cell memory markers

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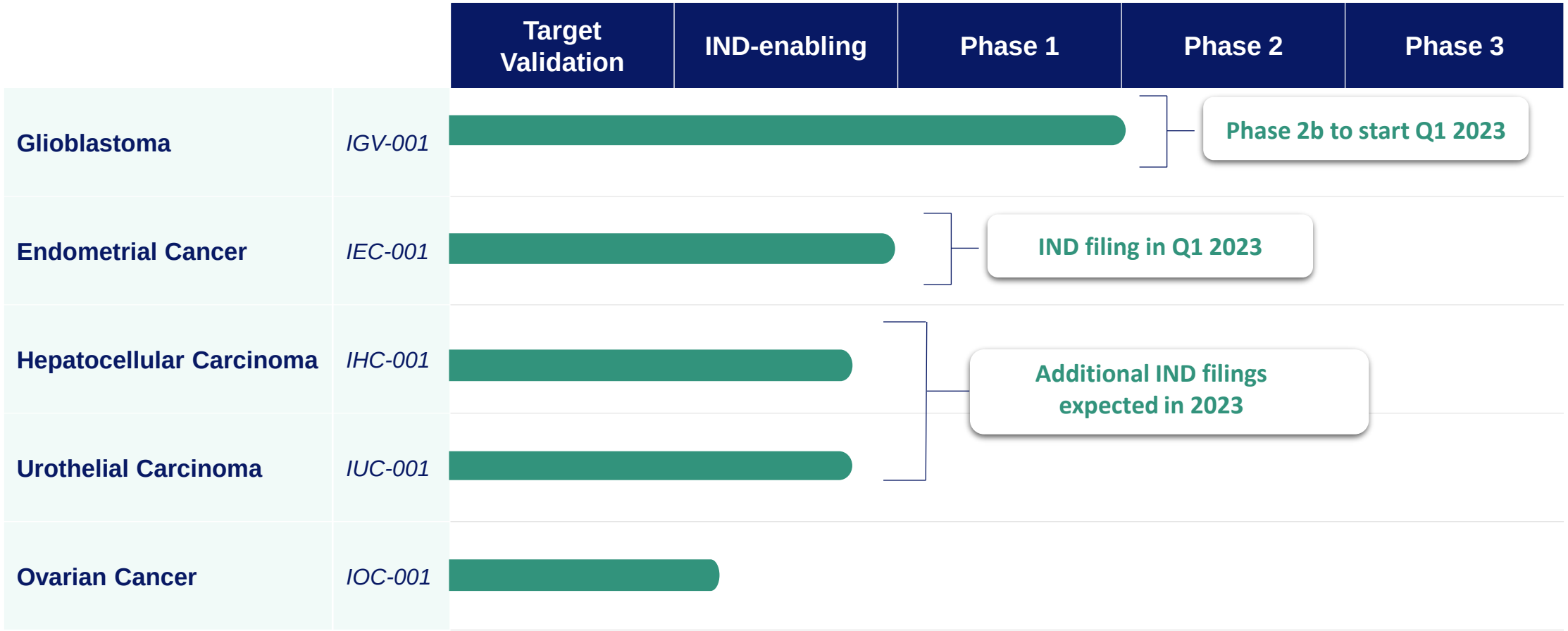
Imvax pipeline: Focused on solid tumor types



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