REVOLUTIONIZE YOUR DRUG DISCOVERY

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About Us

- **Heligenics' proven breakthroughs in biotechnology power our GigaAssay™ to deliver high-quality biologic leads – at accelerated 4x speed!**

- **Our GigaAssay generates a MEGA-MAP™ activity and variant landscape**

- **Outpace the competition – Heligenics supercharges biologics discovery that is scalable at a significantly lower cost**

Tackling Leukemia: A Massive Opportunity

- **21,000 new people are diagnosed annually in the US alone with Acute Myeloid Leukemia (AML), with a mortality of >11,000 per year**

- **Current clinical approaches and their limitations**
  - Today's treatment landscape includes chemotherapy, targeted therapies, and CAR-T – but unmet needs remain
  - Current IFN-α, a treatment molecule, cures AML in mice but is cytotoxic, unstable, and immunogenic – limiting its effectiveness over time

- **Challenges in new IFN-α drug development**
  - High costs and long timelines limit pre-clinical development
  - Slow, tedious methods are choking innovation and stalling lead discovery

IFN-α Solution
Biobetter for Leukemia

GigaAssay: proprietary technology that drives market-shifting cost savings.

- 4X faster
- 90%+ success in validation of lead[1] generation

Market Opportunity	Heligenics Solutions
Increase potency	Rapidly pinpoint potent IFN-α variants from 100k+ leads to reduce dosing and side effects
Improved stability	Discover stable leads that reduce dosing – no guesswork
Reduced immunogenicity	Engineered for immune stealth – maximize efficacy over time
Oral forms	Unlock oral delivery with innovative molecular tweaks and natural modifications
Customized biologics	MEGA-Map landscapes reveal full variant activity 4x faster than conventional single-track technology

[1]prioritized drug candidates

Our GigaAssay Technology for Discovery of Next-Generation IFN-α Drug Leads

Our patented GigaAssay leverages Heligenics' technology to rapidly screen >100k IFNI-α variants for precise, highly impactful results

- **Breakthrough efficiency: lower costs, faster launch**
- **Discovery and lead verification 4x faster than conventional methods**
  - Less than one year with GigaAssay vs US average of 4.5 years
  - Rapid asset validation: *A single GigaAssay will deliver up to 50 actionable leads in <1 year*
- **Tailored assays drive 90%+ projected clinical trial success**
- **GigaAssay accelerates lead validation with vast libraries and MEGA-Map landscapes**

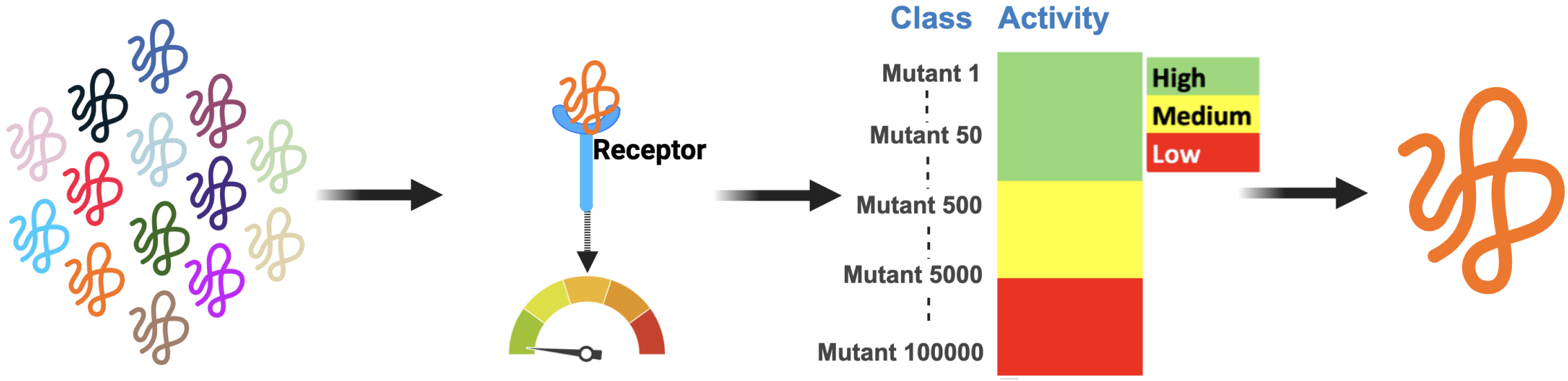
How? – With the GigaAssay
100k's of leads tested in human cells - *simultaneously*

>100k different IFN-α leads simultaneously

Cell-based GigaAssay measures bioactivity

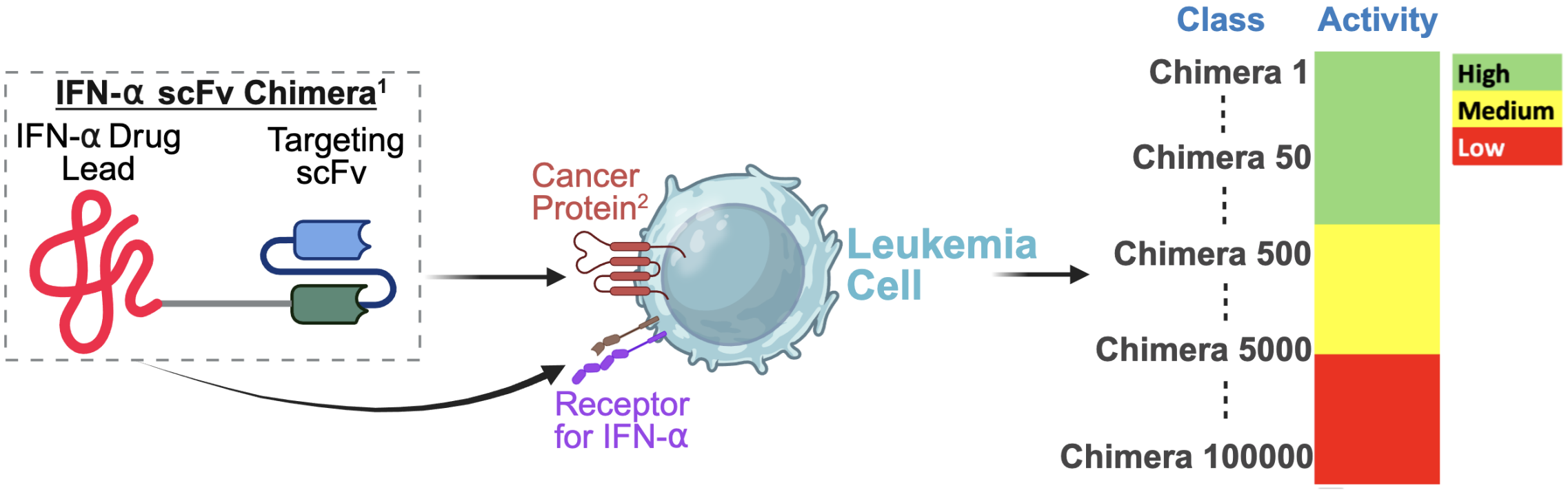
Visualize GigaAssay data output for bioactivity

Purify, verify, patent leads for clinical testing



Targeting IFN-α to Leukemia (GigaAssay Phase II)

- **Combine next-generation IFN-α with a targeting protein (scFv) to attack the source of the leukemia**
- **Reduces off-target side effects – limiting systemic toxicity**



[1]Connect a drug lead and other protein(s) into one molecule
[2]Cancer proteins such as CD33, CD123, FLT-3, CLL-1

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THANK YOU

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Appendix

🔬 Key Surface Markers on AML Myeloblasts

Marker	Function / Notes	Therapeutic Targeting
CD33	Highly expressed on most AML blasts	Target of **gemtuzumab ozogamicin** (Mylotarg, an antibody-drug conjugate)
CD123 (IL-3 receptor α chain)	Overexpressed on AML blasts and **leukemic stem cells (LSCs)**	Targeted in **clinical trials** (e.g., tagraxofusp, CD123 CAR-T, bispecifics)
CD34	Marker of stem/progenitor cells, including LSCs	Used for **diagnosis/prognosis**; less ideal for therapy due to expression on normal HSCs
CD117 (c-Kit)	Tyrosine kinase receptor on some AML subtypes	**KIT inhibitors** under investigation
CD38	Variable expression	Targeted in some AML studies (e.g., with daratumumab)
CLL-1 (CLEC12A)	Expressed on AML cells and LSCs but **not on normal HSCs**	**Promising therapeutic target** (CAR-T, bispecifics)
FLT3	Mutated in ~30% of AML; expressed on blasts	Targeted by **midostaurin, gilteritinib** (TKIs)
TIM-3, CD47, CD70	Immune checkpoint or immune evasion markers	Targeted by **emerging immunotherapies**

- **Ehninger, A. et al. (2014)** *"Targeting CD33 with chimeric antigen receptor T cells for the treatment of acute myeloid leukemia." Haematologica, 99(8), 1304–1312.*
- **Sarfati, M. et al. (2023)** *"CD123 as a biomarker and therapeutic target in hematologic malignancies: recent advances and future directions." Biomarker Research, 11(1).*
- **Laborda, E. et al. (2017)** *"Development of a chimeric antigen receptor targeting CLL-1 for human acute myeloid leukemia." Journal of Hematology & Oncology, 10, 105.*
- **Lv, J. et al. (2021)** *"Recent advances in the development of anti-CD123 antibody-drug conjugates for hematologic malignancies." Frontiers in Oncology, 11, 662460.*
- **Lohmueller, J.J. et al. (2020)** *"Chimeric antigen receptor T cells for treatment of AML: progress and challenges." Frontiers in Oncology, 10, 610009.*
- **Hanekamp, D. et al. (2017)** *"The leukemia stem cell marker CD123 in AML: expression and therapeutic targeting." Frontiers in Oncology, 7, 263.*
- **Zheng, B. et al. (2019)** *"CLL-1 is a selective target for acute myeloid leukemia." Scientific Reports, 9, 4110.*