2021 Sarcoma Exchange

WHAT WE NEED TO KNOW ABOUT TARGETED THERAPIES AND NGS IN 2021

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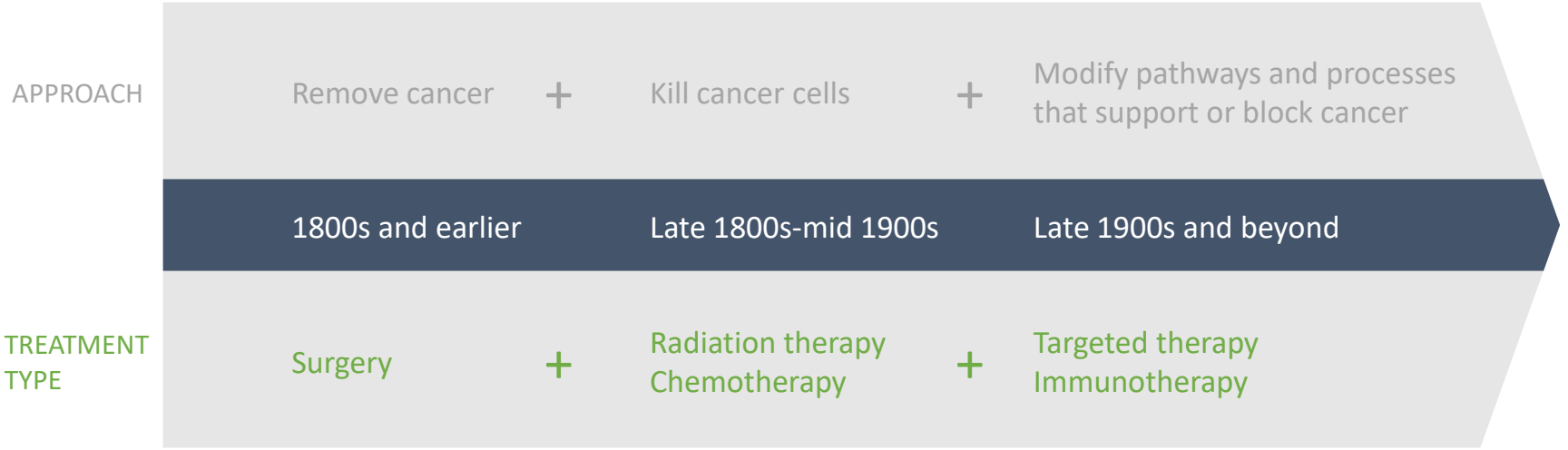
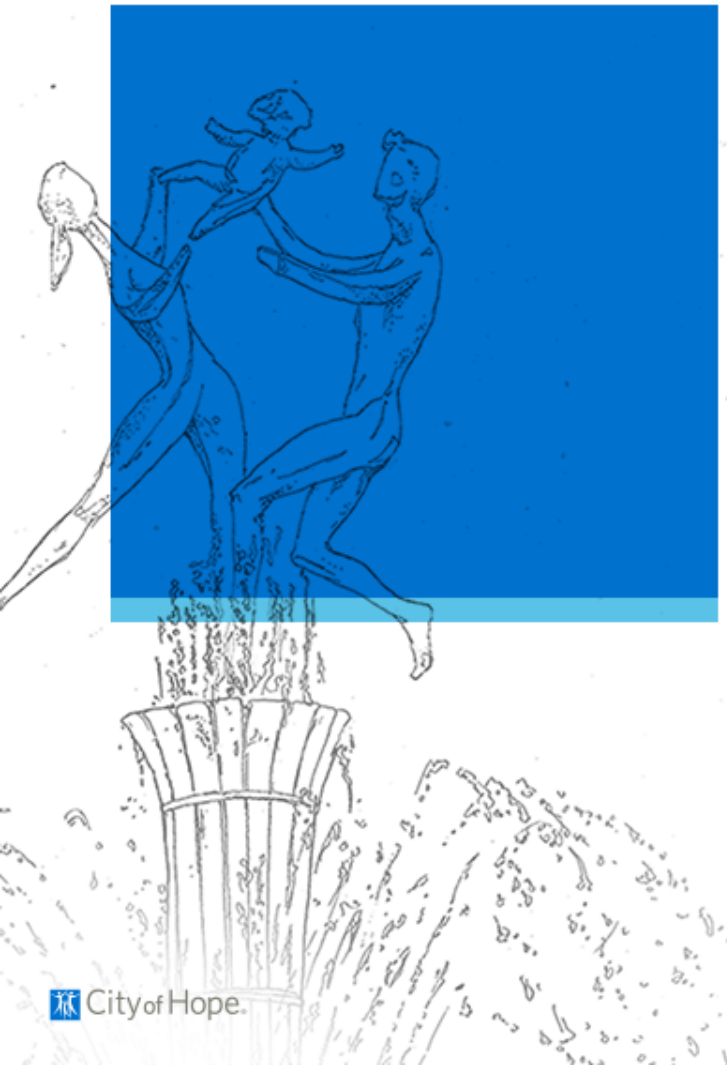
As Cancer Care Has Progressed, There Has Been a Trend Toward Targeted Treatment

Evolution of cancer treatment[2]



Targeted Therapies for Cancer



- Targeted cancer therapies are drugs designed to interfere with specific molecules necessary for tumor growth and progression.
- Ideally- A primary goal of targeted therapies is to fight cancer cells with more precision and potentially fewer side effects.
- Targeted cancer agents are broadly classified as:
  - **Therapeutic monoclonal antibodies** target specific antigens found on the cell surface.
  - **Small molecules** can penetrate the cell membrane to interact with targets inside a cell.

FDA Approved Targeted Therapies

Agent	Target(s)	FDA-approved indication(s)
Ado-trastuzumab emtansine (Kadcyla)	HER2 (ERBB2/neu)	Breast cancer (HER2+)
Afatinib (Gilotrif)	EGFR (HER1/ERBB1), HER2 (ERBB2/neu)	Non-small cell lung cancer
Aldesleukin (Proleukin)		Renal cell carcinoma Melanoma
Alectinib (Alecensa)	ALK	Non-small cell lung cancer
Avapritinib	KIT and PDGFR	GIST
Atezolizumab (Tecentriq)	PD-L1	Urothelial carcinoma Non-small cell lung cancer
Axitinib (Inlyta)	KIT, PDGFRβ, VEGFR1/2/3	Renal cell carcinoma
Bevacizumab (Avastin)	VEGF ligand	Cervical, Fallopian tube and Ovarian cancer Colorectal cancer Glioblastoma Non-small cell lung cancer Renal cell carcinoma
Cabozantinib (Cabometyx [tablet], Cometriq [capsule])	FLT3, KIT, MET, RET, VEGFR2	Medullary thyroid cancer Renal cell carcinoma
Ceritinib (Zykadia)	ALK	Non-small cell lung cancer
Cetuximab (Erbitux)	EGFR (HER1/ERBB1)	Colorectal cancer Squamous cell cancer of the head and neck
Cobimetinib (Cotellic)	MEK	Melanoma
Crizotinib (Xalkori)	ALK, MET, ROS1	Non-small cell lung cancer

Agent	Target(s)	FDA-approved indication(s)
Dabrafenib (Tafinlar)	BRAF	Melanoma
Denosumab (Xgeva)	RANKL	Giant cell tumor of the bone
Erlotinib (Tarceva)	EGFR (HER1/ERBB1)	Non-small cell lung cancer Pancreatic cancer
Everolimus (Afinitor)	mTOR	neuroendocrine tumor Renal cell carcinoma Breast cancer
Gefitinib (Iressa)	EGFR (HER1/ERBB1)	Non-small cell lung cancer
Imatinib (Gleevec)	KIT, PDGFR, ABL	GI stromal tumor Dermatofibrosarcoma protuberans
Ipilimumab (Yervoy)	CTLA-4	Melanoma
Lapatinib (Tykerb)	HER2 (ERBB2/neu), EGFR (HER1/ERBB1)	Breast cancer
Lenvatinib (Lenvima)	VEGFR2	Renal cell carcinoma Thyroid cancer
Necitumumab (Portrazza)	EGFR (HER1/ERBB1)	Squamous non-small cell lung cancer

Agent	Target(s)	FDA-approved indication(s)
Nivolumab (Opdivo)	PD-1	Head and neck squamous cell carcinoma Melanoma Non-small cell lung cancer Renal cell carcinoma Urothelial carcinoma
Olaparib (Lynparza)	PARP	Ovarian cancer
Osimertinib (Tagrisso)	EGFR	Non-small cell lung cancer
Palbociclib (Ibrance)	CDK4, CDK6	Breast cancer
Panitumumab (Vectibix)	EGFR (HER1/ERBB1)	Colorectal cancer
Pazopanib (Votrient)	VEGFR, PDGFR, KIT	Renal cell carcinoma Soft tissue sarcoma
Pembrolizumab (Keytruda)	PD-1	Melanoma Non-small cell lung cancer (PD-L1+) Head and neck squamous cell carcinoma
Pertuzumab (Perjeta)	HER2 (ERBB2/neu)	Breast cancer
Pexidartinib	CSF1R	tenosynovial giant cell tumor
Ramucirumab (Cyramza)	VEGFR2	Colorectal cancer Gastric cancer or Gastroesophageal junction Non-small cell lung cancer
Regorafenib (Stivarga)	KIT, PDGFRβ, RAF, RET, VEGFR1/2/3	Colorectal cancer Gastrointestinal stromal tumors

Agent	Target(s)	FDA-approved indication(s)
Ribociclib (Kisqali)	CDK4, CDK6	Breast cancer
Ripretinib	KIT and PDGFRα inhibitor	GIST
Sipuleucel-T (Provenge)		Prostate cancer
Sonidegib (Odomzo)	Smoothened	Basal cell carcinoma
Sorafenib (Nexavar)	VEGFR, PDGFR, KIT, RAF	Hepatocellular carcinoma Renal cell carcinoma Thyroid carcinoma
Sunitinib (Sutent)	VEGFR, PDGFR, KIT, RET	Renal Cell Carcinoma GIST Pancreatic NET
Tazemetostat	EZH2	Epithelioid Sarcoma
Temsirolimus (Torisel)	mTOR	Renal cell carcinoma
Trametinib (Mekinist)	MEK	Melanoma
Trastuzumab (Herceptin)	HER2 (ERBB2/neu)	Breast cancer Gastric cancer
Vandetanib (Caprelsa)	EGFR (HER1/ERBB1), RET, VEGFR2	Medullary thyroid cancer
Vemurafenib (Zelboraf)	BRAF	Melanoma
Vismodegib (Erivedge)	PTCH, Smoothened	Basal cell carcinoma
Ziv-aflibercept (Zaltrap)	PIGF, VEGFA/B	Colorectal cancer

Pazopanib- Multi-tyrosine Kinase Inhibitor

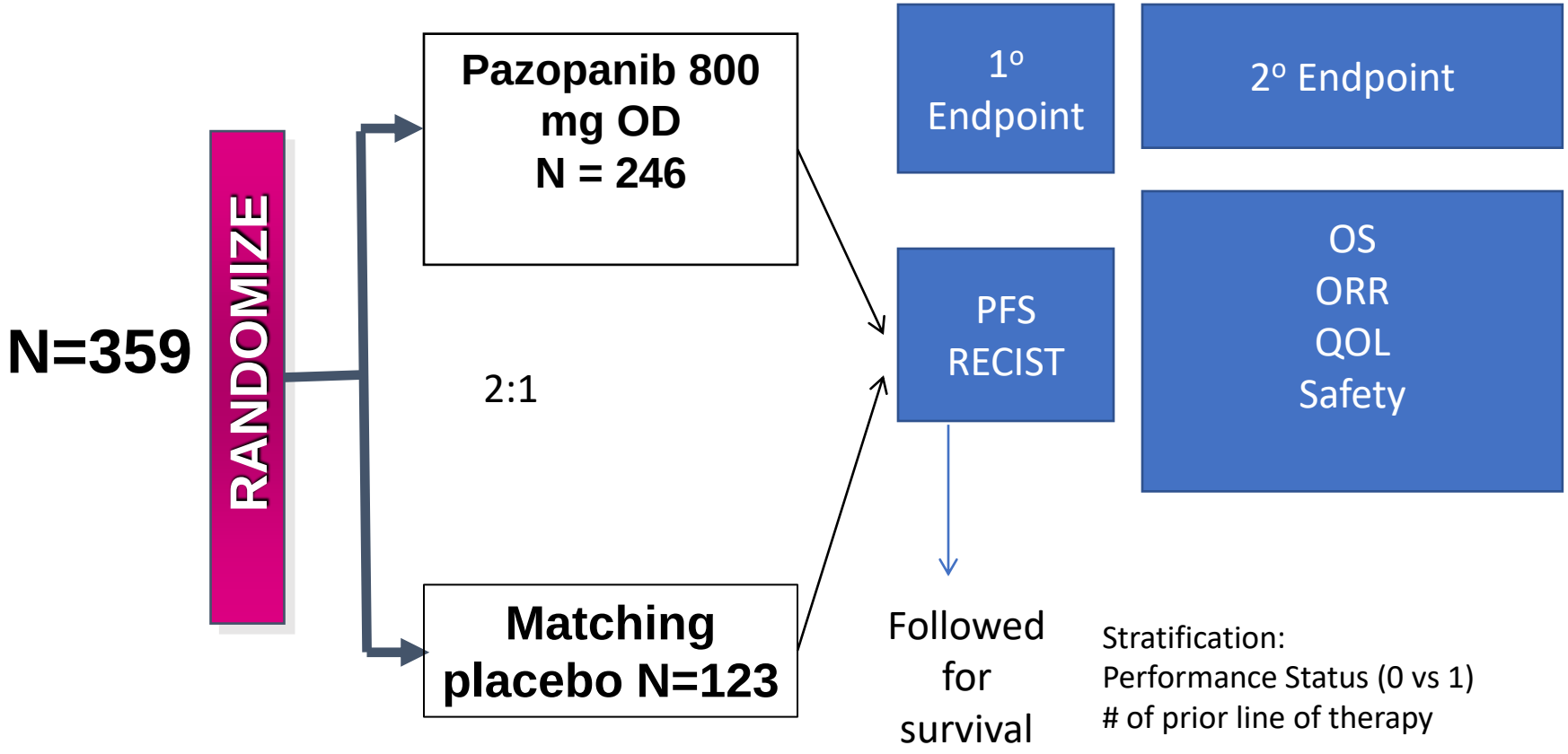
Pazopanib is a small-molecule TKI of growth factor receptors associated with angiogenesis and tumor cell proliferation

Pazopanib exhibits inhibition of:

- Vascular endothelial growth factor receptors (VEGFR-1, -2, and -3)
- Platelet-derived growth factor receptors (PDGFR-α and -β)
- Fibroblast growth factor receptors (FGFR-1 and -3)
- Stem cell factor receptor (c-Kit)
- Interleukin-2 receptor inducible T-cell kinase (Itk)
- Leukocyte-specific protein tyrosine kinase (Lck)
- Transmembrane glycoprotein receptor tyrosine kinase (c-Fms)

Sleijfer et. al., J Clin Oncol 2009; 3126

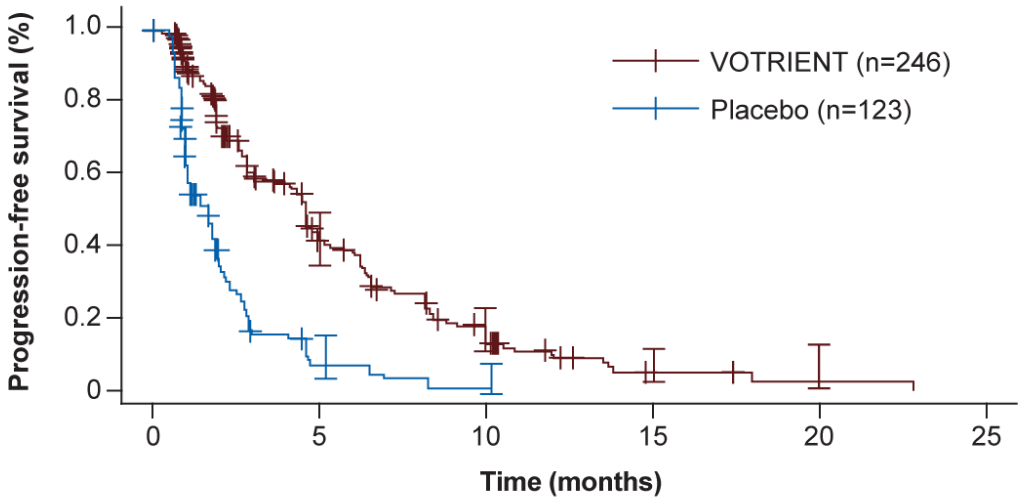
PALETTE (PAzopanib ExpLorEd in SofT-TissuE Sarcoma; EORTC 62072): Phase III Trial Pazopanib vs. Placebo in STS



Van Der Graaf et. al. J Clin Oncol 2011 Suppl: Abstr LBA10002; *Lancet.* 2012;379(9829):1879

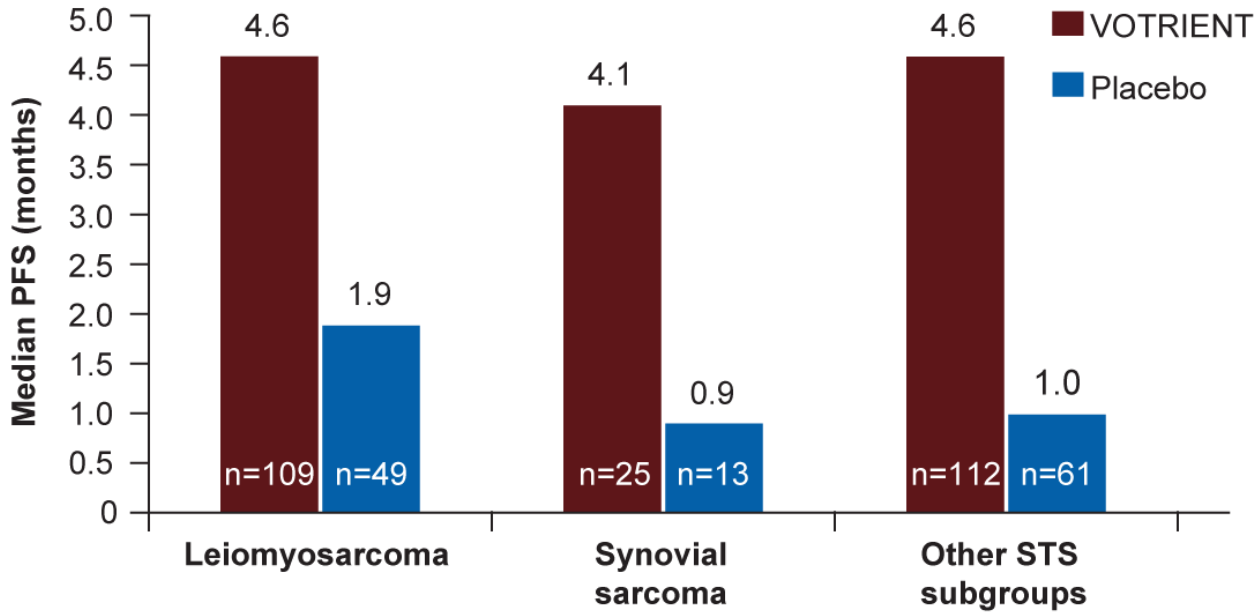
PALETTE Study
Efficacy: primary endpoint

Median PFS	pazopanib (n=246)	Placebo (n=123)
Months	4.6	1.6
HR (95% CI)	**0.35 (0.26-0.48)** *P<0.001*	
HR, Hazard ratio		



PALETTE Study
Efficacy: primary endpoint (cont'd)

- Pazopanib demonstrated PFS benefit in prespecified subgroups based on STS histology



Phase 3 Trials in Advanced STS

2012	2014	2015	2016	2017
PALETTE[1] pazopanib vs. placebo mOS: 12.5 vs. 10.7 mo HR: 0.86 (95% CI, 0.67-1.11) PFS: 4.6 vs. 1.6 mo	EORTC-62012[2] dox vs. dox + ifosfamide mOS: 12.8 vs. 14.3 mo HR: 0.83 (95% CI, 0.67-1.03) PFS: 4.6 vs. 7.4 mo	PICASSO-III[3] dox vs. dox + palifosfamide mOS: 16.9 vs. 15.9 mo HR: 1.05 (95% CI, 0.79-1.39) PFS: 5.2 vs. 6.0 mo	ET743-SAR-3007[4] trabectedin vs. dacarbazine mOS: 13.7 vs. 13.1 mo HR: 0.93 (95% CI, 0.75-1.15) PFS: 4.2 vs. 1.5 mo	SARC 21[6] dox vs. dox + evofosfamide mOS: 19.0 vs. 18.4 mo HR: 1.06 (95% CI, 0.88-1.29) PFS: 6.0 vs. 6.3 mo
			E7389-G000-309[5] eribulin vs. dacarbazine mOS: 13.5 vs. 11.5 mo HR: 0.77 (95% CI, 0.62-0.95) PFS: 2.6 vs. 2.6 mo	GeDDiS[7] dox vs. doce + gemcitabine mOS: 17.6 vs. 15.5 mo HR: 1.14 (95% CI, 0.83-1.57) PFS: 5.4 vs. 5.5 mo

Led to drug approval

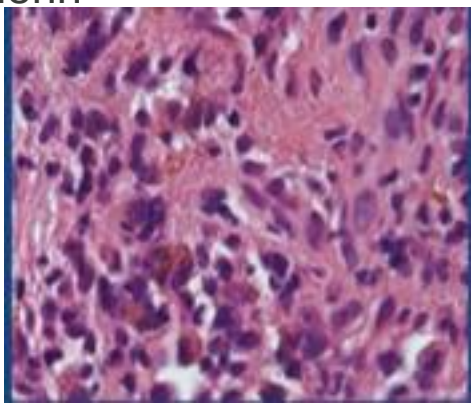
First Line | Second Line + | Third Line +

dox, doxorubicin; doce, docetaxel; EORTC, European Organisation for Research and Treatment of Cancer; GEDDIS, gemcitabine and docetaxel versus doxorubicin as first-line treatment in previously untreated advanced unresectable or metastatic soft-tissue sarcomas; mOS, median overall survival; mo, month; PICASSO, palifosfamide-tris with doxorubicin for soft tissue sarcoma; SARC, Sarcoma Alliance for Research Through Collaboration; STS, soft tissue sarcoma; wks, weeks.

1. Van der Graaf et al. *Lancet* 2012; 2. Judson I et al. *Lancet Oncol* 2014; 3. Ryan et al. *J Clin Oncol* 2016; 4. Trabectedin US prescribing information 2019; 5. Schöffski et al. *Lancet* 2016; 6. Tap et al. *Lancet Oncol* 2017; 7. Seddon et al. *Lancet Oncol* 2017

Tenosynovial Giant Cell Tumor (Pigmented Villonodular Synovitis)

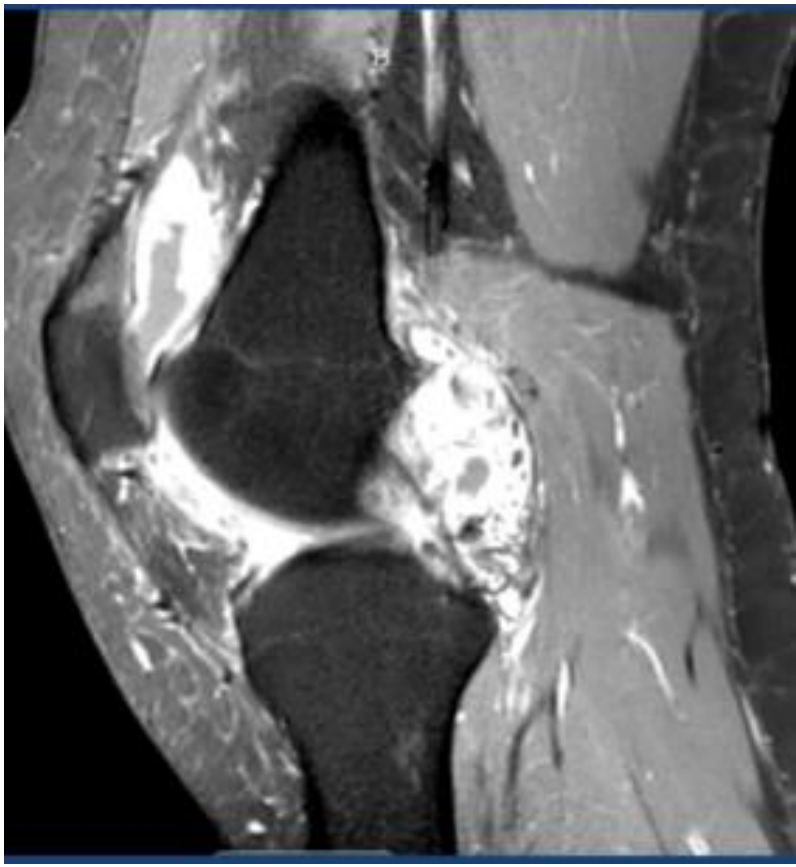
- Vascular, proliferative, inflammatory synovium
  - Multinucleated giant cells, macrophages, and hemosiderin



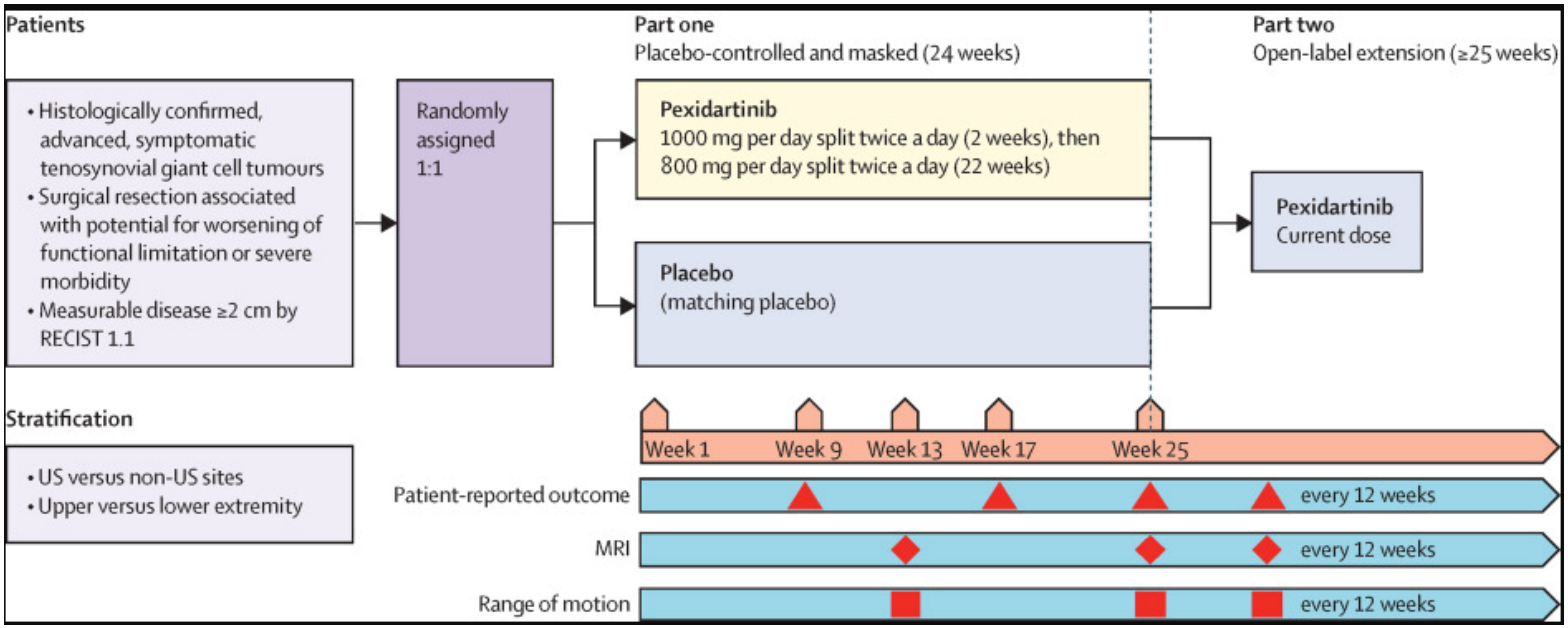
- Localized or diffuse-type growth pattern
- Intra- or extra articular locations
- Translocations (1p13)/alterations involving CSF1 gene
- ↑ CSF1 expression → macrophage recruitment to tumor site → CSF1/CSF1R autocrine/ paracrine loop of neoplastic/ non-neoplastic cells

Tenosynovial Giant Cell Tumor Therapies

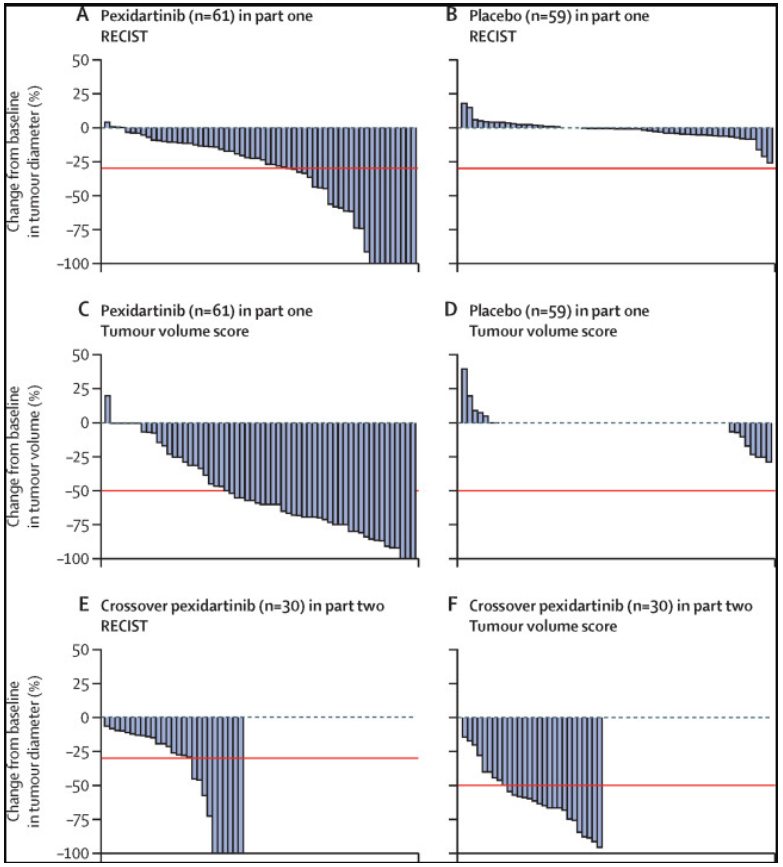
- Localized
  - Surgical- subtotal/ total resection, synovectomy to arthroplasty
  - Radiation/ radio-synovectomy
- Systemic → anti-CSF1R therapies



Study Design



Maximum change in tumor size according to RECIST and tumor volume score



On August 2, 2019, the FDA approved pexidartinib capsules for adult patients with symptomatic tenosynovial giant cell tumor (TGCT) associated with severe morbidity or functional limitations and not amenable to improvement with surgery.

Pexidartinib is the first systemic therapy approved for patients with TGCT.

The approval was based on durable ORR.

After 25 weeks of treatment, the ORR was 38% (95% confidence interval: 27, 50), with a 15% complete response rate and a 23% partial response rate.

No patients receiving placebo had a response ($p<0.0001$).

22 of 23 patients who responded and had been followed for a minimum of 6 months after the initial response maintained the response for ≥6 months.

Epithelioid Sarcoma

PHASE 2, INTERNATIONAL, MULTICENTER STUDY OF TAZEMETOSTAT IN ADULT PATIENTS WITH INI1-NEGATIVE TUMORS OR RELAPSED/REFRACTORY SYNOVIAL SARCOMA (NCT02601950)

COHORT 5* – INI1-NEGATIVE EPITHELIOID SARCOMA (ES) PATIENTS

- 2-stage Green-Dahlberg design
- Enrollment in cohort 5 initiated in December 2015, closed in September 2018
- Last data cut for cohort 5: September 17, 2018
- Conducted at 32 sites across USA, Europe and Asia
- Sponsored by Epizyme

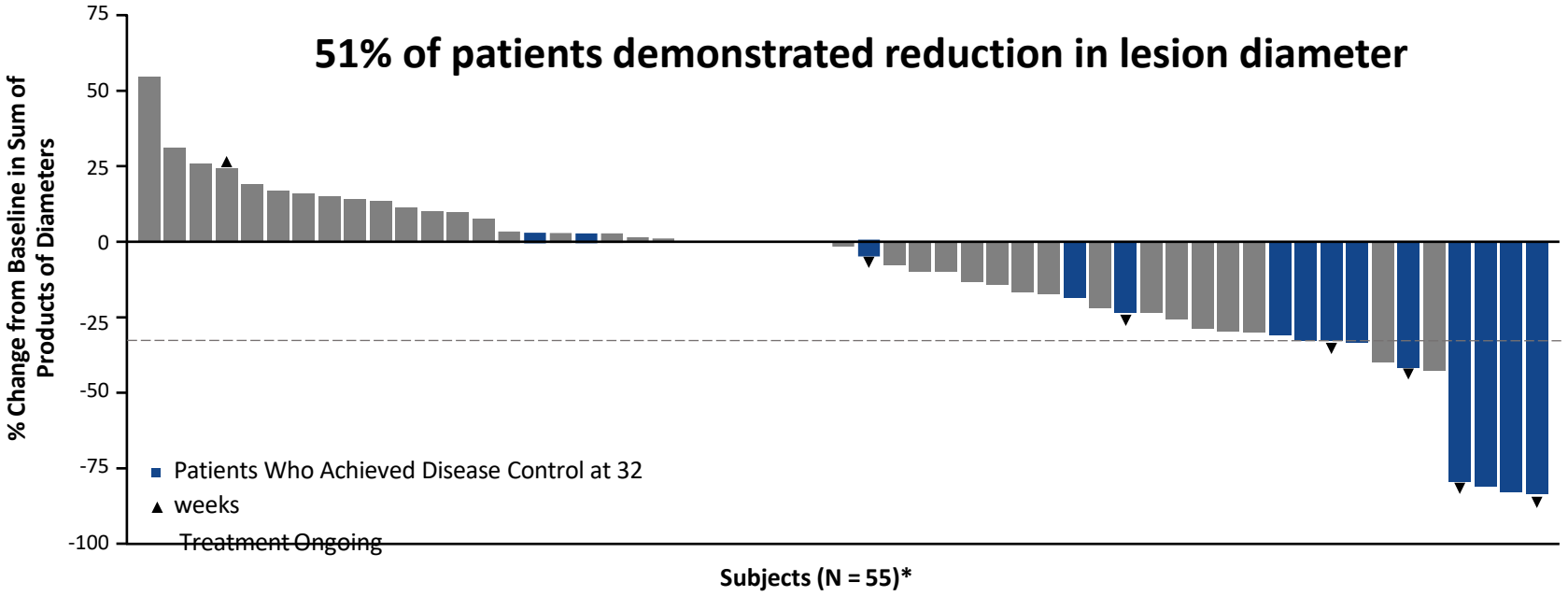
* Cohort 5 was 1 of 7 cohorts within the study.
ES, epithelioid sarcoma.

PRIMARY STUDY ENDPOINT: OBJECTIVE RESPONSE RATE (ORR) PER RECIST

Endpoint Category (RECIST), n (%)	No Prior Systemic Therapy (n=24)	Prior Systemic Anticancer Therapy (n=38)	Total (N=62)
ORR [CR+PR]¶ 95% CI	6 (25%) (9.8–46.7)	3 (8%) (1.7–21.4)	9 (15%) (6.9–25.8)
CR	0	0	0
PR	6 (25%)	3 (8%)	9 (15%)
SD	15 (63%)	20 (53%)	35 (56%)
PD	2 (8%)	11 (29%)	13 (21%)
Not evaluable	1 (4%)	4 (11%)	5 (8%)

¶ ORR is the percentage of subjects achieving a confirmed CR or PR from the start of tazemetostat until PD or the start of subsequent anticancer therapy, whichever is earlier. CI, confidence interval; CR, complete response; PD, progressive disease; PR, partial response; RECIST, response evaluation criteria in solid tumors.

BEST PERCENT CHANGE IN SUM OF DIAMETERS PER INVESTIGATOR ASSESSMENT



* Post-baseline sum of diameters was not calculated for 7 subjects in the Intent-to-Treat population due to a lack of complete tumor diameter measurements at any post-baseline visit. These subjects were excluded from the figure.

SUMMARY

FIRST PROSPECTIVE STUDY CONDUCTED IN EPITHELIOID SARCOMA

TREATMENT WITH TAZEMETOSTAT, AN INVESTIGATIONAL, FIRST-IN-CLASS ORAL EZH2 INHIBITOR, ACHIEVED

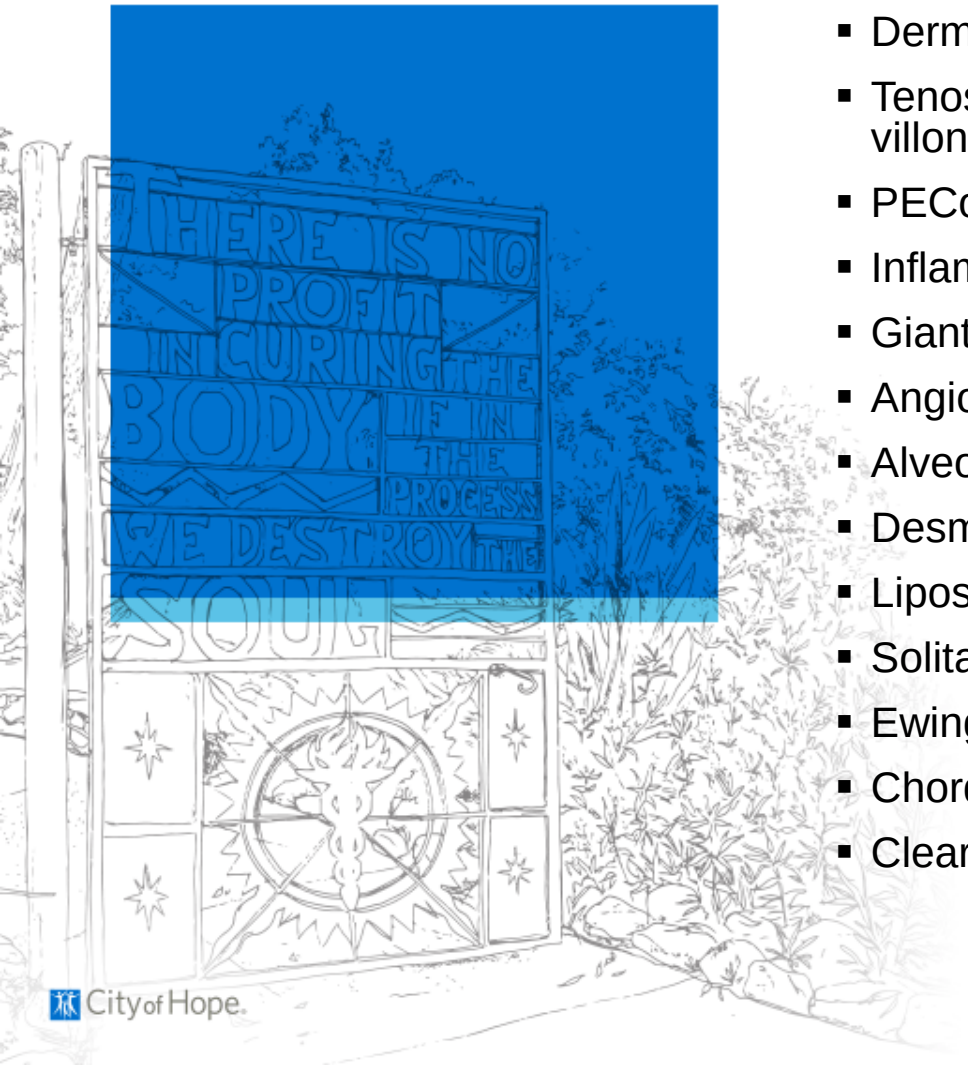
- AN ORR BY RECIST IN 15% OF ALL PATIENTS
- A DECREASE IN TUMOR SIZE IN 51% OF ALL PATIENTS
- DURABLE RESPONSES. AT A MEDIAN FOLLOW-UP OF 59.9 WEEKS, THE MEDIAN DOR WAS NOT REACHED
- A MEDIAN PFS OF 23.7 WEEKS, WITH 21.3% PATIENTS PROGRESSION-FREE AT 1 YEAR.
- A MEDIAN OS OF 82.4 WEEKS

TAZEMETOSTAT WAS GENERALLY WELL TOLERATED WITH NO TREATMENT-RELATED DEATHS AND <2% DEFINITIVE DISCONTINUATIONS

TAZEMETOSTAT, WAS APPROVED 1/23/2020 FOR ACCELERATED APPROVAL FOR THE TREATMENT OF PATIENTS WITH METASTATIC OR LOCALLY ADVANCED EPITHELIOID SARCOMA NOT ELIGIBLE FOR CURATIVE SURGERY

DOR, duration of response; ES, epithelioid sarcoma; EZH2, enhancer of zeste homolog 2; INI1, integrase interactor 1; ORR, objective response rate; PFS, progression-free survival; RECIST, response evaluation in solid tumors.

Sarcoma as a “targetable” disease



- Dermatofibrosarcoma protuberans
- Tenosynovial giant cell tumor/Pigmented villonodular synovitis
- PEComa
- Inflammatory myofibroblastic tumor
- Giant cell tumor of bone
- Angiosarcoma
- Alveolar soft part sarcoma
- Desmoid tumor/deep fibromatosis
- Liposarcomas
- Solitary fibrous tumor / HPC
- Ewing sarcoma
- Chordoma
- Clear cell sarcoma

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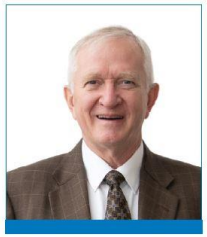

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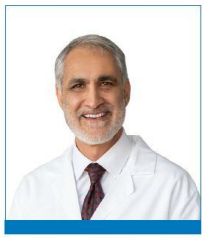

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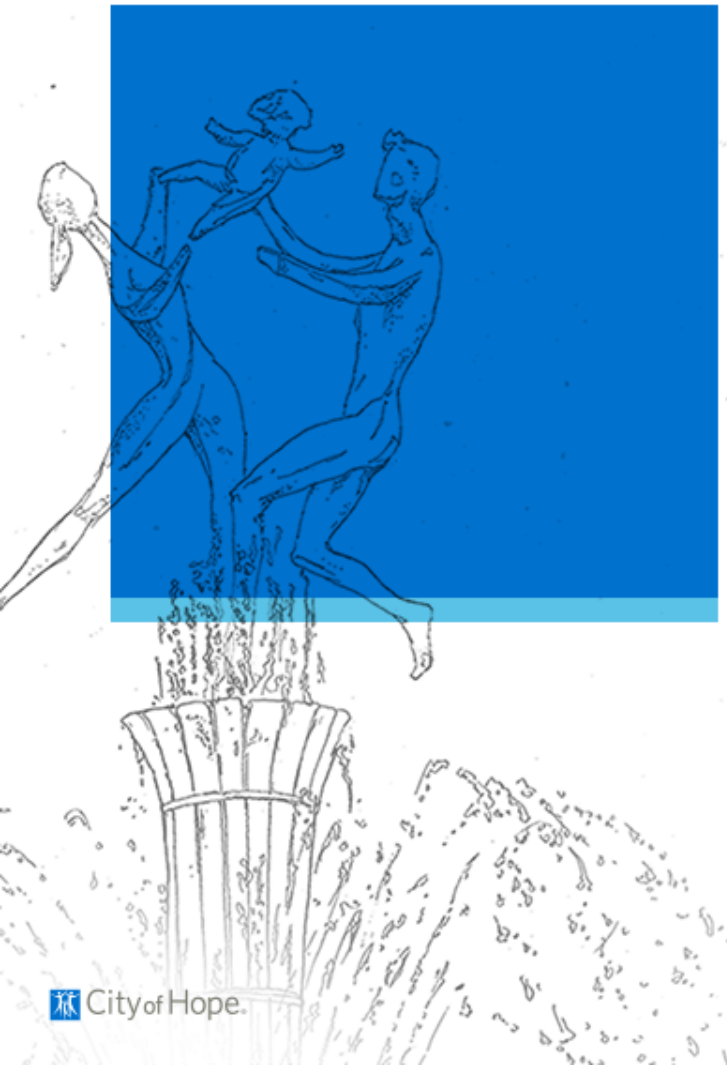
Future Directions



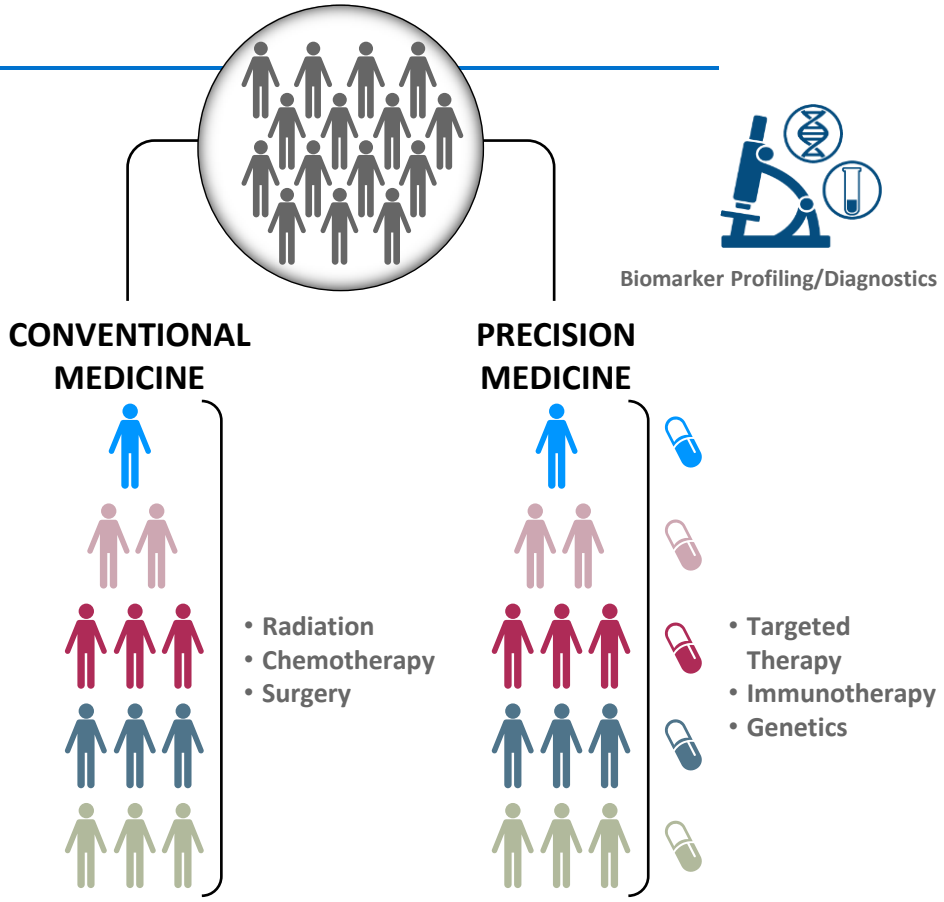
- As cancer care has progressed, there has been a trend toward targeted treatment
- The potential benefits of targeted therapies is realized only if patients can be tested and matched to appropriate treatments
- Many genomic alterations are rare: testing for only one (rare) alteration is not feasible; broad molecular profiling is needed
- Various methodologies are currently available, including several NGS platforms for comprehensive diagnostic testing

Growing Role of Precision Medicine

- With advances in medicine and genetics, over 135 drugs have been developed since 2018 that include genetic (pharmacogenomic) information in their labels
- A 2015 survey showed that over 40% of drugs in development now include biomarkers in their research and development study design
- An estimated 69% increase in the number of therapies developed by the year 2020
- Cancer remains at the vanguard of precision medicine: >70% of investigational cancer therapies are dependent on biomarker data
    - Broad range of actionable oncogenic biomarkers

Key Drivers in Growing Role of Precision Medicine

- Growing prevalence of cancer; increased understanding of cancer biology
- Large-scale human genome databases, NGS and computational tools
- Advances in targeted therapy against specific oncogenic molecular targets



Precision Medicine Has Particular Relevance to Oncology

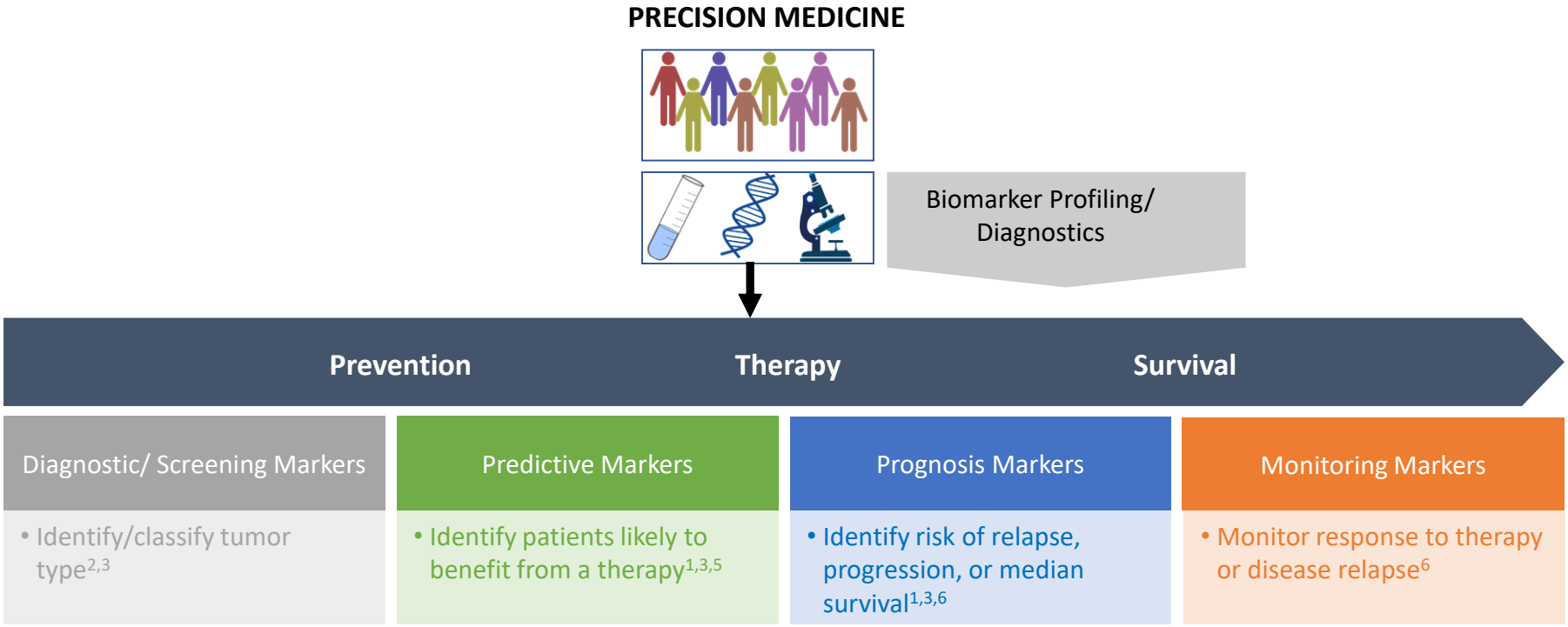
Precision Medicine: Aim
Prevent, diagnose, treat disease based on specific biological, genetic, environmental, disease characteristics[1,2]

Precision Medicine in Cancer
Use of therapies that may benefit subsets of patients with tumors that have specific genetic alterations or protein expression patterns

Genomic Profiling
30%-49% of patients with cancer undergoing genomic profiling have an actionable alteration that can be paired with an approved or investigational therapy

- 1. Yates LR et al. *Ann Oncol.* 2018;29:30-35. 2. National Cancer Institute. Dictionary of Cancer Terms: precision medicine. https://www.cancer.gov/publications/dictionaries/cancer-terms/def/precision-medicine. Accessed February 8, 2018. 3. Boland GM, et al. *Oncotarget.* 2015;6(24):20099-20110. 4. Massard C, et al. *Cancer Discov.* 2017;7(6):586-595.

Precision Medicine Impacts All Aspects of Cancer Care[1]



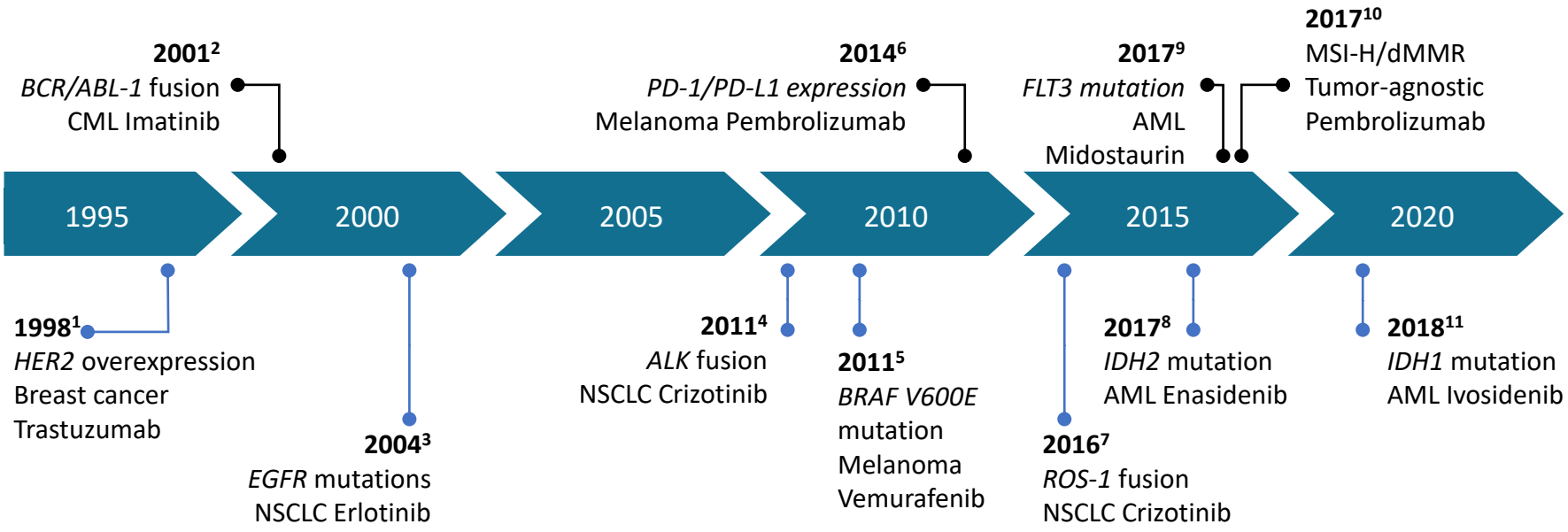
- 1. Meric_Bernstam F et al. *JCO* 2013;31:1849-1857. 2. Rossing M, et al. *Acta Oncol.* 2018;57(1):58-66. 3. Goossens N, et al. *Transl Cancer Res.* 2015 Jun;4(3):256-269. 4. Davare MA, et al. *Biol Cell.* 2015;107(5):111-129. 5. Fang B, et al. *Chin J Cancer.* 2015;34(7):295-309. 6. Dupain C, et al. *Mol Ther Nucleic Acids.* 2017;6:315-326.

Matching Actionable Alterations with Appropriate Therapies

- To realize the potential benefits of precision oncology for a specific cancer, actionable alterations must be matched with appropriate therapies[1]
- A genetic alteration may be actionable if it produces a protein product (or its immediate downstream effectors) that[2]:
  - Is part of a defined molecular pathway for which there is a corresponding FDA-approved or investigational drug
  - Can be differentially recognized in tumor cells versus normal cells by an established or experimental agent[2]
  - Predicts sensitivity or resistance to approved or standard therapies[3]

- 1. Schwaederle M, et al. *Oncoscience.* 2015;2(10):779–780.
  2. Goodman AM, et al. *JCO Precis Oncol.* 2017;1:1-13.
  3. Ross JS, et al. *Oncologist.* 2014;19:235-242.

Advent of Anti-cancer Treatments Targeting Specific Actionable Alterations

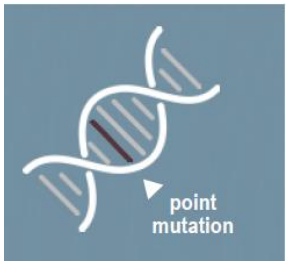


The Importance of Identifying Actionable Genomic Alterations

- The potential benefits of targeted therapies is realized only if patients can be tested and matched to appropriate treatments[1]
- Many genomic alterations are rare: testing for only one (rare) alteration is not feasible- broad molecular profiling is needed
  - No single biomarker will be relevant for every patient[1]
- Various methodologies are currently available, each with distinct strengths and limitations[2]
- Clinicians must make informed decisions about when/whom to test and which assays to use[3]

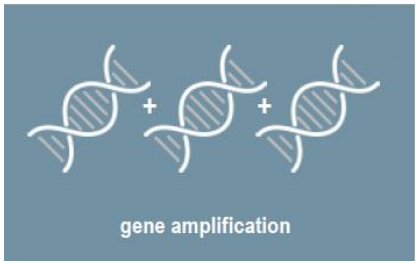
- 1. Frampton GM, et al. *Nat Biotechnol.* 2013 Nov;31(11):1023-31; 2. Lyons YA, et al. *NPJ Precis Oncol*. 2017;1(1):26; 3. Seidman AD, et al. *Popul Health Manag*. 2017;20(4):252-254.

Major Types of Genomic Alterations in Cancer



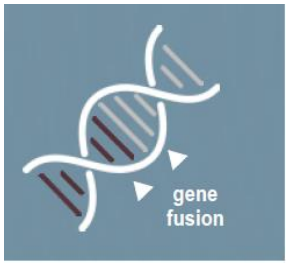
Mutations
Changes in the DNA sequence that makes up a gene[1]

- Includes base substitutions, insertions, deletions, and duplications[2]



Copy number variations/alterations
Changes in the number of copies of a gene[3]

- Includes gene amplification



Structural variations/rearrangements
Changes in the orientation, location, or number of copies of larger DNA segments[3]

- Includes fusions, translocations, inversions, deletions, and duplications

Distinguishing between the various alterations is important because not all alterations are driver alterations or are actionable

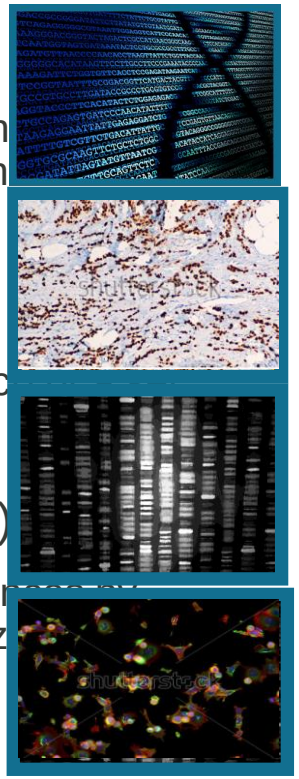
Most Actionable Genomic Alterations are Uncommon, Highlighting the Need for Comprehensive Genomic Testing[1]

- An NGS-based cancer genome profiling test interrogated 4,557 exons of 287 cancer-related genes
  - Detected all classes of clinically relevant genomic alterations in a single, tissue-sparing test
- The average number of actionable alterations in any individual patient's sample was low (1.57)
- However, wide variety of alterations found across *all* samples:– 1579 unique alterations
- Comprehensive testing can accurately detect most genomic alterations in all therapeutically relevant cancer genes in a single assay

• 1. Frampton GM, et al. *Nat Biotechnol* 2013;31:1023-1031.

Detecting Actionable Genomic Alterations

- Next Generation Sequencing (NGS)
  - High-throughput nucleic acid sequencing that allow parallel sequen
    multiple targets and multiple samples in order to detect concomitan
    in the same run[1]
- Immunohistochemistry (IHC)
  - Detects antigen of interest (protein) using a labelled antibody (direc
    of monoclonal or polyclonal antibodies (indirect)[2]
- Reverse Transcription Polymerase Chain Reaction (RT-PCR)
  - RNA molecules converted into complementary DNA (cDNA) sequen
    reverse transcriptases, followed by amplification of newly synthesiz
    standard qPCR procedures
- Fluorescence in situ Hybridization (FISH)
  - Uses DNA or RNA probes labeled with a fluorophore or modified nucleotide to bind complementary sequences



Future Directions in Precision Oncology

- 73% of oncology compounds in development are precision medicines[1]

- NCI-MATCH (NCT02465060) is a precision oncology clinical trial[2]
  - Determine how treatment directed by genetic testing works in patients with solid tumors or lymphomas that have progressed after ≥ 1 line of standard treatment or for which no agreed upon treatment approach exists
  - Patients to receive treatment based on genomic alterations found in their tumors rather than tissue type

Eligibility Criteria

- Age ≥18 years
- Histologically documented solid tumors or confirmed diagnosis of lymphoma or multiple myeloma
- Measurable disease
- ECOG performance status ≤ 1 and life expectancy of ≥ 3 months

Treatment Arms*

- 22 different treatments/interventions
- 50 different tumor types
  - solid tumors
  - Lymphomas
  - multiple myeloma

Endpoints

- Primary endpoint
  - ORR- the percentage of patients whose tumors have a complete or partial response to treatment (up to 3 years)
- Secondary endpoints
  - OS, evaluated specifically for each drug
  - PFA
  - Time to progression

*Currently enrolling treatment arms as of September, 2018

Impact of Next Generation Sequencing (NGS) On the Treatment of Patients with Sarcoma

Background:

- NGS is increasingly being used for patients with Sarcoma
- The role of NGS in the management of patients with Sarcoma remains undefined
- Basket trials, and tissue agnostic therapies are increasingly prevalent and may be compelling options for patients in later lines of treatment

Objective:

- To review usage of NGS testing in patients with Sarcoma
- To characterize the effect of NGS on management of patients with Sarcoma
- Better qualify instances in which NGS was utilized in order to understand incidence of mutations within a large population of Sarcoma patients

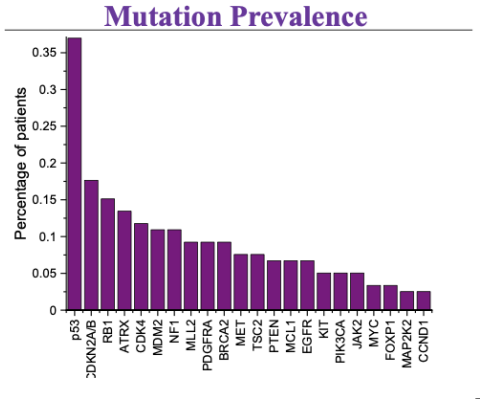
Results

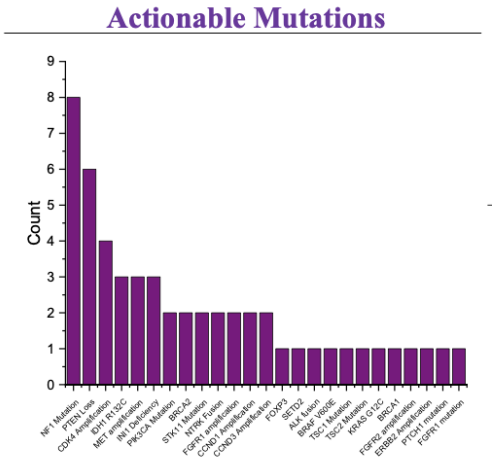
- A Total of 117 patients were analyzed with assays performed between 2018 and 2020

- **Patient Demographics and Clinical**

Characteristics	Frequency (%)
Age- Median (Range)	55 (20-94)
Male **Female**	57 (48%) 60 (52%)
Assays Per Patient (Range)	1 (1-4)
Histologic Subtypes **Leiomyosarcoma** **STS NOS** **Angiosarcoma** **Liposarcoma** **Other**	 36 (31%) 21 (18%) 13 (11%) 11 (9%) 36 (31%)

Results





Patients for whom NGS Altered Management

Histology	Mutation	Treatment
Ewings Sarcoma	PTEN Loss	Copanlisib
Leiomyosarcoma	ALK Fusion	Alectinib
Leiomyosarcoma	MSI-H	Nivolumab
Chondrosarcoma	IDH1	Ivosidenib
Synovial Sarcoma	BRAF V600E	Encorafenib
STS NOS	CDK4 Amplification	Palbociclib
STS NOS	CCND1 Amplification	Palbociclib
STS NOS	NTRK Fusion	Larotrectinib
Myxofibrosarcoma	High TMB	Atezolizumab

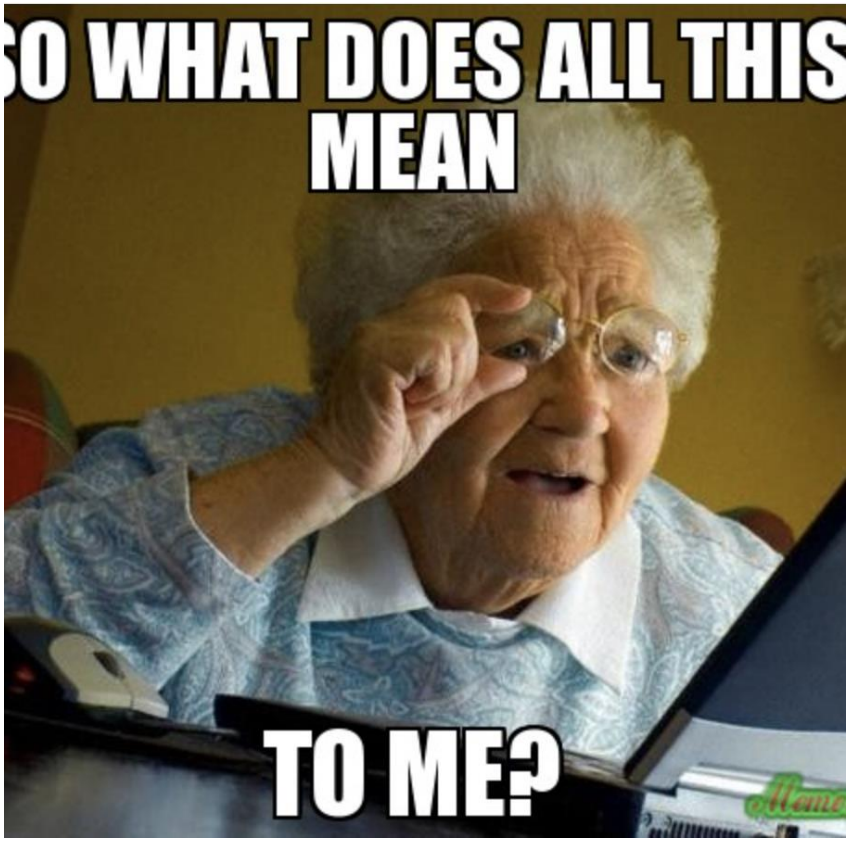
Conclusions

34% of patients had potentially actionable Mutations

Treatment of 8% of patients was altered by NGS results

Incidence of actionable mutations increased over time

Partial responses in select refractory patients

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Questions, Comments or More Information

https://www.cityofhope.org/research/find-a-clinical-trial


THERE IS NO
PROFIT
IN CURING THE
BODY IF IN
THE
PROCESS
WE DESTROY THE
SOUL