Investor Day

March 6, 2019

www.g1therapeutics.com

NASDAQ: GTHX

Forward-looking statements

This presentation and the accompanying oral commentary contain “forward-looking” statements within the meaning of the Private Securities Litigation Reform Act of 1995. Words such as "may," "will," "expect," "plan," "anticipate," "estimate," "intend" and similar expressions (as well as other words or expressions referencing future events, conditions or circumstances) are intended to identify forward-looking statements. Forward-looking statements in this presentation include, but are not limited to the following: the therapeutic potential of trilaciclib, lerociclib and G1T48; initial success in our ongoing clinical trials may not be indicative of results obtained when these trials are completed or in later stage trials; our development of trilaciclib to reduce chemotherapy-induced myelosuppression is novel, unproven and rapidly evolving and may never lead to a marketable product; our product candidates may cause undesirable side effects that could delay or prevent their marketing approval, limit the commercial profile of an approved label, or result in significant negative consequences following marketing approval, if any; we may not have the ability to recruit, enroll and complete clinical trials for, obtain approvals for, or commercialize any of our product candidates; we face substantial competition, which may result in others discovering, developing or commercializing competing products before or more successfully than we do; we may incur additional costs or experience delays in completing clinical trials; future legislation may increase the difficulty and cost for us to obtain marketing approval of and commercialize our product candidates and affect the prices we may obtain; and market conditions. Each of these forward-looking statements involves risks and uncertainties and are based on our expectations and assumptions as of the date of this presentation. Factors that may cause our actual results to differ from those expressed or implied in the forward-looking statements in this presentation are further discussed in our filings with the U.S. Securities and Exchange Commission (SEC), including the "Risk Factors" section in our annual report on Form 10-K for the fiscal year ended December 31, 2018 filed with the SEC. Such factors may be amended or updated from time to time in our subsequent periodic and other filings with the SEC, which are accessible on the SEC’s website at www.sec.gov. We assume no obligation to update any forward-looking statement after the date of this presentation to reflect any change in expectations or future developments, even as new information becomes available.

Housekeeping items

Q&A sessions will follow the trilaciclib and lerociclib/G1T48 sections of the agenda

- please use microphones in the room
- webcast participants: submit questions via “Ask a Question” link on top right of screen

Full presentation and speaker bios available at

→ **http://investor.g1therapeutics.com/events-and-presentations**

Please contact Jeff Macdonald with questions/requests

→ **jmacdonald@g1therapeutics.com**

G1 OVERVIEW

Mark Velleca, M.D., Ph.D.
Chief Executive Officer

Hematopoietic stem and progenitor cells (HSPCs)

Vision: improve the lives of those affected by cancer

1

Three wholly-owned investigational therapies with potential to improve patient care and generate significant value for shareholders

2

Relentless focus on patients, operational efficiency and financial discipline

3

Explore value-creating partnerships as we move toward commercialization of our product candidates

Robust clinical-stage pipeline

Three wholly-owned product candidates addressing distinct multi-billion dollar markets

Trilaciclib

First-in-class myelopreservation therapy

- ✓ Backbone with chemo
- ✓ 2018: positive SCLC data in three randomized Ph 2 trials

Next milestone in 2Q19: regulatory update

Lerociclib

Oral CDK4/6 inhibitor

- ✓ Combine with targeted Rx
- ✓ 2018: demonstrated POC in ER+ BC Ph 1 trial

Next milestones in 2H19: Clinical data updates in ER+ BC and EGFRm NSCLC trials

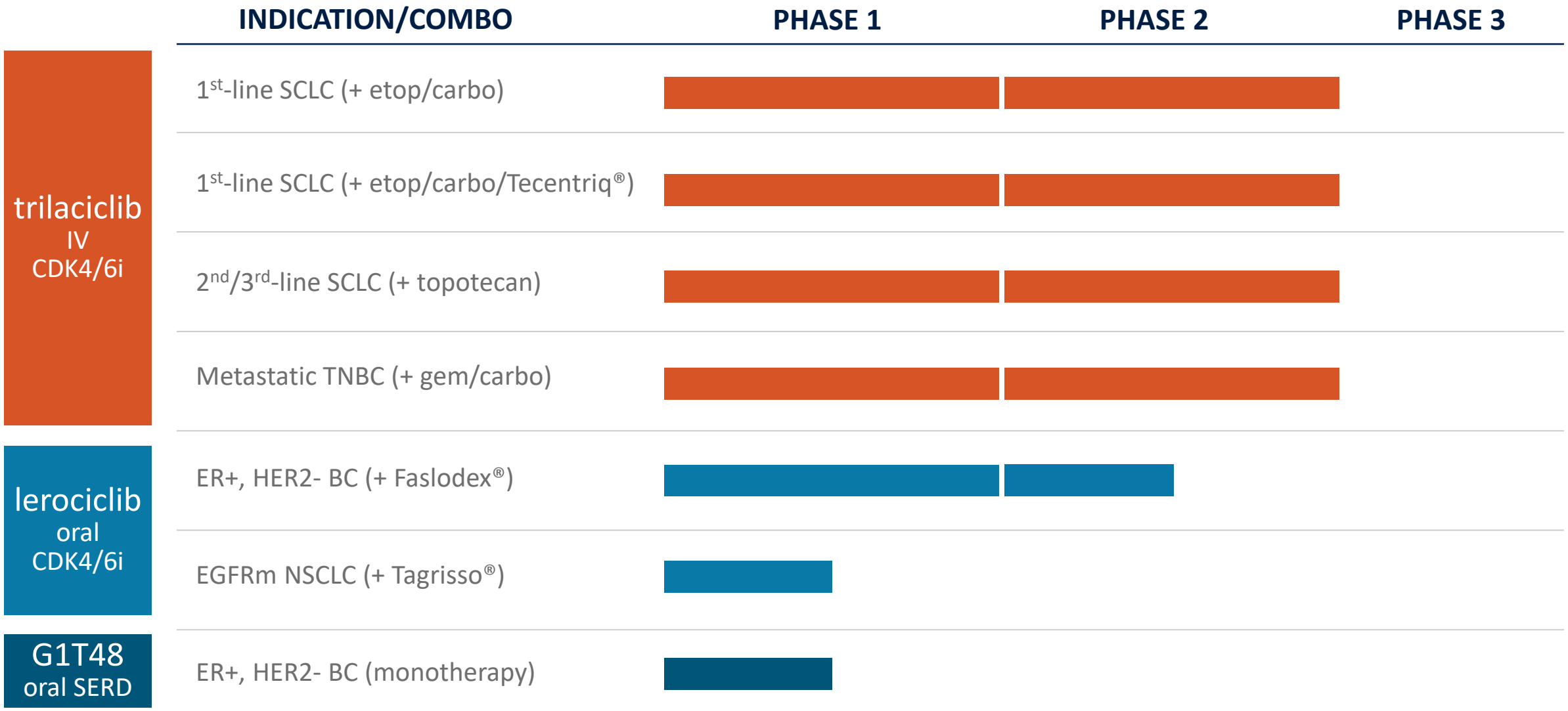
G1T48

Oral SERD ER+ breast cancer

- ✓ Monotherapy & lero combo
- ✓ 2018: initiated ER+ BC Ph 1 trial

Next milestone in 4Q19: Phase 1 data

Delivered on all clinical milestones in 2018



Experienced management team

Mark Velleca, M.D., Ph.D.
Chief Executive Officer

John Demaree
Chief Commercial Officer

J. Stillman Hanson
General Counsel

Raj Malik, M.D.
Chief Medical Officer and SVP, R&D

Terry Murdock
Chief Operating Officer

Buck Phillips
Chief Financial Officer and SVP, Corporate Development

Jay Strum, Ph.D.
Chief Scientific Officer

Today's agenda

Consequences of chemotherapy in the cancer patient

- **Jeff Crawford, M.D.,** George Barth Geller Professor for Research in Cancer
  Co-Director, Solid Tumor Therapeutics Program, Duke Cancer Institute

Trilaciclib development update

- **Raj Malik, M.D.,** G1 Chief Medical Officer and Senior Vice President, R&D

Myelopreservation and trilaciclib: implications for clinical practice

- **Lowell Hart, M.D.,** Scientific Director of Clinical Research, Florida Cancer Specialists
  Associate Professor of Medicine, Hematology and Oncology, Wake Forest University School of Medicine

Trilaciclib commercial strategy

- **John Demaree,** G1 Chief Commercial Officer

Lerociclib and G1T48 development update

- **Raj Malik, M.D.,** G1 Chief Medical Officer and Senior Vice President, R&D

DUKE CANCER INSTITUTE

A National Cancer Institute-designated Comprehensive Cancer Center

DukeMedicine

The Consequences of Chemotherapy in the Cancer Patient

Jeffrey Crawford, MD

George Barth Geller Professor for Research in Cancer

Co-Director, Solid Tumor Therapeutics Program

Duke Cancer Institute

Disclosures
Jeffrey Crawford, M.D.

- **Scientific Advisor:** AstraZeneca, Coherus, Enzychem, G1 Therapeutics, Merck, Pfizer, Spectrum

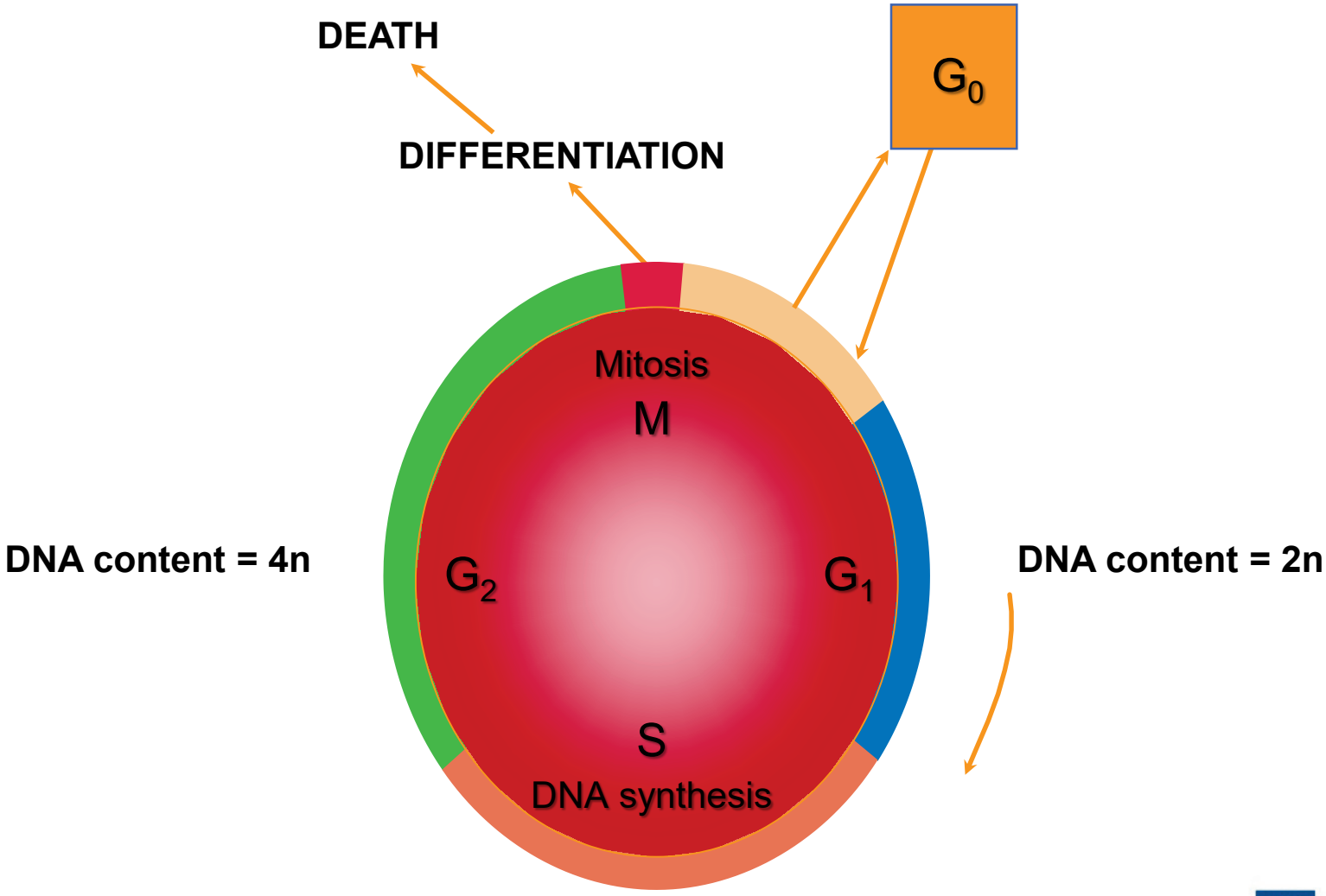
- **DSMB Member:** BeyondSpring, Mylan, Roche

- **Principal Investigator/Institutional Research Funding:** AstraZeneca, Genentech, Helsinn

Scientific Background

- Medical Oncologist with focus in lung cancer and thoracic malignancies
- Longstanding clinical and academic interest on supportive care in cancer, particularly hematopoietic growth factors and biosimilars in conjunction with chemotherapy
- PI for registrational trial of G-CSF (filgrastim) leading to FDA approval and subsequent NEJM publication
- Chair of NCCN Myeloid Growth Factor Committee for 15 Years
- Member of ASCO and ESMO Guidelines on CSFs
- Member of ANC Steering Committee, focused on outcomes research regarding neutropenia and chemotherapy dosing

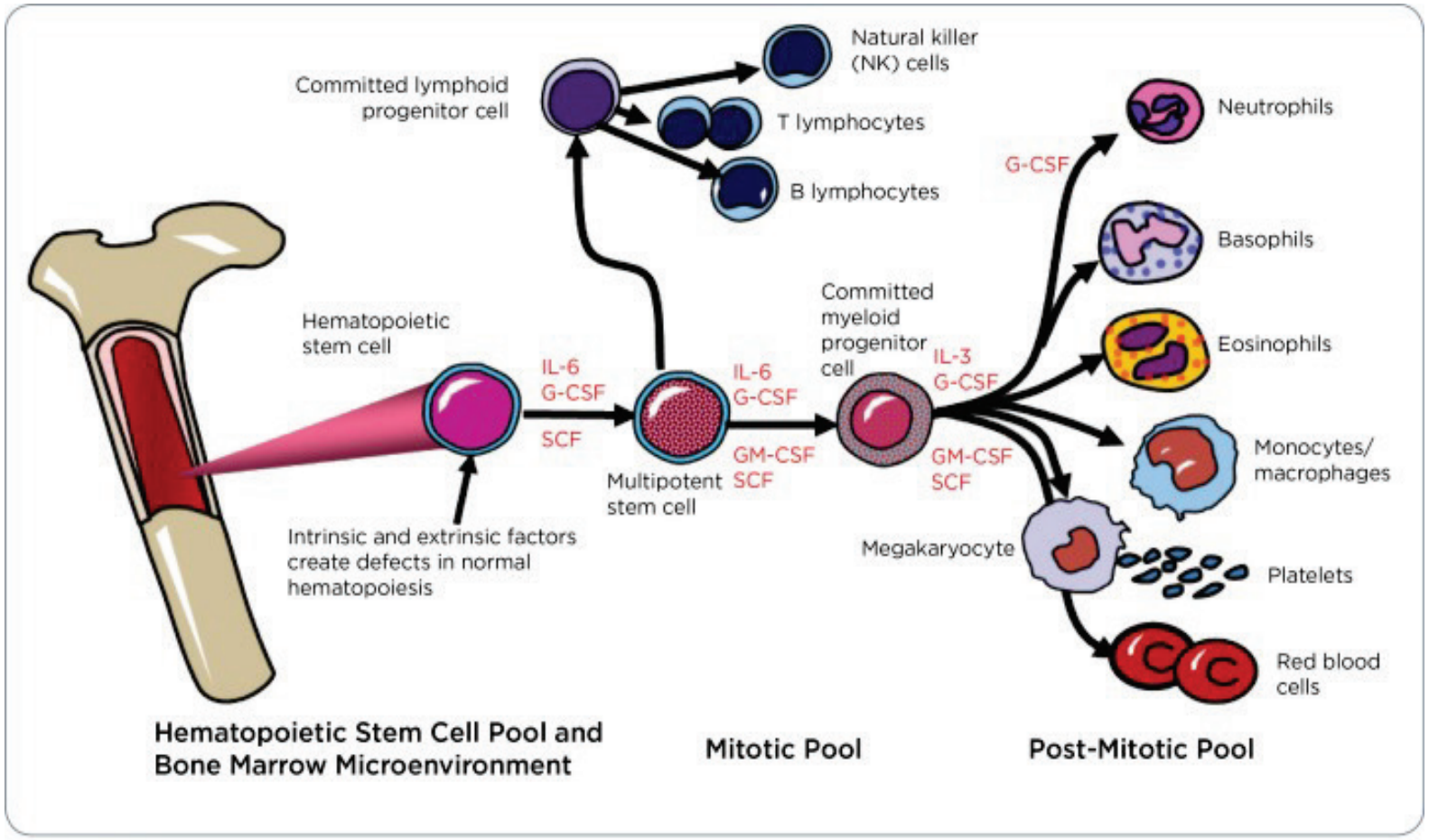
Chemotherapy Disrupts the Cell Cycle of Cancer and Normal Cells



Chemotherapy and the Bone Marrow

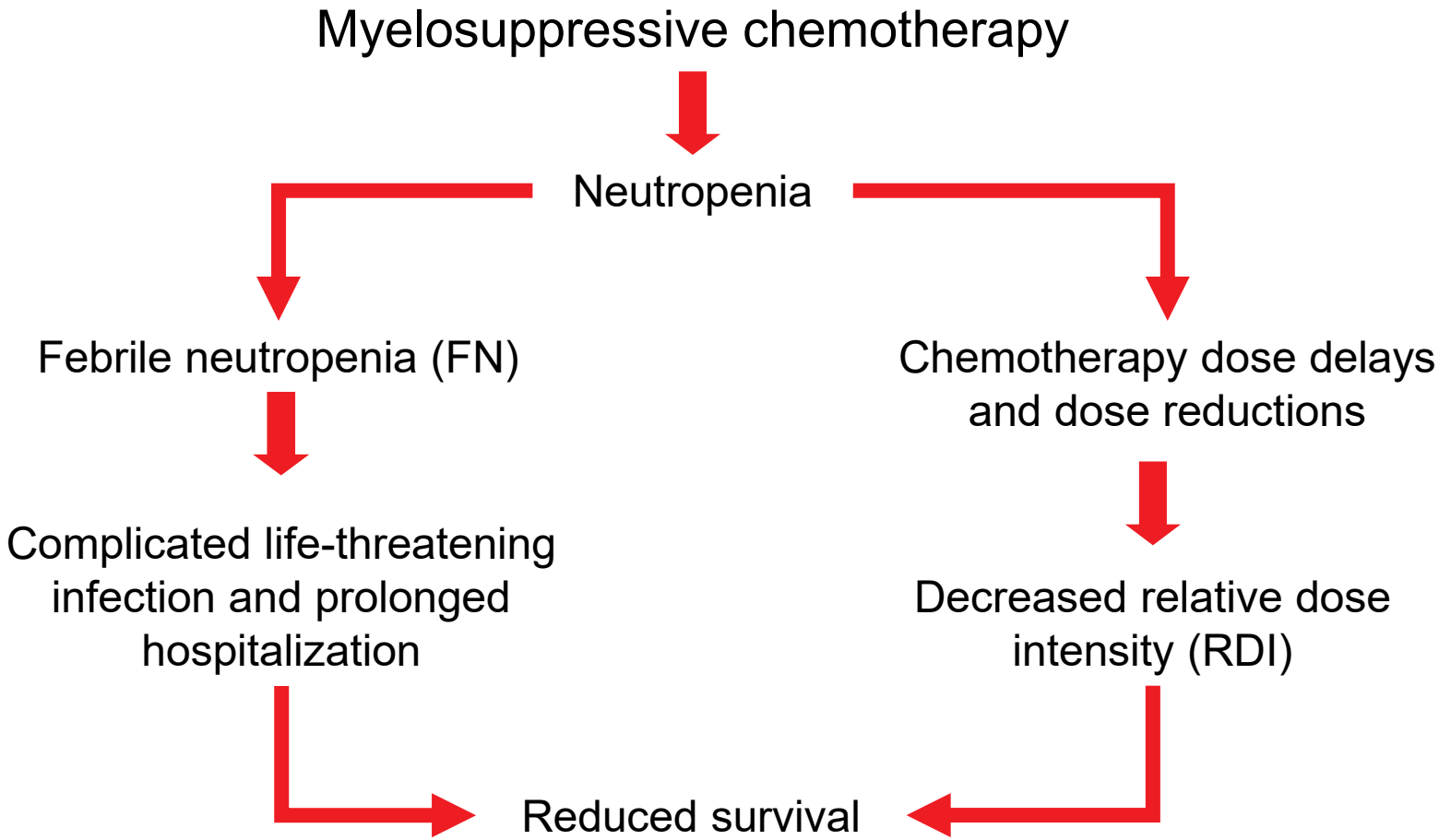
- Chemotherapy remains the most common systemic cancer treatment in both the curative and palliative settings
- Myelosuppression, or decreased blood cell production from the bone marrow, is a direct result of the cytotoxic effect of chemotherapy
- Myelosuppression is the most common dose limiting and life threatening complication of chemotherapy
- Therapies to reduce myelosuppression to date have focused on stimulation of the production of bone marrow cells by the use of growth factors after chemotherapy

Normal Hematopoiesis



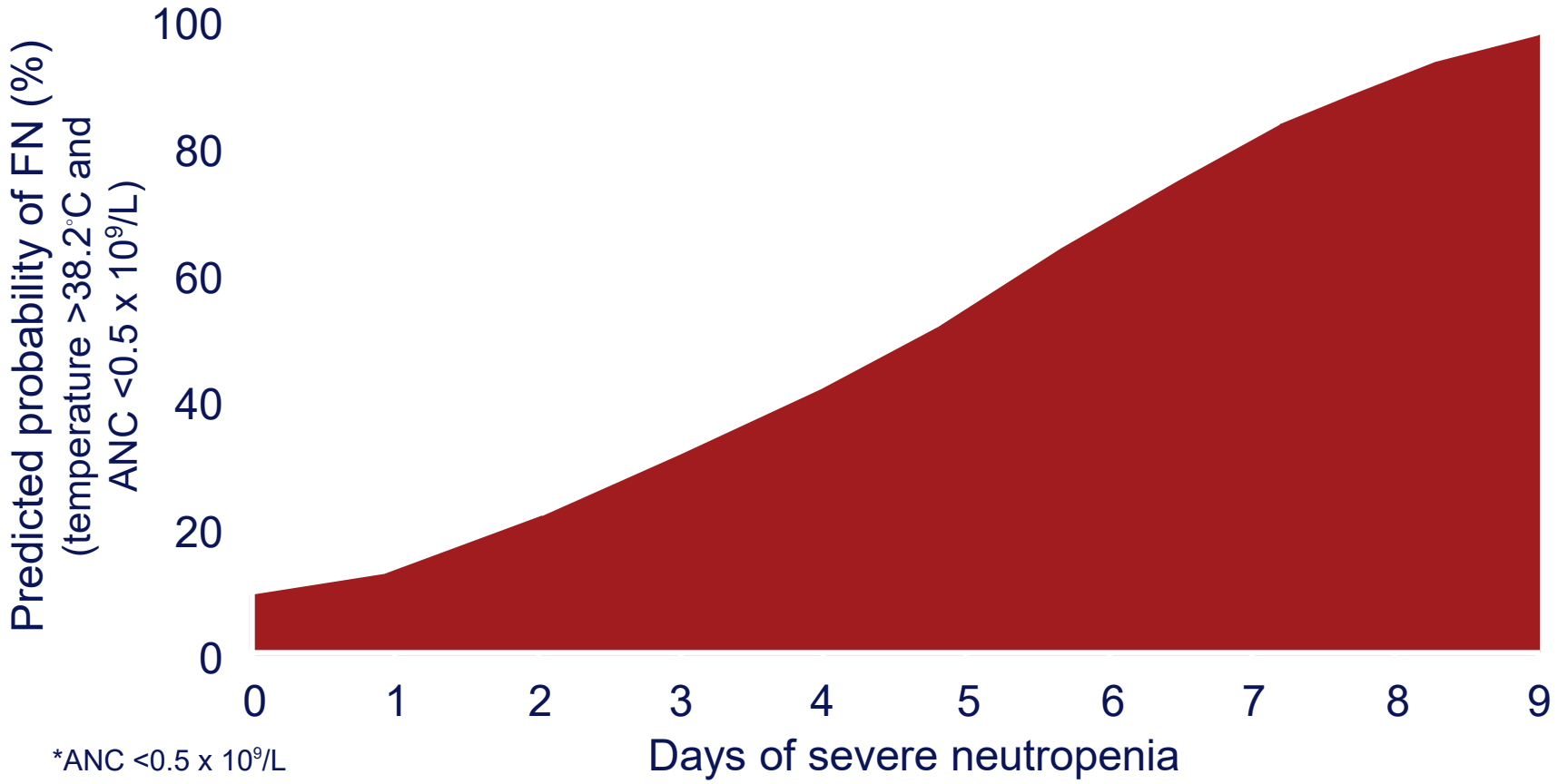
Kurtin S. J Adv Pract Oncol. 2012 Jul-Aug; 3(4): 209–224.

Chemotherapy-induced neutropenia and its complications



Kuderer NM et al. *Cancer* 2006;106:2258–2266
Chirivella I et al. *J Clin Oncol* 2006;24;abstract 668
Bosly A et al. *Ann Hematol* 2008;87:277-283

Risk of FN increases with duration of severe neutropenia*



*ANC <0.5 x 10^9/L

Adapted from Blackwell S, Crawford J. In: Morstyn G, Dexter TM. Filgrastim; (r-metHuG-CSF) in Clinical Practice. New York: Marcel Dekker; 1994 p 103–116
Crawford J et al. *N Engl J Med* 1991;325:164–170

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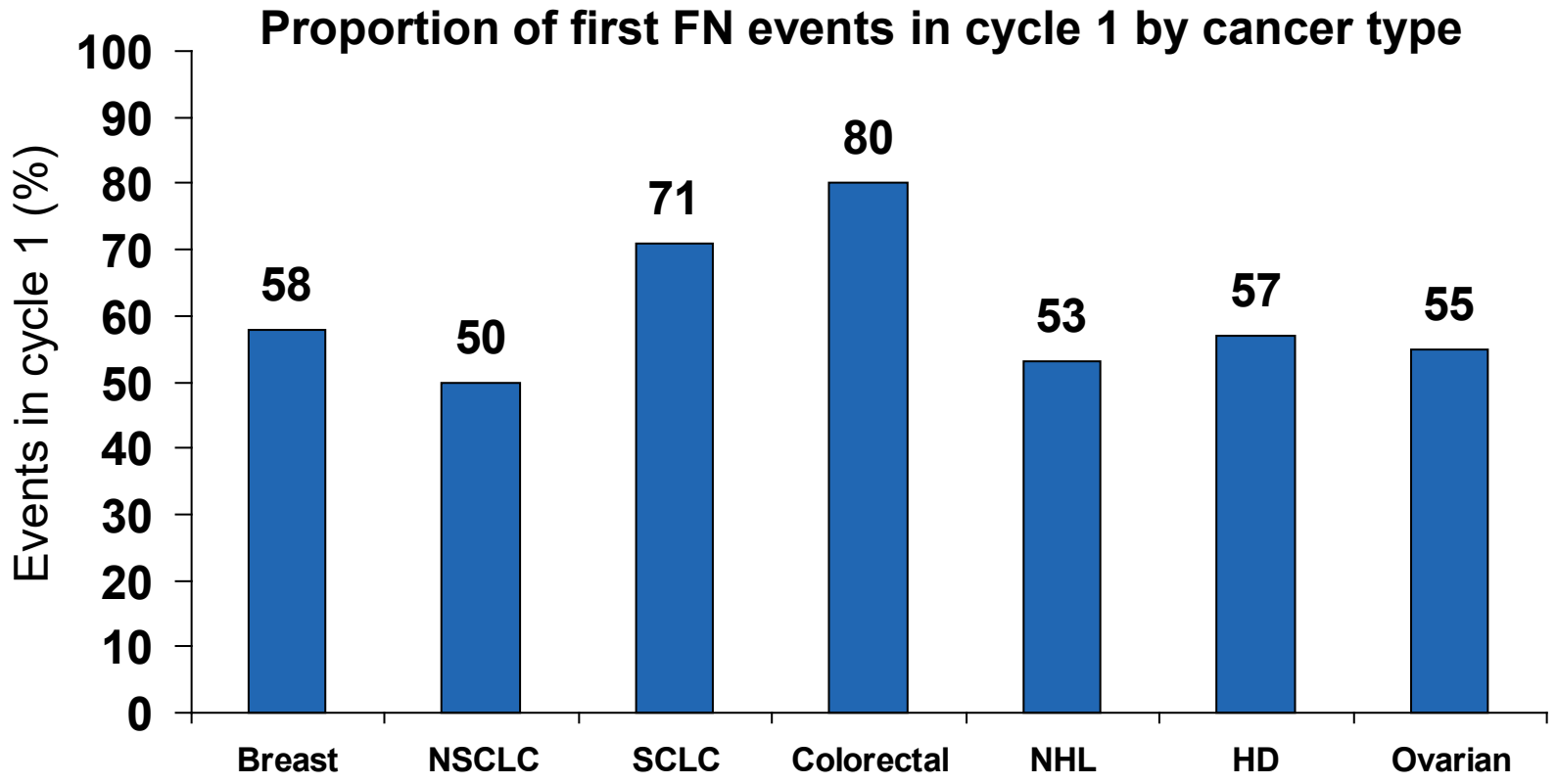
Most initial FN events occur during the first cycle of chemotherapy

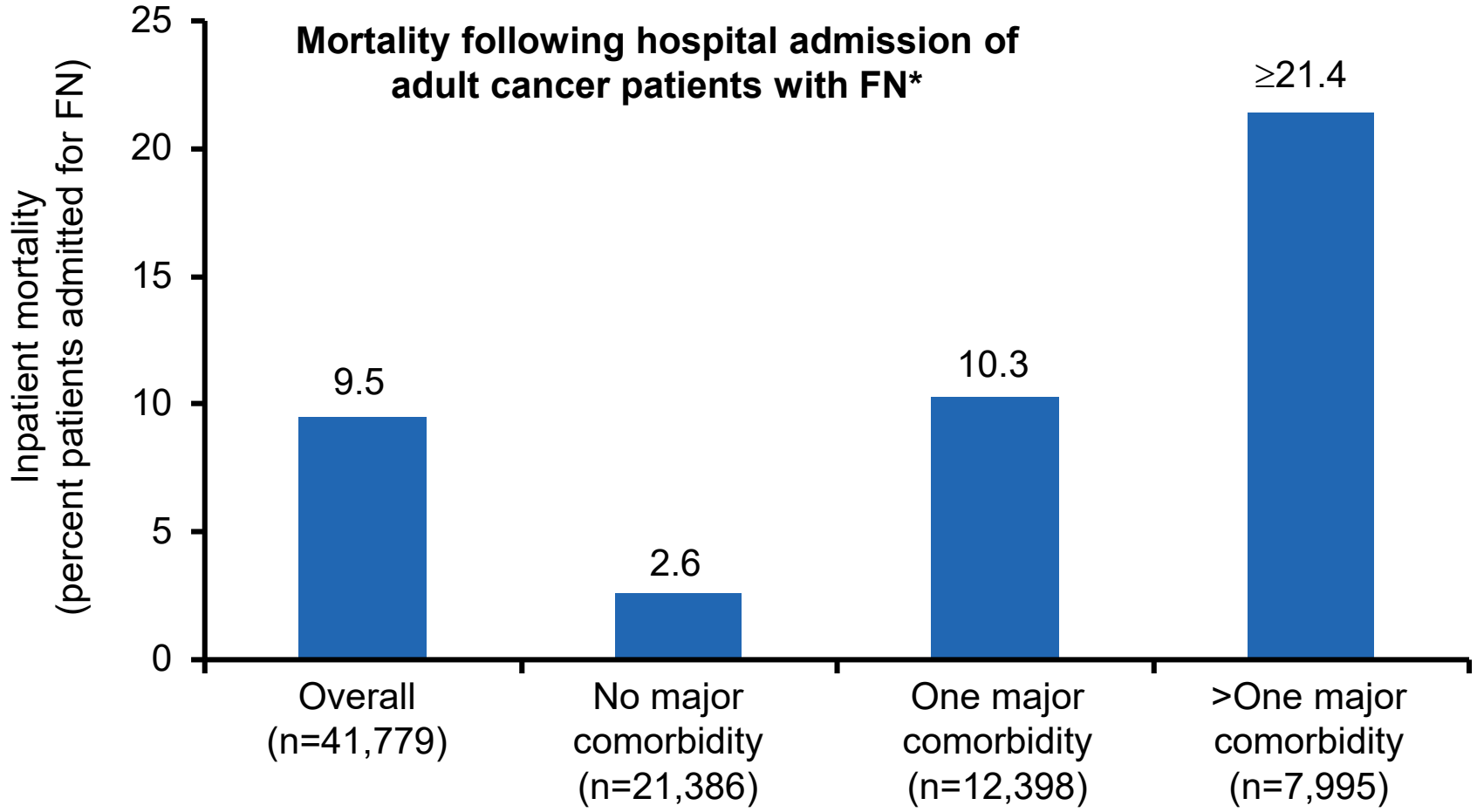


- FN events documented in 287/2692 (10.7%) of adult cancer patients during the 1st three cycles of chemotherapy

NSCLC – non-small cell lung cancer;
SCLC – small cell lung cancer;
NHL - non-Hodgkin's lymphoma;
HD – Hodgkin disease

Adapted from Crawford J et al. *JNCCN* 2008;6:109–118

Death as a result of FN hospitalization

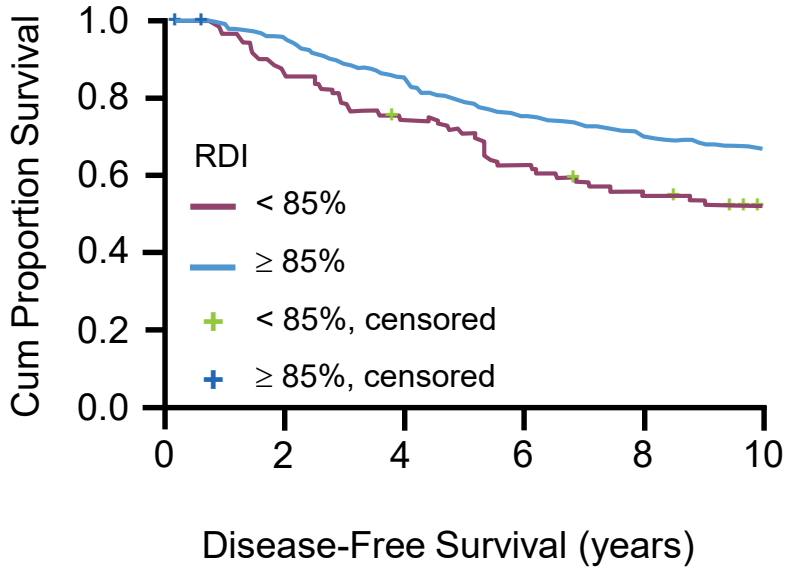


*Data based on a single admission per patient

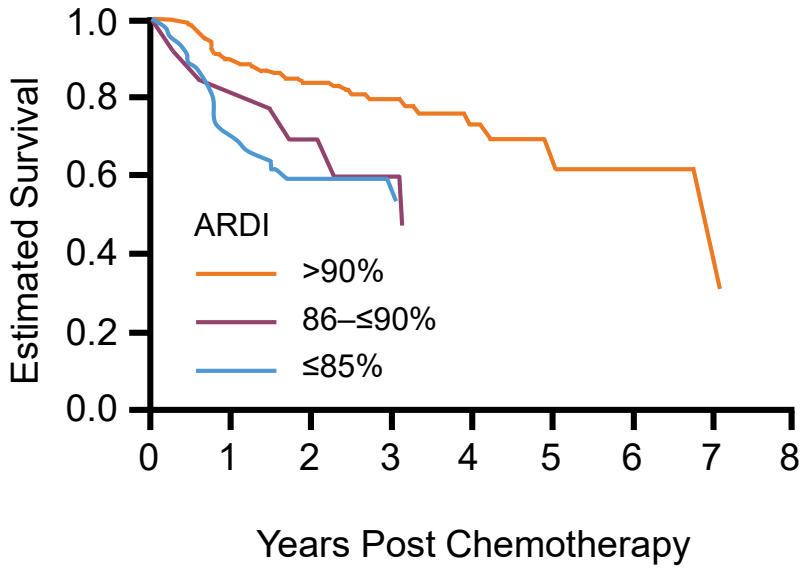
Kuderer NM et al. *Cancer* 2006;106:2258–2266

Clinical consequences of neutropenia and febrile neutropenia

- Reduction in relative dose intensity (RDI) of chemotherapy is associated with survival



- **Reduced RDI was associated with lower OS in ESBC receiving anthracycline-containing chemotherapy[1]**



- **Reduced RDI was associated with lower OS in patients with DLBCL receiving CHOP-21 chemotherapy[2]**

OS, overall survival; ARDI, average relative dose intensity

1 Chirivella I, et al. *Breast Cancer Res Treat* 2009;114(3):479-484
2 Bosly A, et al. *Ann Hematol* 2008; 87:277–283.

Strategies For Management of Chemotherapy-Induced Neutropenia

- **Prevention (Primary vs. Secondary)**
  - Chemotherapy dose reduction/delay
  - Myeloid growth factors
    - G-CSF (filgrastim, tbo-filgrastim, filgrastim-sndz) [Cat. 1]
    - Pegfilgrastim [Cat. 1]
    - GM-CSF (sargramostim)

- **Antibiotics**

- **Treatment**
  - Observation if afebrile
  - Antibiotics
  - Myeloid growth factors

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NCCN Guidelines Version 2.2018
Myeloid Growth Factors

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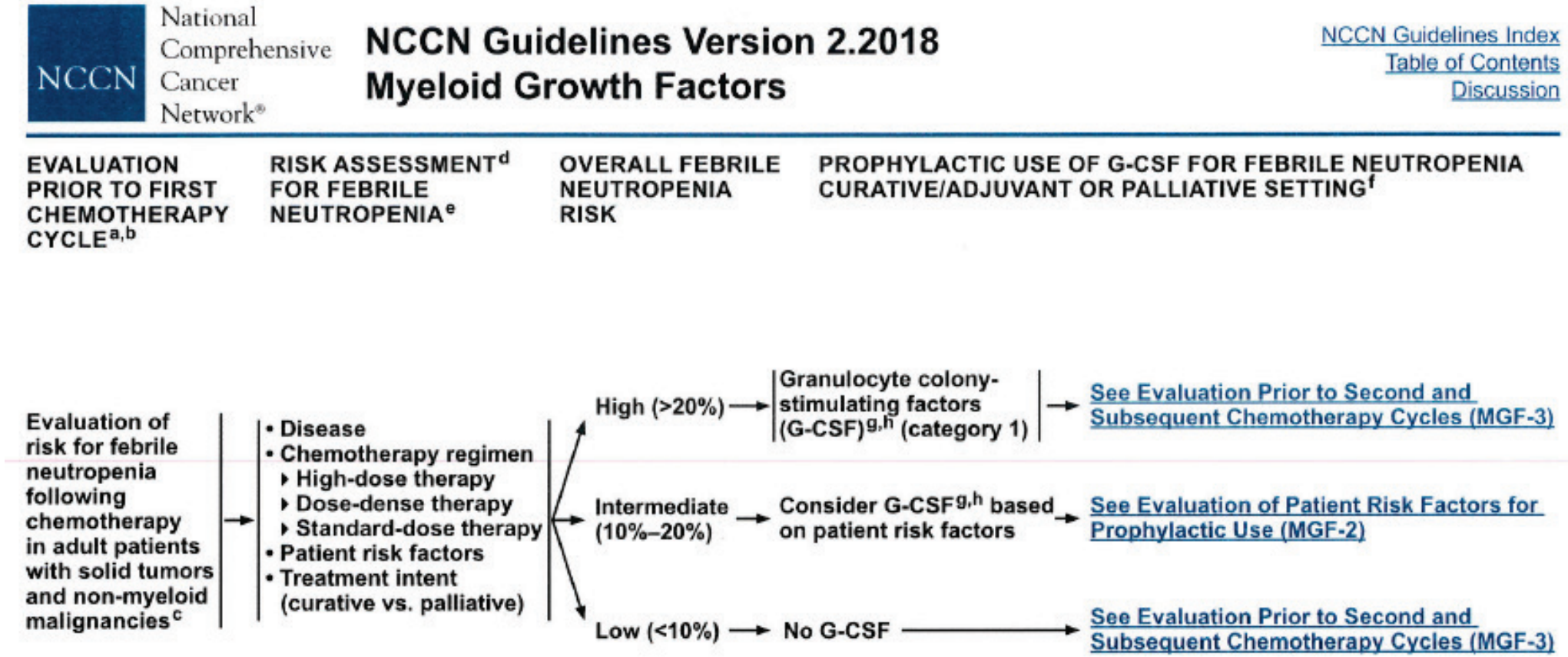


TOXICITY RISKS WITH MYELOID GROWTH FACTORS

Filgrastim and Derivative Products Including Pegfilgrastim[a,b,c]

- Warnings
  - Allergic reactions
    - Skin: rash, urticaria, facial edema
    - Respiratory: wheezing, dyspnea
    - Cardiovascular: hypotension, tachycardia, anaphylaxis
  - Bleomycin-containing regimens: pulmonary toxicity[d]
  - Splenic rupture[d]
  - Acute respiratory distress syndrome
  - Alveolar hemorrhage and hemoptysis
  - Sickle cell crises (only in patients with sickle cell disease)
  - MDS and AML[e]
- Precautions
  - Cutaneous vasculitis
  - Immunogenicity
- Adverse reactions
  - Bone pain

Sargramostim[a,c]

- Warnings
  - Fluid retention: edema, capillary leak syndrome, pleural and/or pericardial effusion
  - Respiratory symptoms: Sequestration of granulocytes in pulmonary circulation, dyspnea
  - Cardiovascular symptoms: Occasional transient supraventricular arrhythmia. Use with caution in patients with preexisting cardiac disease.
  - Renal and hepatic dysfunction: Elevation of serum creatinine or bilirubin and hepatic enzymes. Monitor patients who display renal or hepatic dysfunction prior to initiation of treatment.
- Adverse events occurring in >10% of patients receiving sargramostim in controlled clinical trials and reported in a higher frequency than placebo
  - AML - fever, skin reactions, metabolic disturbances, nausea, vomiting, weight loss, edema, anorexia
  - Autologous hematopoietic cell transplant or peripheral blood progenitor cell transplant - asthenia, malaise, diarrhea, rash, peripheral edema, urinary tract disorder
  - Allogeneic hematopoietic cell transplant or peripheral blood progenitor cell transplant - abdominal pain, chills, chest pain, diarrhea, nausea, vomiting, hematemesis, dysphagia, GI hemorrhage, pruritus, bone pain, arthralgia, eye hemorrhage, hypertension, tachycardia, bilirubinemia, hyperglycemia, increased creatinine, hypomagnesemia, edema, pharyngitis, epistaxis, dyspnea, insomnia, anxiety, high blood urea nitrogen (BUN), and high cholesterol

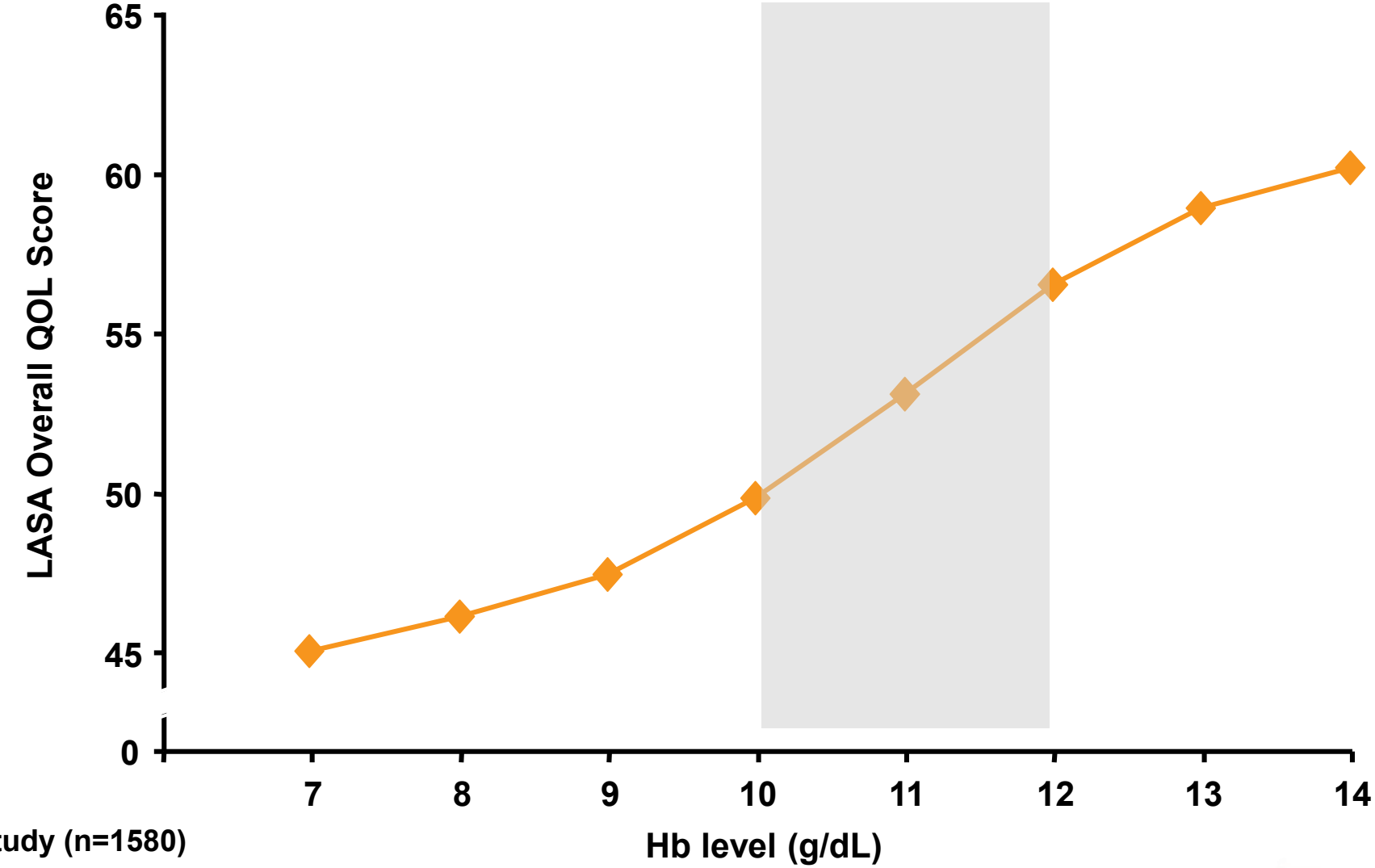
[a]See full prescribing information for specific product information.
[b]Not all of the toxicities listed have been seen with each preparation, but similar toxicities are expected with filgrastim and pegfilgrastim.
[c]The toxicities listed are from the prescribing information and are based on studies from different patient populations. For filgrastim and derivative products, the toxicities are based on non-myeloid malignancies. For sargramostim, the toxicities are based primarily on studies from leukemia and transplant patients, and the listed toxicities may reflect intravenous route of administration and may differ from those of subcutaneous administration.
[d]See Discussion for details.
[e]Lyman et al reported an increase in absolute and relative risk of AML/MDS of 0.41% and 1.92, respectively, related to G-CSF. Overall mortality was decreased. See Discussion for details and reference.

Note: All recommendations are category 2A unless otherwise indicated.
Clinical Trials: NCCN believes that the best management of any patient with cancer is in a clinical trial. Participation in clinical trials is especially encouraged.

Anemia in the Cancer Patient

- Anemia is common in the cancer patient secondary to both cancer and myelosuppressive chemotherapy
- Symptoms of anemia include fatigue, shortness of breath, dizziness, pale skin, rapid heart rate, and impaired mental function
- In patients with cardiovascular disease and other conditions, anemia can lead to myocardial infarction, arrhythmias or stroke

Higher Hb is Associated with Higher HRQoL



1 study (n=1580)

Crawford J, et al. *Cancer*. 2002;95(4):888-895.

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Cancer- and Chemotherapy-Induced Anemia

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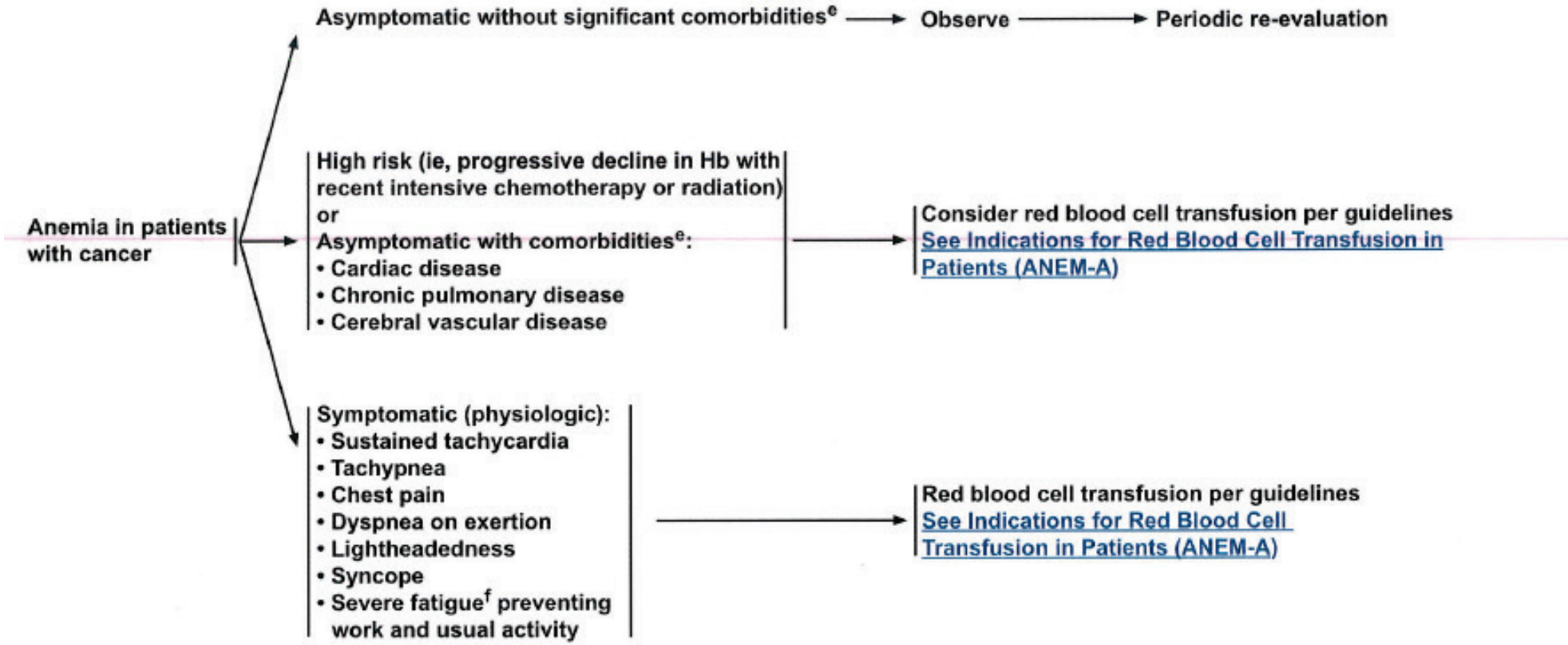
RISK ASSESSMENT AND INDICATIONS FOR INITIAL TRANSFUSION IN ACUTE SETTING



See Comparison of Risks and Goals of ESA Use Versus Red Blood Cell Transfusion (ANEM-3)

See Special Categories in Considering ESA Use (ANEM-4)

COMPARISON OF RISKS AND GOALS OF ESA USE VERSUS RED BLOOD CELL TRANSFUSION[g]

Discuss the following risks and goals with patients when considering anemia treatment options:

	ESA in the Cancer Setting	Red Blood Cell Transfusion
Risks	• Increased thrombotic events • Possible decreased survival • Time to tumor progression shortened	• Transfusion reactions (eg, hemolytic, febrile, non-hemolytic, lung injury) • Transfusion-associated circulatory overload (TACO) • Virus transmission (eg, hepatitis, HIV) • Bacterial contamination • Iron overload • Increased thrombotic events • Possible decreased survival • Alloimmunization • Increased risk of poor response to future platelet transfusions due to HLA immunization
Goals	• Transfusion avoidance • Gradual improvement in anemia-related symptoms	• Rapid increase of Hb and hematocrit levels • Rapid improvement in anemia-related symptoms

See Erythropoietic Therapy - Dosing, Titration, and Adverse Effects (ANEM-B)

When considering ESAs:

- Discuss the risks of ESAs with patients including the potential for tumor growth, death, blood clots, and serious heart problems.
- Refer patients to the following medication guides for more information on the benefits and risk of ESAs: Epoetin Alfa Medication Guide, Epoetin Alfa-epbx Medication Guide and Darbepoetin Alfa Medication Guide

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SPECIAL CATEGORIES IN CONSIDERING ESA USE

Cancer and chronic kidney disease (moderate to severe) → **Consider ESAs by FDA dosing/dosing adjustments[j,k,l]** →

Patient undergoing palliative treatment[h] →

Remainder of patients with anemia on myelosuppressive chemotherapy without other identifiable cause of anemia[h] →

Consider based on patient preferences:
- **ESAs by FDA dosing/dosing adjustments[j,k]** →
  or
- **Red blood cell transfusion per guidelines (See ANEM-A)**
  or
- **Clinical trial**

→ **See Evaluation of Iron Deficiency (ANEM-5)**

Select patients who refuse blood transfusions → **Consider ESAs by FDA dosing/dosing adjustments[j,k]**
See Management of Patients Who Refuse Blood Transfusions (ANEM-D) →

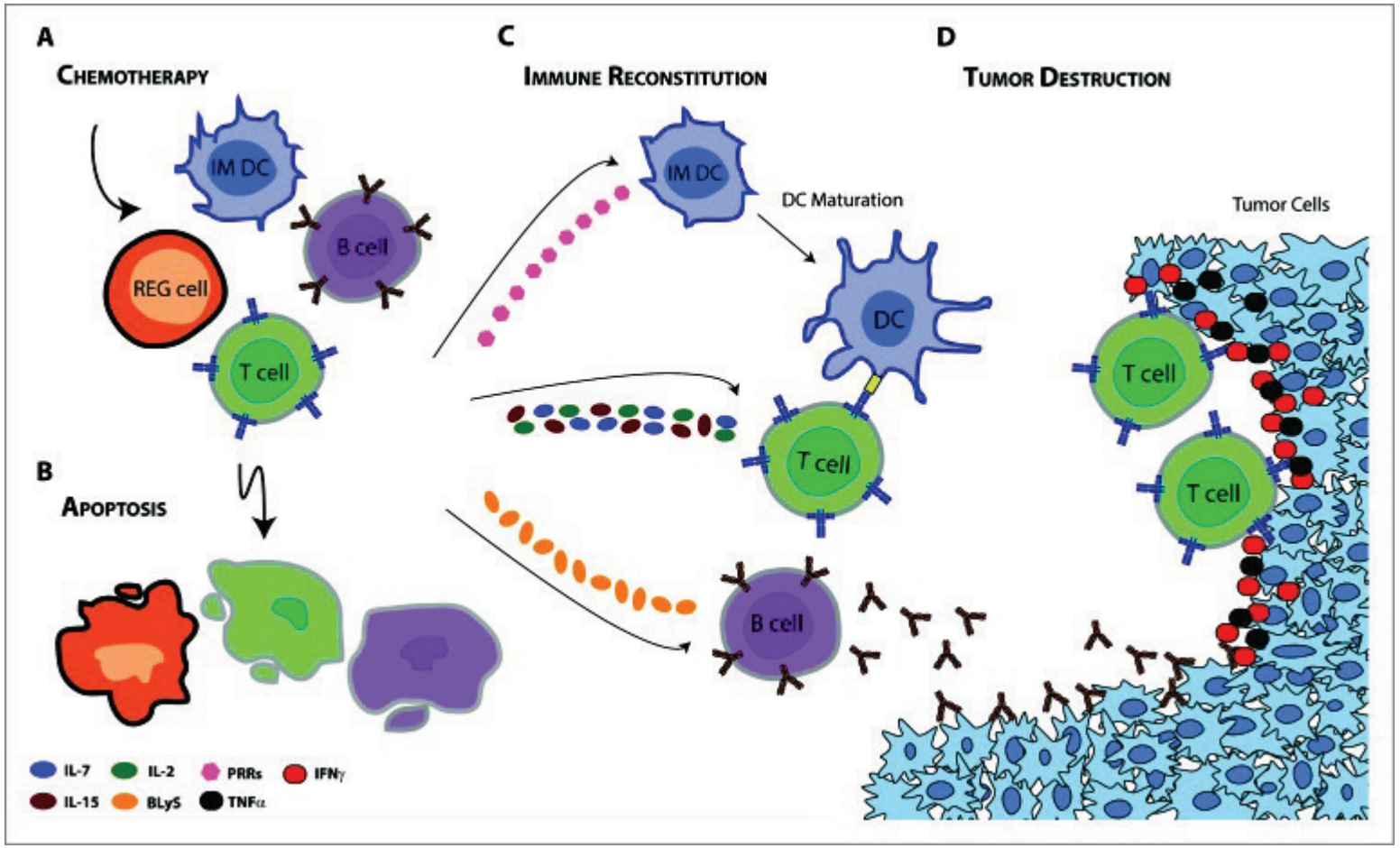
- **Patients with cancer not receiving therapy**
- **Patients receiving non-myelosuppressive therapy**
- **Patients receiving myelosuppressive chemotherapy with curative intent[i]**
  (Examples of cancers for which there is therapy with curative intent: Early-stage breast cancer, Hodgkin lymphoma, non-Hodgkin's lymphomas, testicular cancer, early-stage non-small cell lung cancer, small cell lung cancer, etc.)

→ **There is not enough evidence to support ESA use in these patient populations; therefore, ESAs are not recommended at this time**

Thrombocytopenia in the Cancer Patient

- Thrombocytopenia (low platelet count) is less prevalent than neutropenia or anemia in the cancer patient
- It occurs more commonly in highly myelosuppressive regimens, and in patients with myeloid malignancies and those that received prior chemotherapy
- Thrombocytopenia increases the risk of bleeding and may limit chemotherapy dose and frequency
- Thrombopoietic agents are available for treatment of benign platelet disorders, but they have not been shown to be clinically effective in reducing thrombocytopenia in cancer patients
- Platelet transfusion is the standard treatment intervention

Chemotherapy, Lymphopenia and The Immune Response



Sanchez-Perez L. Oncoimmunology. 2014; 3(7): e944054.

Key Points

- Chemotherapy is the most common systemic treatment in both the curative and palliative settings for cancer patients
- Myelosuppression from chemotherapy limits its effectiveness and places cancer patients at significant risk of neutropenia and infections, anemia and cardiovascular complications, and thrombocytopenia and bleeding
- The consequences of lymphopenia are not well studied, but may contribute to certain types of infection and impaired antitumor responses, particularly in the era of immuno-oncology
- Currently available treatments for myelosuppression include growth factors resulting in bone marrow stimulation that may have adverse clinical effects, and/or transfusion with other resultant risks
- Alternative strategies are needed

TRILACICLIB DEVELOPMENT UPDATE

Raj Malik, M.D.
Chief Medical Officer and Senior Vice President, R&D

Hematopoietic stem and progenitor cells (HSPCs)

Key takeaways

1

Substantial unmet need

- ~1 million patients in U.S. receive chemotherapy each year
- chemo to remain a cornerstone of cancer treatment
- myelosuppression still prevalent

2

Phase 2 program showed benefits across different indications, lines of therapy and chemotherapy regimens

- myelopreservation in SCLC
- PFS in mTNBC

3

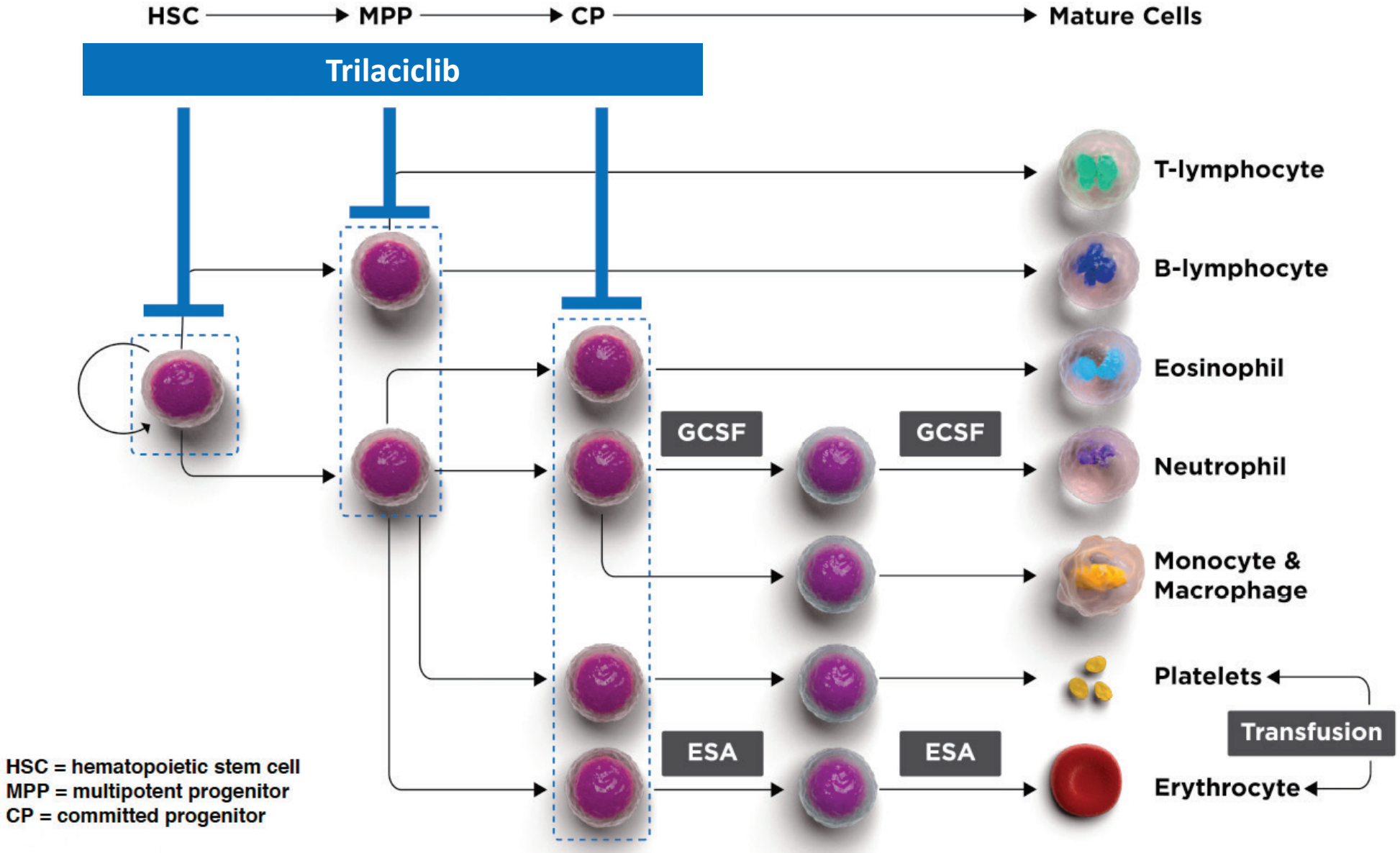
Next steps in trilaciclib development

- meet with U.S. and European regulators
- target initial indication: SCLC
- additional trials initiating in 2H19

Evolution of cancer care with myelosuppressive chemotherapy

ANTI-CANCER THERAPY	1st generation (before growth factors)	2nd generation (after growth factors)	3rd generation (after HSPC protectant added to SOC)
REGIMEN	**Cytotoxic chemotherapy**	**Chemotherapy + growth factors, supportive care**	**Chemotherapy + trilaciclib, OR Chemotherapy + immuno-oncology agents + trilaciclib**
LIMITATIONS AND CHALLENGES	Chemotherapy-induced hematologic and non-hematologic toxicities were dose limiting and thereby limiting of the ability to deliver more efficacious chemotherapy doses. Broad HSPC damage impacts hematopoietic health of the patient	Hematologic dose-limiting toxicity (DLT) is reduced but dose reductions/ delays, hospitalizations, transfusions are not eliminated. Broad HSPC damage impacts hematopoietic health of the patient and use of growth factors leads to HSC exhaustion and reduced immune system function.	Protection of HSPCs; hematologic toxicity is no longer a primary DLT. Fewer supportive care measures including fewer RBC transfusions/ESAs, fewer dose reductions/hospitalizations, and potential for overall improved patient outcomes through improved immune-mediated efficacy due to preserved HSPCs and reduction of HSC exhaustion.

Trilaciclib's MOA provides multi-lineage benefits vs. current lineage-specific interventions

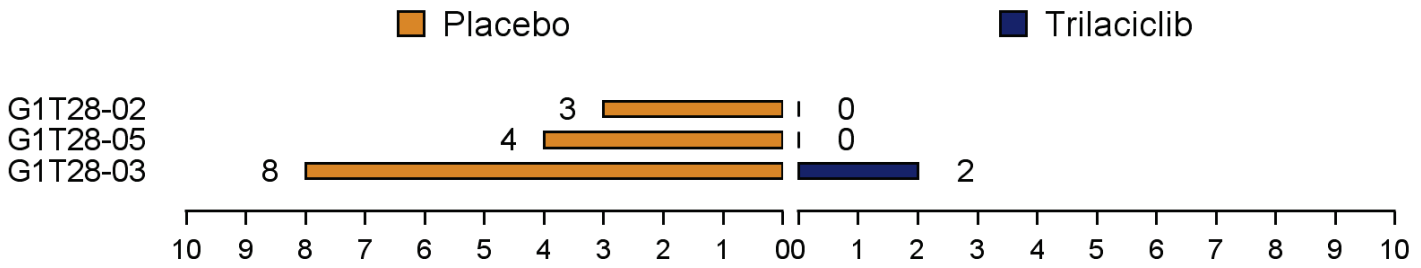


Positive multi-lineage myelopreservation results from three randomized SCLC Phase 2 trials in 2018

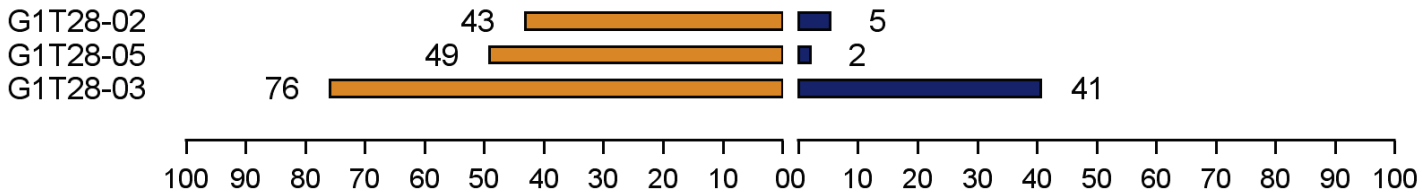
TRIAL/TUMOR TYPE	REGIMEN	TRIAL DESIGN
1st-line Small Cell Lung Cancer (G1T28-02)	+ etoposide/ carboplatin (EP)	• 77 patients, randomized, placebo-controlled, double-blind
1st-line Small Cell Lung Cancer (G1T28-05)	+ EP/Tecentriq®	• 107 patients, randomized, placebo-controlled, double-blind
2nd/3rd-line Small Cell Lung Cancer (G1T28-03)	+ topotecan	• 92 patients, randomized, placebo-controlled, double-blind

Achieved statistical significance on primary endpoints: duration and occurrence of severe neutropenia

Mean duration of severe neutropenia in cycle 1 (days)



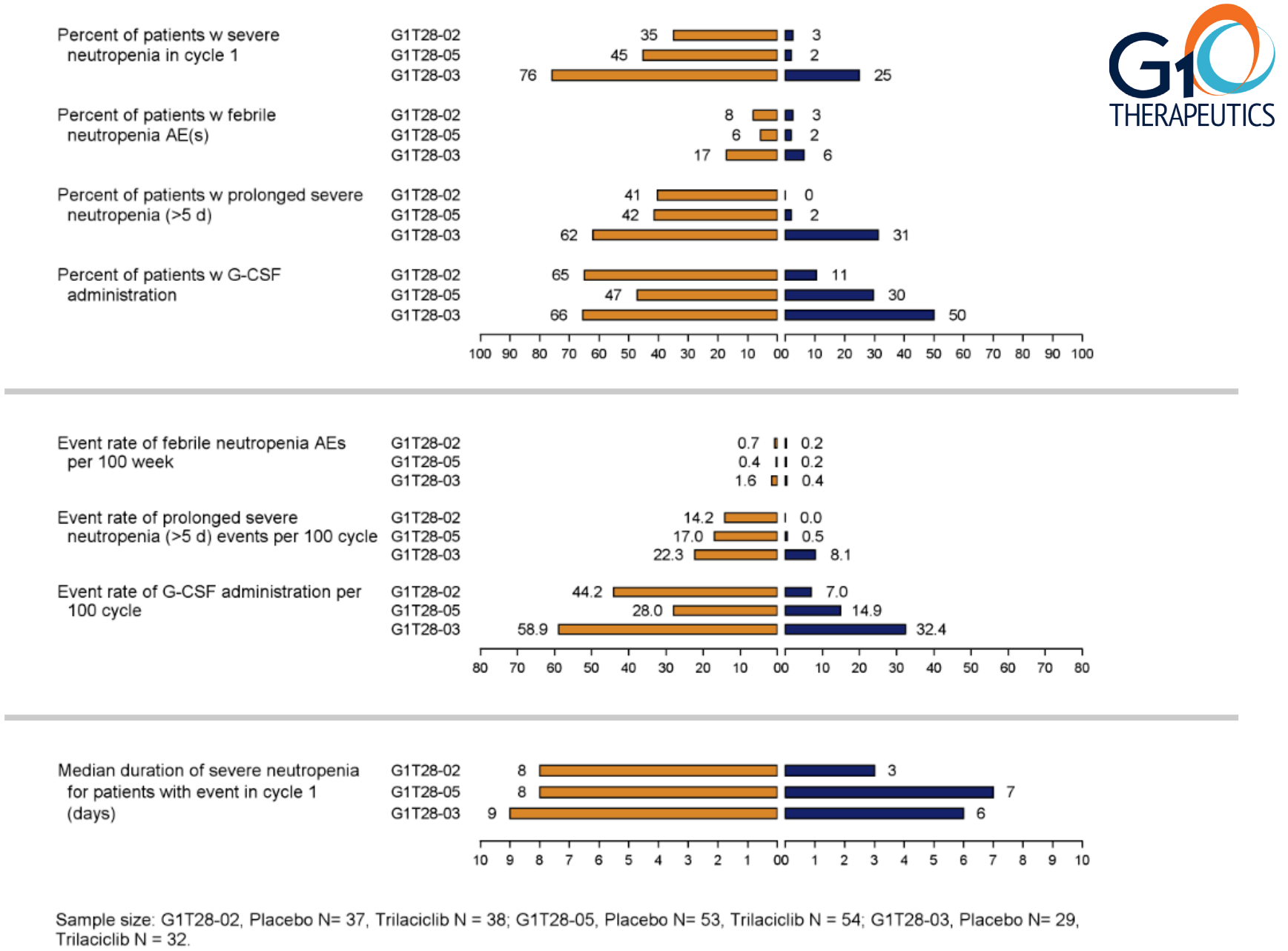
Percent of patients w severe neutropenia



Sample size: G1T28-02, Placebo N= 37, Trilaciclib N = 38; G1T28-05, Placebo N= 53, Trilaciclib N = 54; G1T28-03, Placebo N= 29, Trilaciclib N = 32.

G1T28-02 = 1st-line SCLC trilaciclib/chemo; G1T28-03 = 2nd/3rd-line SCLC trilaciclib/chemo; G1T28-05 = 1st-line SCLC trilaciclib/chemo/Tecentriq®

Neutrophil endpoints favored trilaciclib



Sample size: G1T28-02, Placebo N= 37, Trilaciclib N = 38; G1T28-05, Placebo N= 53, Trilaciclib N = 54; G1T28-03, Placebo N= 29, Trilaciclib N = 32.

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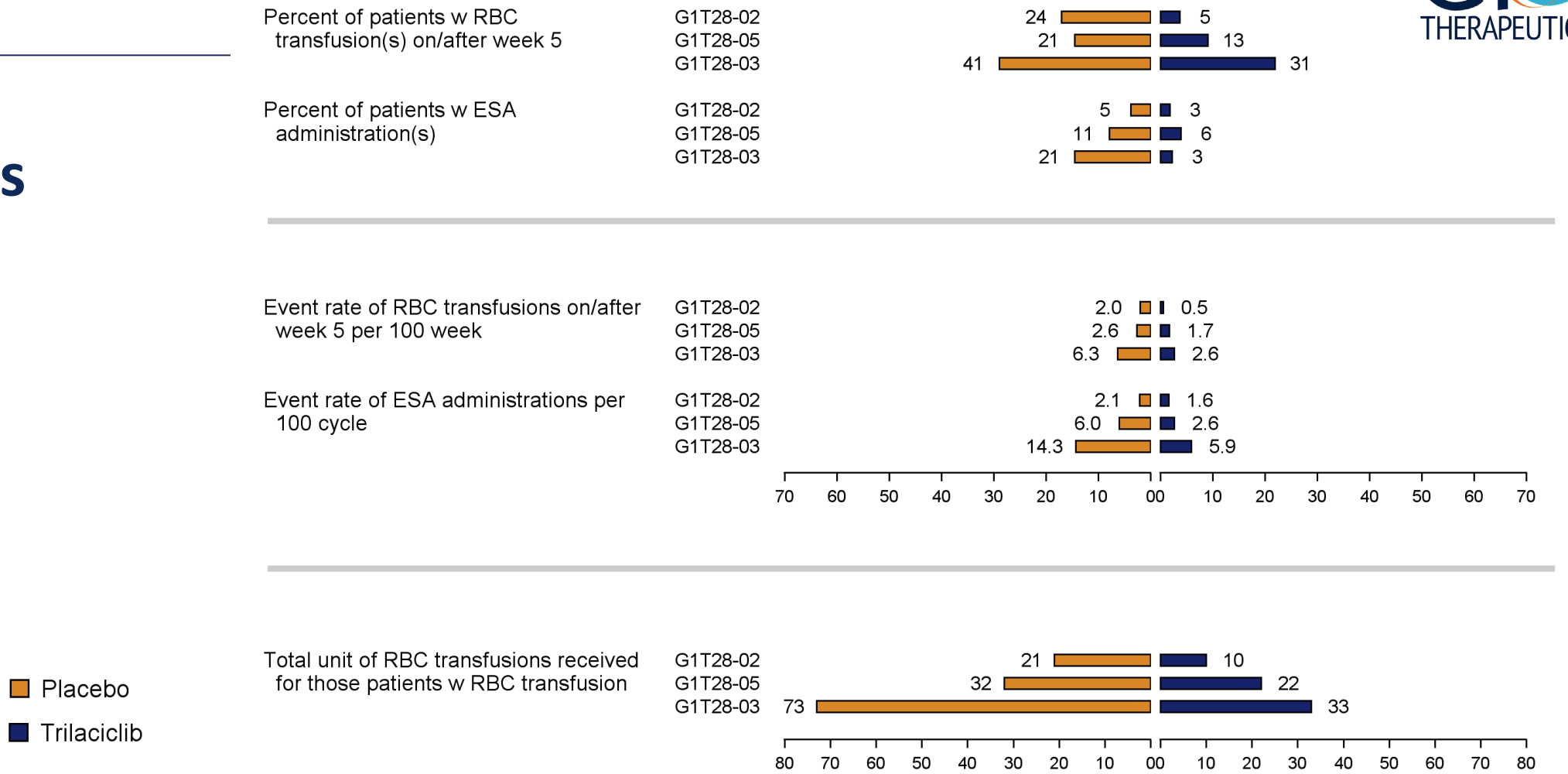
RBC endpoints favored trilaciclib



Sample size: G1T28-02, Placebo N= 37, Trilaciclib N = 38; G1T28-05, Placebo N= 53, Trilaciclib N = 54; G1T28-03, Placebo N= 29, Trilaciclib N = 32.

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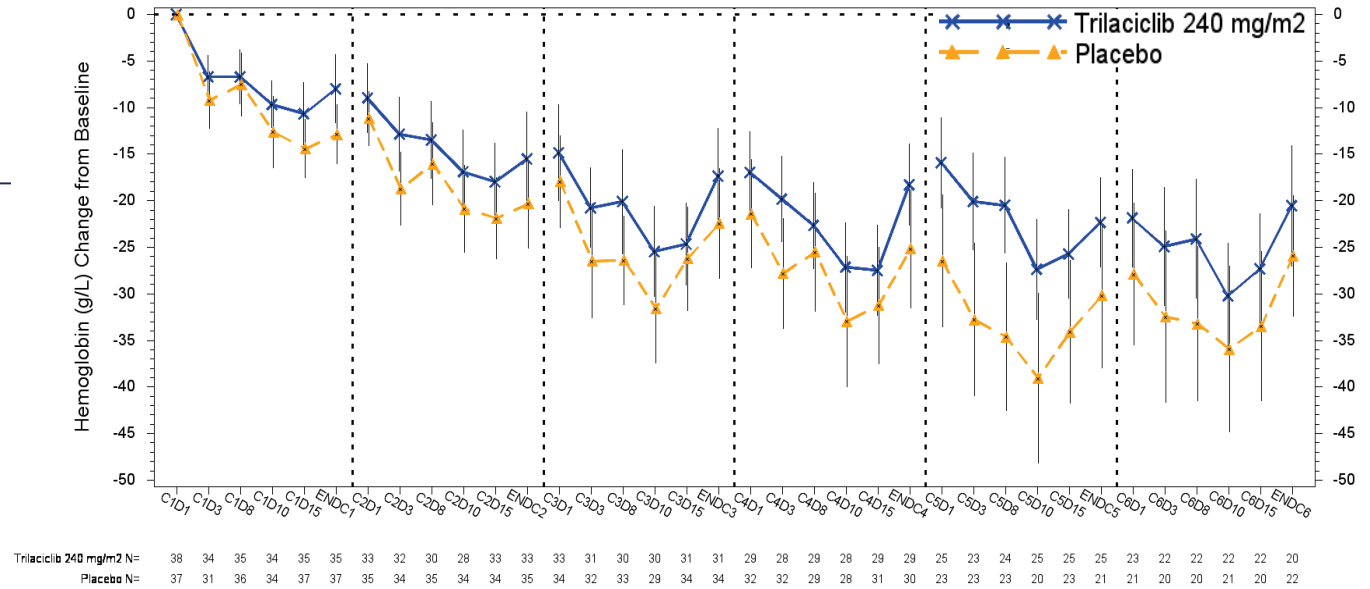
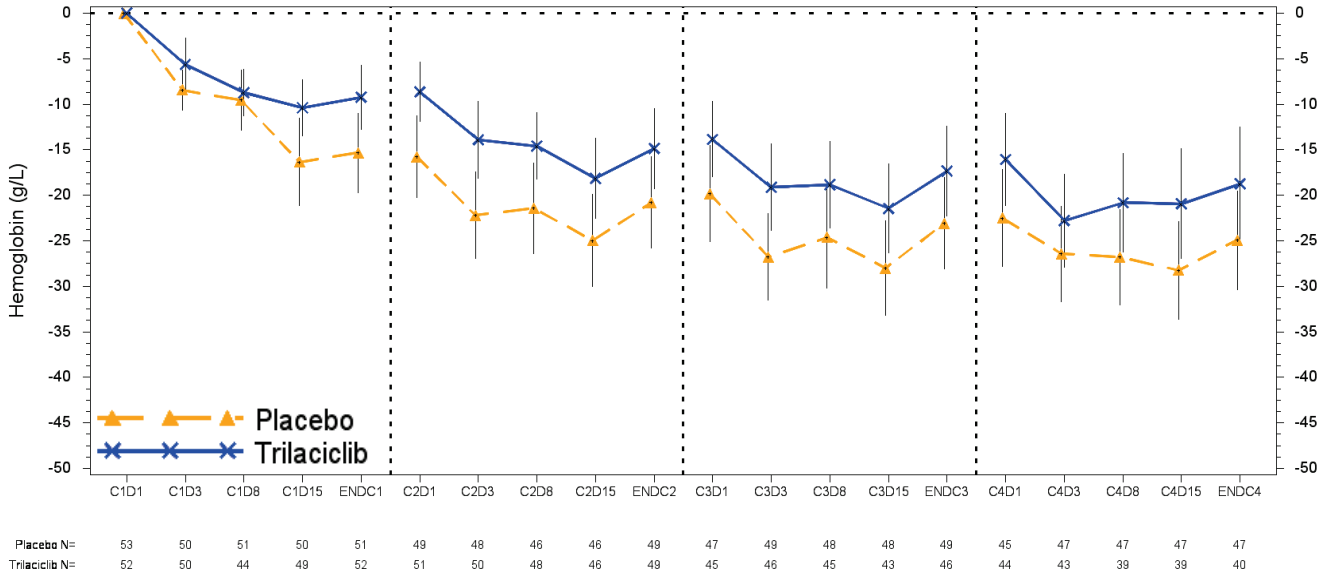
Trilaciclib maintained hemoglobin levels in 1st-line SCLC patients

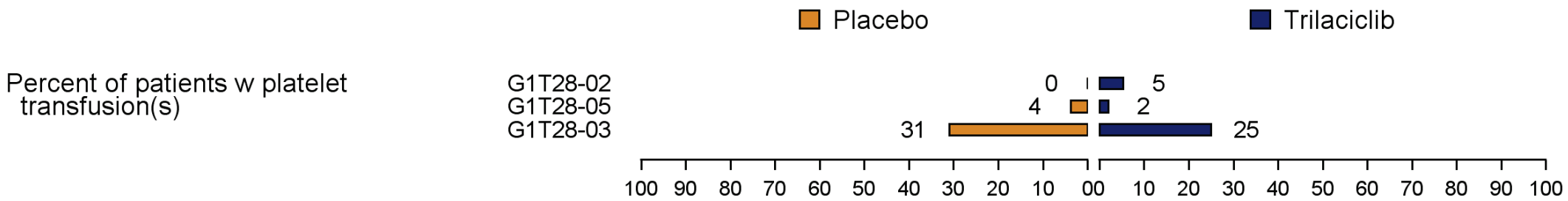


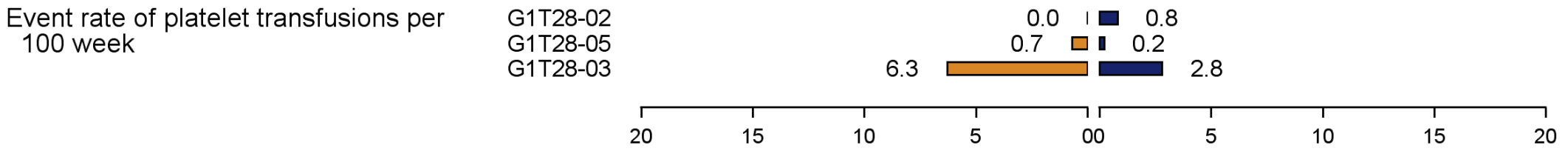
Trila + chemo
Mean change from baseline for hemoglobin over time



Trila + chemo + Tecentriq
Mean change from baseline for hemoglobin over time

Platelet endpoints favored trilaciclib

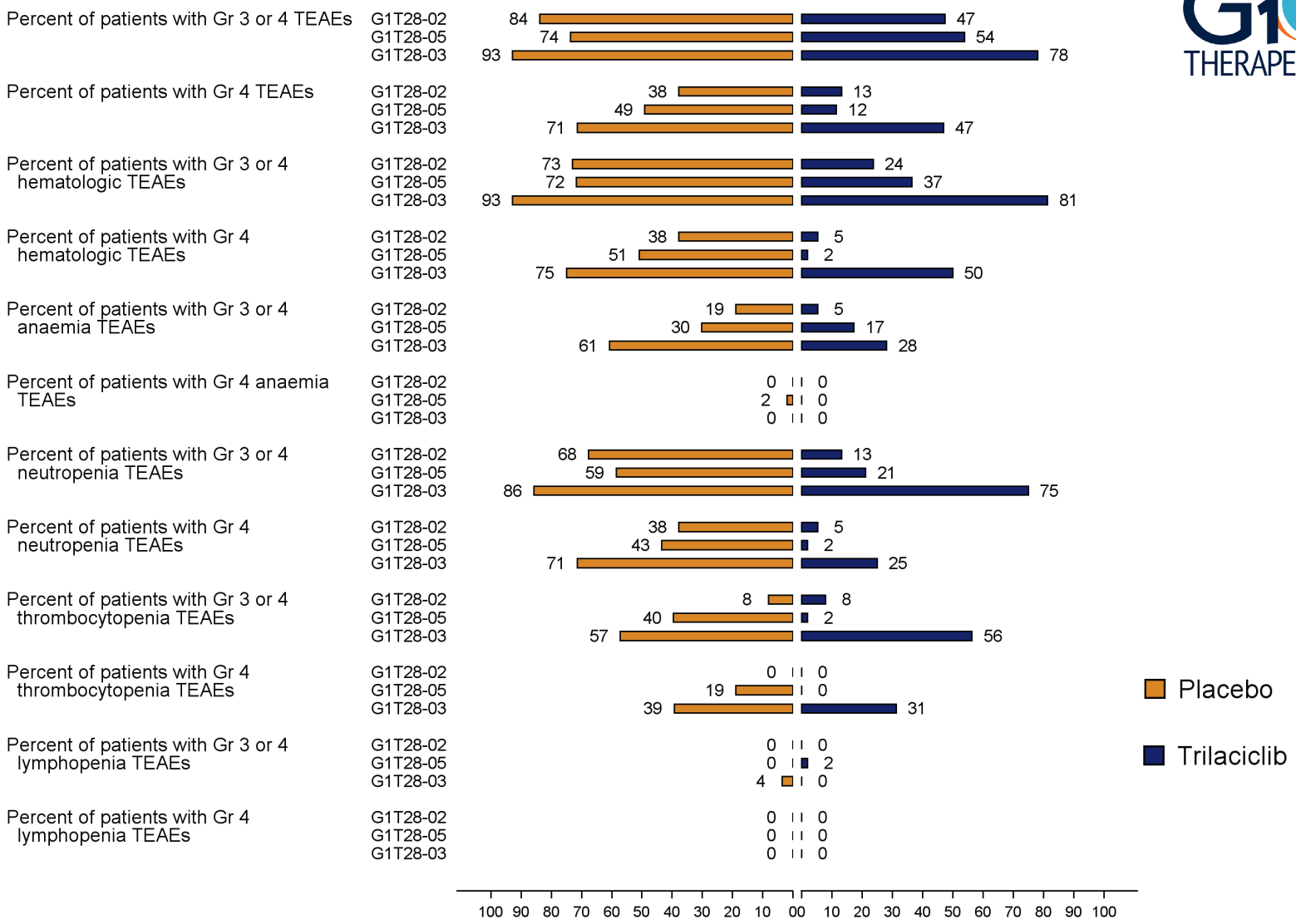




Sample size: G1T28-02, Placebo N= 37, Trilaciclib N = 38; G1T28-05, Placebo N= 53, Trilaciclib N = 54; G1T28-03, Placebo N= 29, Trilaciclib N = 32.

G1T28-02 = 1st-line SCLC trilaciclib/chemo; G1T28-03 = 2nd/3rd-line SCLC trilaciclib/chemo; G1T28-05 = 1st-line SCLC trilaciclib/chemo/Tecentriq®

Trilaciclib consistently improved safety



Sample size: G1T28-02, Placebo N= 37, Trilaciclib N = 38; G1T28-05, Placebo N= 53, Trilaciclib N = 52; G1T28-03, Placebo N= 28, Trilaciclib N = 32.

G1T28-02 = 1st-line SCLC trilaciclib/chemo; G1T28-03 = 2nd/3rd-line SCLC trilaciclib/chemo; G1T28-05 = 1st-line SCLC trilaciclib/chemo/Tecentriq®

Patient reported outcomes (PRO) in SCLC trials

FACT-G, FACT-An and FACT-L were assessed in all three SCLC studies

FACT-G measures health-related quality of life (HRQoL) and includes assessments of:

- physical well being (PWB)
- emotional well being (EWB)
- social well being (SWB)
- functional well being (FWB)

FACT-An is a module that can be added to FACT-G to assess the impact of fatigue and other anemia-related symptoms for patients with cancer

FACT-L is a module that can be added to FACT-G to measure the impact of lung cancer-specific symptoms for patients with cancer

Data collected in the context of randomized, double-blind, placebo-controlled trials; instrument completion rates were high and comparable between arms

Preliminary PRO results show meaningful benefit for trilaciclib across SCLC Phase 2 trials

- Preliminary analyses suggest trilaciclib + SOC demonstrated health-related quality of life (HRQoL) benefits over placebo + SOC during the treatment period in both treatment-naïve and previously treated SCLC patients

- Strongest trends (or statistical significance) favoring trilaciclib were reported primarily in functional well being (FWB) and physical well being (PWB) domains and some related symptoms, including fatigue and feeling tired

- Anticipate data presentation at medical meeting in 2019

Trilaciclib does not impair efficacy of chemotherapy

	trila/chemo 1st-line			trila/chemo/Tecentriq 1st-line			trila/chemo 2nd/3rd-line		
	placebo N = 37	trila N = 38	HR* or historical RR	placebo N = 53	trila N = 54	HR* or historical RR	placebo N = 29	trila N = 32	HR* or historical RR
Median OS (months)	10.6	10.9	**HR=0.87**	immature			immature		
Median PFS (months)	5.0	6.1	**HR=0.71**	5.4	5.9	**HR=0.78**	4.2	4.2	**HR=0.85**
Overall Response Rate	56.8%	66.7%	52%	63.5%	56.0%	60.2 - 64.4%	23.1%	16.7%	10.1 - 16.9%
Clinical Benefit Rate	86.5%	91.7%	75%	90.4%	96.0%	81.1 - 85.7%	61.5%	60.0%	61.5 - 73.4%

- Lack of efficacy impairment measured by HR ("do no harm")
- Trilaciclib achieves comparable OS and PFS
- Response rates (RR) within historical ranges**

*HR=hazard ratio

** Socinski et al. *J Clin Oncol* 2009; 27: 4787-92; Horn et al. *N Engl J Med* 2018; 379:2220-2229; von Pawel et al. *J Clin Oncol* 2014; 32:4012-4019; Evans et al. *J Thorac Oncol* 2015; 10: 1221–1228

Data cut: December 21, 2018

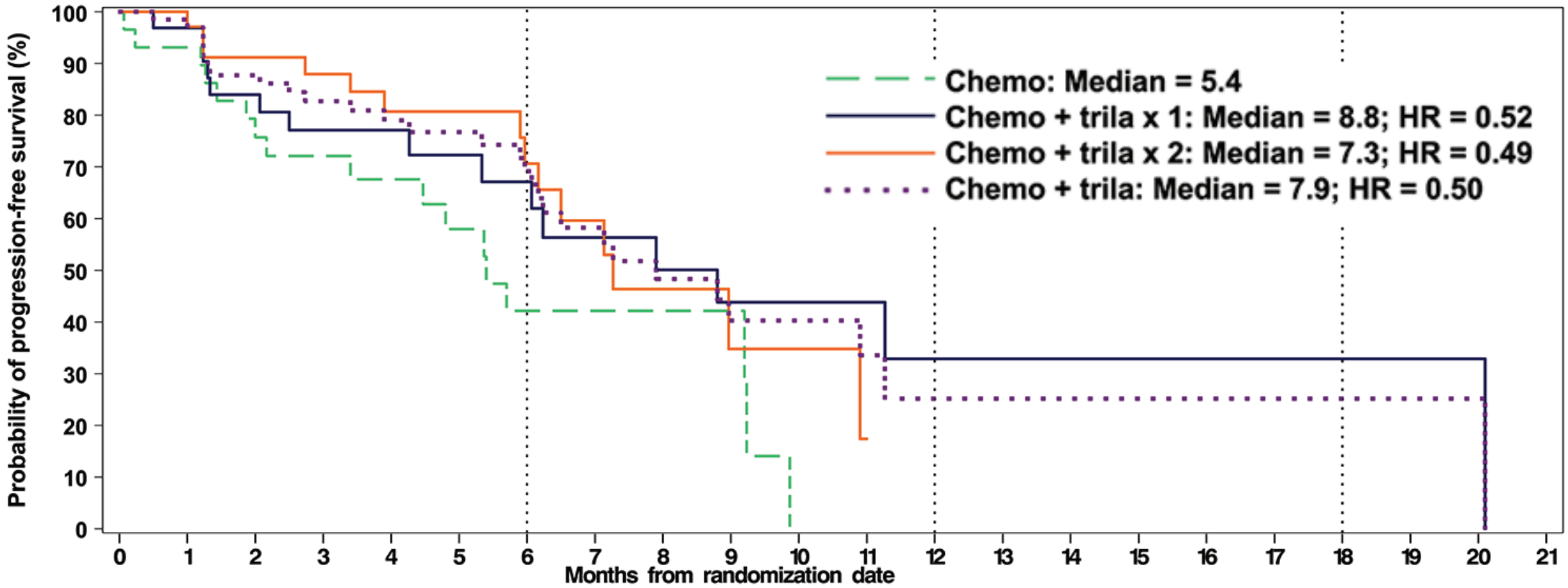
Preliminary results in randomized mTNBC Phase 2 trial demonstrated trilaciclib improved PFS

TRIAL/TUMOR TYPE	REGIMEN	TRIAL DESIGN
Metastatic Triple-Negative Breast Cancer (G1T28-04)	+ gemcitabine/ carboplatin	102 patients, randomized, open-label

- Patients on trilaciclib received more chemotherapy cycles than those in the control arm

- Safety profile consistent with previously reported trials; no trilaciclib-related serious adverse events reported

Preliminary results demonstrated median PFS is longer when trilaciclib is added to chemotherapy



data from SABCS 2018

Development strategy: two pathways to establish trilaciclib as an essential part of chemo and chemo/IO treatment

Establish trilaciclib as first-in-class myelopreservation therapy

Label expansion trials

Fluoropyrimidine GI trial
e.g., colorectal, gastro-esophageal

Taxane basket trial
e.g., NSCLC, ovarian, breast

SCLC
Three randomized Phase 2 trials
demonstrated multi-lineage myelopreservation benefits

MYELOPRESERVATION

Broad trilaciclib label across chemo +/- IO regimens and across tumor types

+ SURVIVAL

Demonstrated trilaciclib survival improvements in specific tumors

mTNBC
pending mature Phase 2 data

Chemo/IO combo
pending outcome of trila/Tecentriq trial

Summary and next steps for trilaciclib development

1

Compelling data from four randomized Phase 2 trials

- achieved primary endpoints in SCLC trials
- PRO benefits seen across SCLC trials
- PFS benefit in mTNBC
- well tolerated safety profile in ~300 participants across all trials

2

Meetings with U.S. and European regulators in 1H19 to discuss next steps for trilaciclib development

- update in 2Q19

3

Evaluating anti-tumor efficacy in ongoing SCLC/Tecentriq and mTNBC trials

- additional anti-tumor efficacy development pending mature data

FLORIDA CANCER SPECIALISTS & Research Institute

Myelopreservation and trilaciclib: implications for clinical practice

Lowell L. Hart, MD FACP
Scientific Director of Clinical Research

Disclosures

- Advisory or Consulting: Genentech, Novartis, Lilly, Merck, Pharmacyclics, Nanostring, Guardant Health and G1
- Speakers Bureau: Lilly, Pfizer and Genentech
- Research funding to Florida Cancer Specialists: Novartis, Genentech, Merck, BI and G1

Professional background

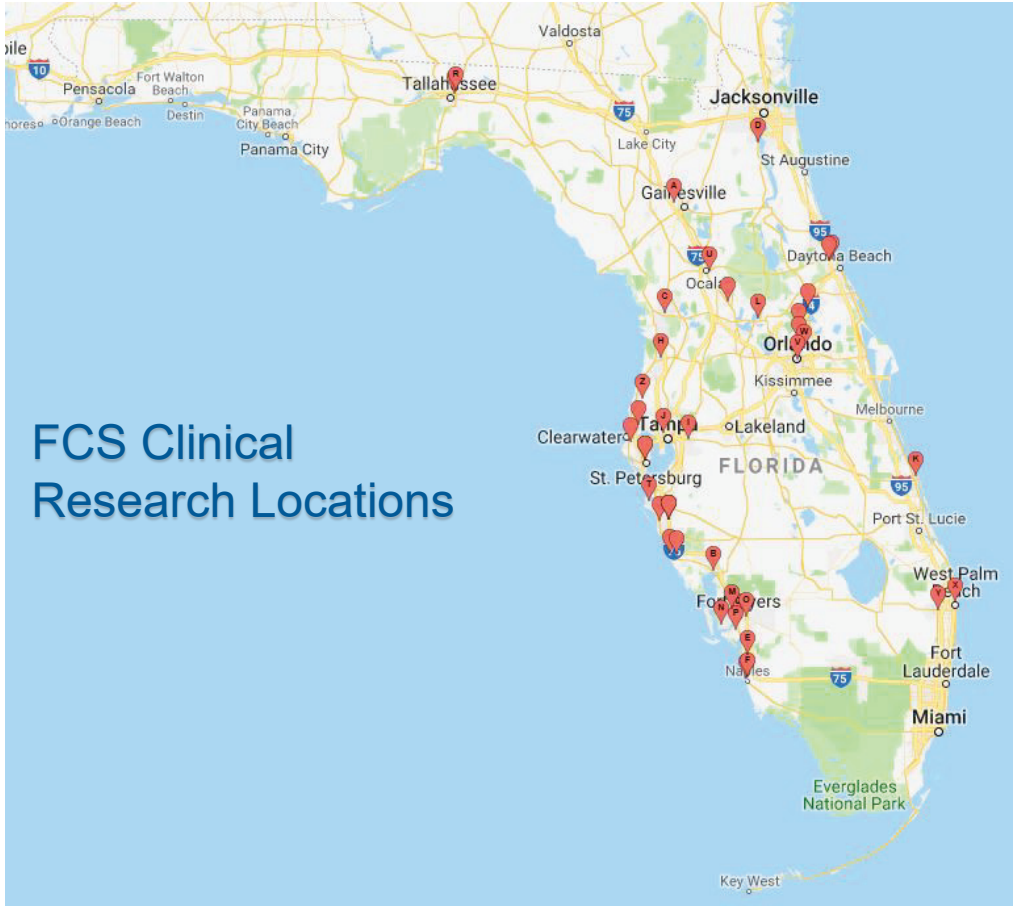
- Training: undergraduate at Columbia University; M.D. from State University of NY Upstate Medical College in Syracuse; residency at University of Miami in internal medicine; fellowship in hematology and oncology at Duke
- Co-founder and Scientific Director of Clinical Research, Florida Cancer Specialists
- Associate Professor of Medicine, Hematology and Oncology, Wake Forest University School of Medicine
- Treating 100+ cancer patients per week
- Investigator on 200+ oncology clinical trials since 2010
  - Clinical investigator on all four Phase 2 trilaciclib trials

Florida Cancer Specialists & Research Institute
Research Department

Our Mission

To be the preferred choice for patients and referral sources for superior, compassionate, community based cancer care in Florida; providing the most advanced cancer treatment, utilizing cutting edge technologies and research, in a setting where patients can be close to home, surrounded and supported by family and friends.

- 71,000 new patients 296,000 total visits in 2018
- 200+ oncologists on staff
- 200+ oncology nurses/nurse practitioners/ physician assistants on staff
- 85 clinic sites



FLORIDA CANCER SPECIALISTS & Research Institute

FDA Oncology Approvals with FCS Participation

In the past 3 years, **the majority of new cancer drugs approved for use in the U.S.** were studied in clinical trials with Florida Cancer Specialists participation.

Treating cancer with chemotherapy in a community setting

- Intake/patient evaluation process
- Treatment options
- Use of rescue interventions for myelosuppression
- Patient experience on current standard of care

Trilaciclib in clinical trial setting

- Florida Cancer Specialists enrolled 45 patients; personally treated 22 patients
- Integration into standard treatment paradigm
- Potential patient benefits
- Patient experience

Key takeaways on trilaciclib

- Improved patient outcomes through proactive patient care
- In clinical trials, reduced use of rescue interventions (G-CSF, transfusions) and need for hospitalizations/unscheduled office visits
- Integrated into existing treatment paradigm – convenient for patient and practice

TRILACICLIB COMMERCIAL STRATEGY

John Demaree
Chief Commercial Officer

Hematopoietic stem and progenitor cells (HSPCs)

Physicians describe the substantial unmet need for patients with SCLC

"If a patient is doing well it means that they are not having too many side effects from treatment. Our goal for incurable cancer is to prolong survival without negatively impacting their quality of life."

–Dr. B., lung cancer specialist

"Being on chemo over the long term is awful for patients; every patient dreads chemo because of what it does to the body."

–Dr. I., breast cancer specialist

"SCLC is a terrible disease for patients; finding something that tolerates well –that's at least preventing a problem instead of reacting to it."

–Dr. W., lung cancer specialist

Key takeaways for trilaciclib

1

There is still substantial unmet need for patients experiencing myelosuppression, despite the availability of rescue interventions like G-CSF, ESAs and transfusions

- no significant innovations for chemotherapy-induced myelosuppression
- chemotherapy will remain a cornerstone of treatment

2

Multi-lineage myelopreservation in SCLC represents an advance for patients and a significant opportunity:
$500M - $1B WW at peak

- physicians see proactive myelopreservation as a better approach and anticipate significant use
- payers value the patient benefits and are willing to pay without significant restrictions

3

Expanding the label across tumors to a broad myelopreservation indication may add >$2B to peak sales

- myelopreservation launch in SCLC meets a substantial unmet need and serves as proof of concept in other tumor types
- efficacy enhancement, OS or PFS data, would provide additional patient benefit and revenue upside, but is not required to generate use in a high % of patients

Patients’ Unmet Need

G1 THERAPEUTICS

Still substantial need for SCLC patients despite availability of rescue interventions like G-CSF, ESAs and transfusions

- With current SOC, a significant percentage of patients still experience severe myelosuppression and the associated consequences:

	Incidence of Grade 3/4[1]	Current Treatments	Current Treatment Unmet Needs
Neutropenia 	23%	G-CSF rescue	~70% bone pain (~25% severe[2]) induced by G-CSFs (severe pain treated with NSAIDs and opioids)
Anemia 	14%	ESA rescue, transfusion rescue	Black Box warning for shortened overall survival and increased risk of tumor progression
Thrombocytopenia 	10%	Transfusion rescue	No options other than transfusions

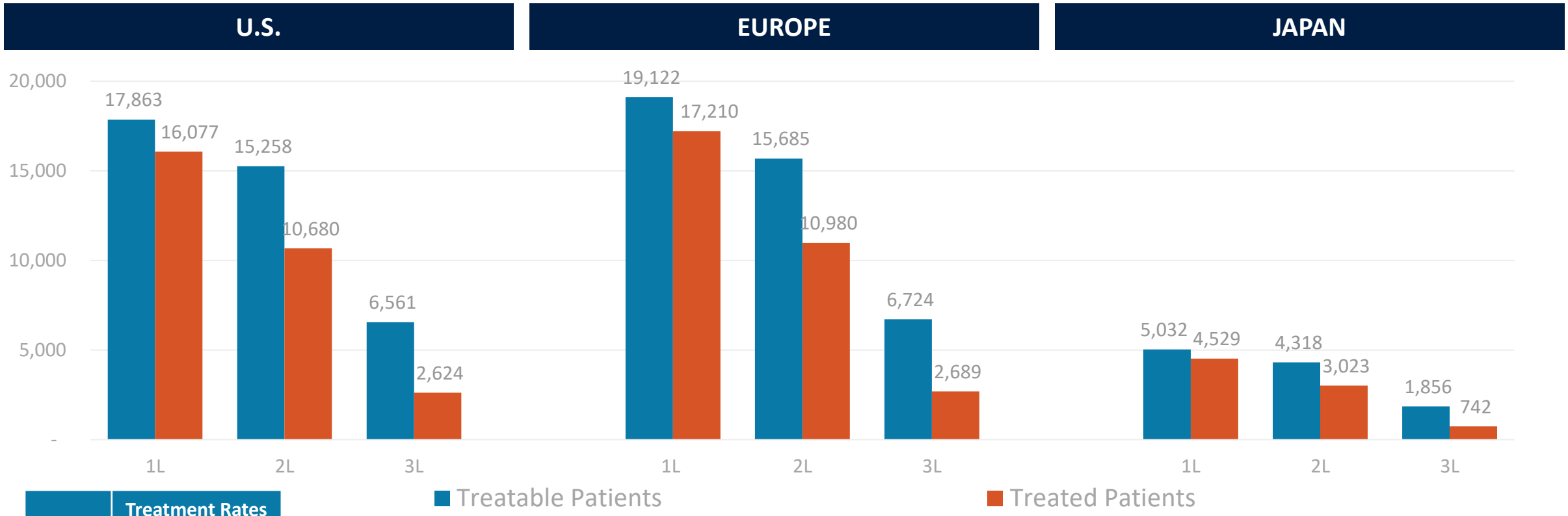
Trilaciclib has the potential to address multi-lineage myelosuppression and reduce the need for rescue therapies and their associated side effects

Sources:
[1]IMpower133 Trial, atezolizumab + E/P arm (n=198), NEJM, 2018
[2]Kirshner et al: Prevention of pegfilgrastim-induced bone pain. JCO, 2012.

Opportunity

There are a significant number of ES-SCLC treated patients that could benefit from multi-lineage myelopreservation

ES-SCLC Patients



	Treatment Rates
1L	90%
2L	70%
3L	40%

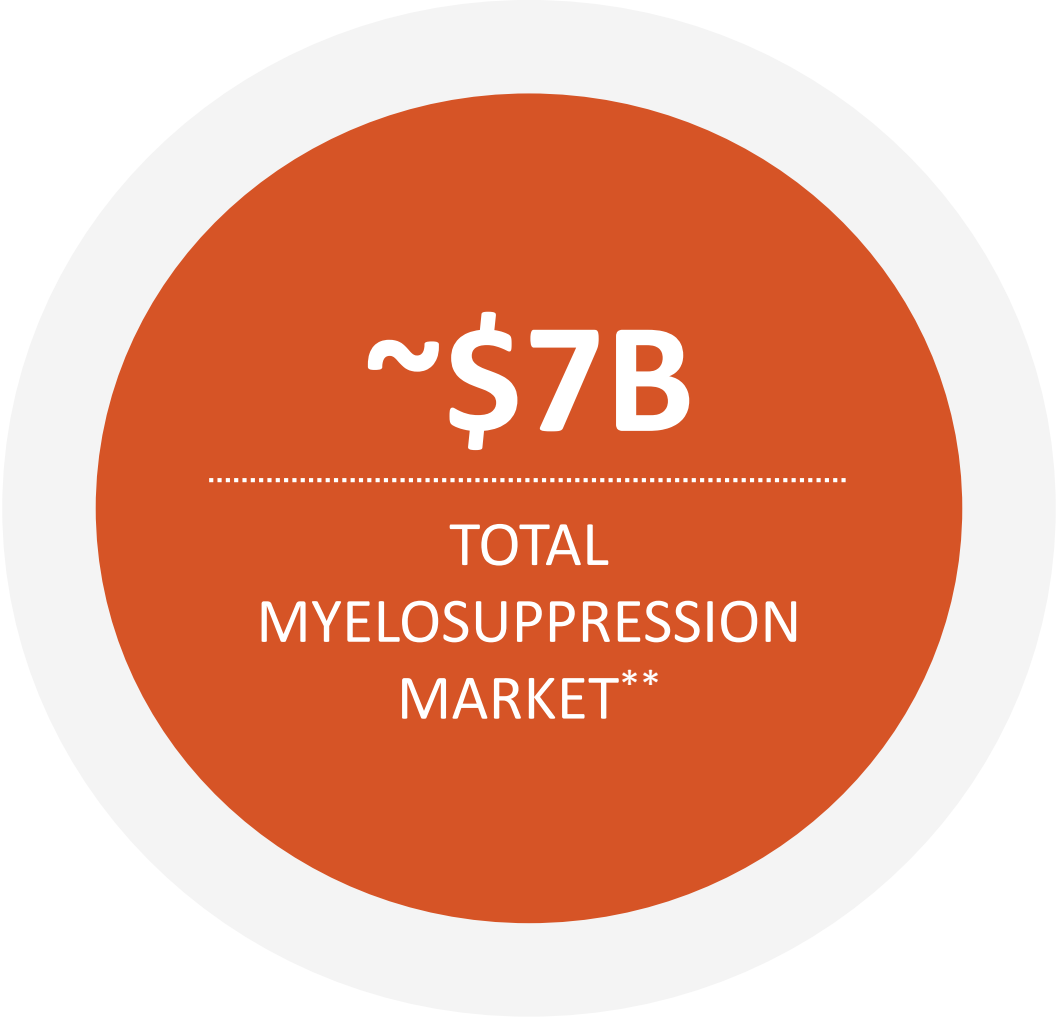
~ 68k treated patients across major markets

1. 2030 estimates, secondary epi source & ZS forecast model

The value of the single-lineage focused, growth factors market for myelosuppression is ~$7B

G-CSF Market	2017 Worldwide Sales*
Neulasta®	$4.5B
Neupogen®/Zarxio®/Granix®	$0.7B
Total Worldwide	**$5.2B**

ESA Market	2017 Worldwide Sales*
Aranesp®	$2.1B
Epogen®	$1.1B
Procrit®	$0.6B
Total Worldwide	**$3.8B**



* Sales from Annual reports; includes sales for all indications.
** Future Market Insights, 2018 valuation of global chemotherapy-induced myelosuppression treatment market.

Market Research & Forecast

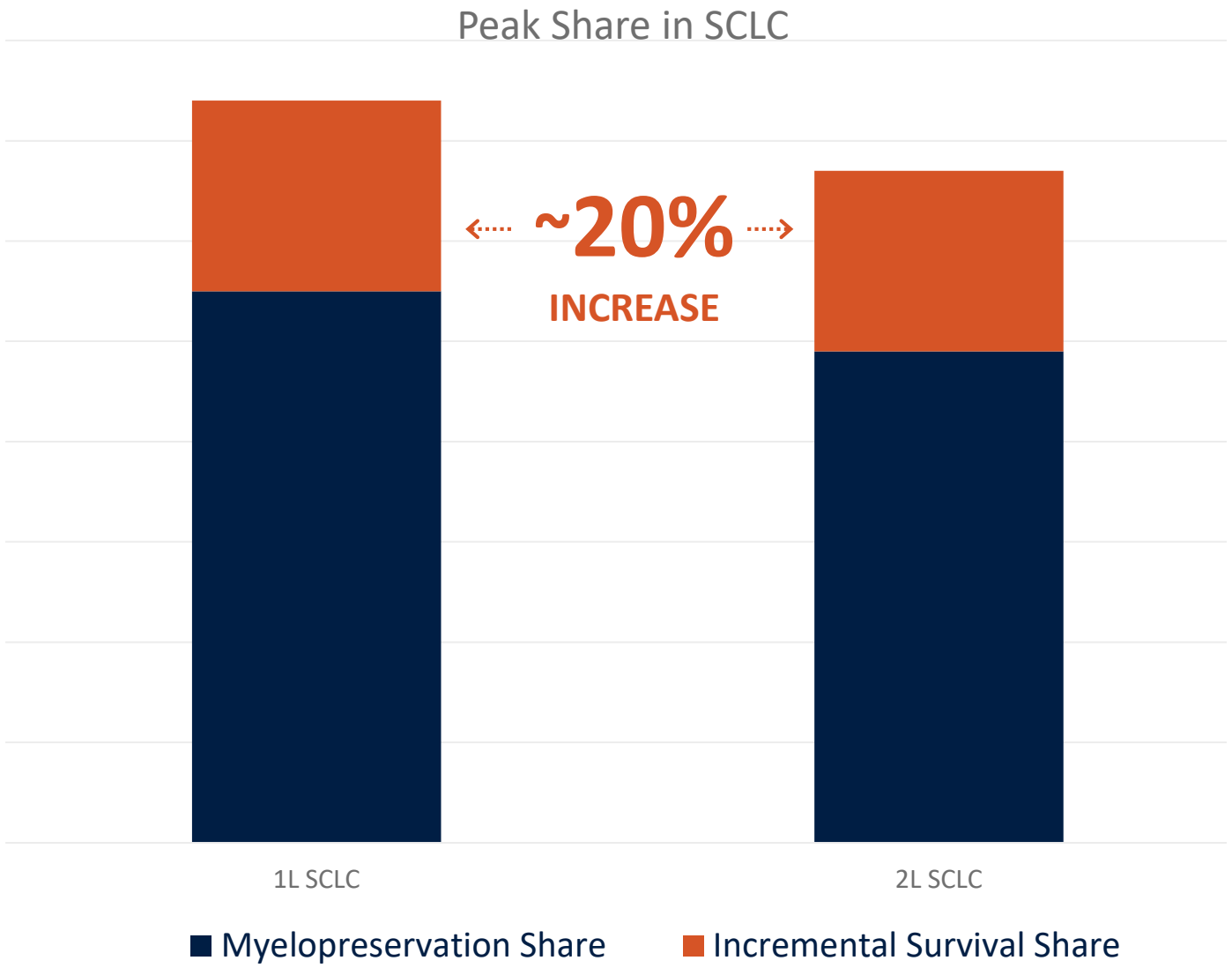
100+ PHYSICIANS AND
15+ PAYORS ACROSS
5 COUNTRIES

---

interviewed using target product profile based on the actual data from trilaciclib + chemo 1st-line SCLC trial

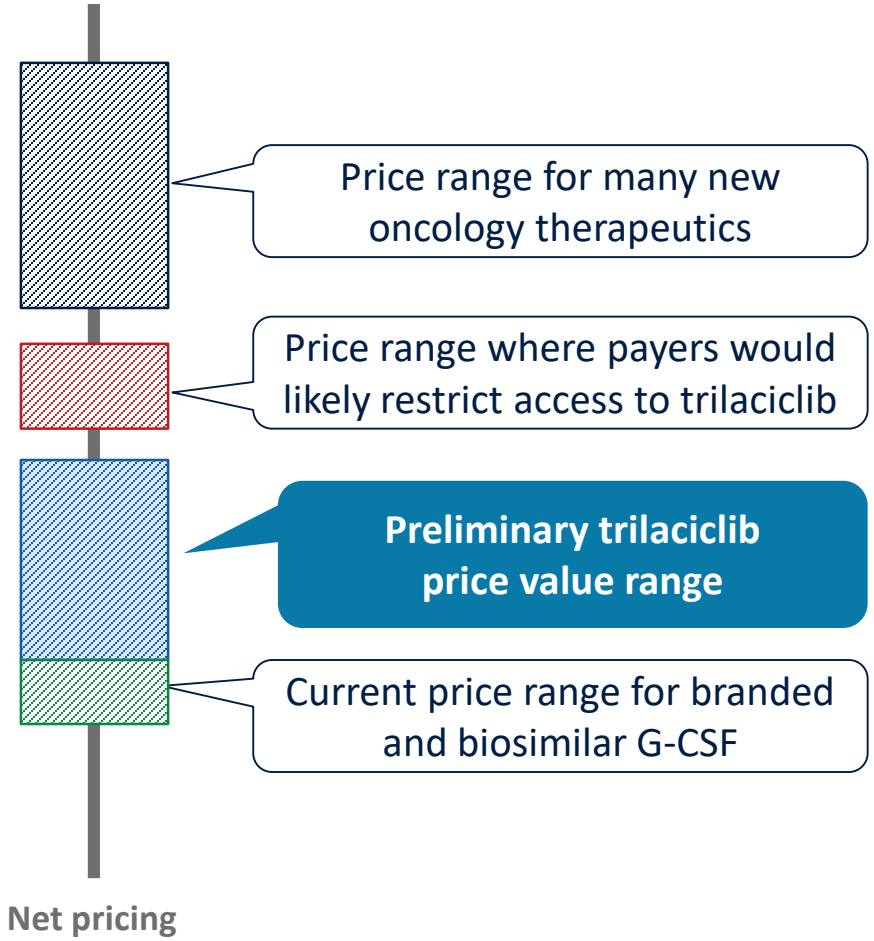
G1 THERAPEUTICS

Myelopreservation alone, without a survival benefit, drives significant share in SCLC



- Physicians were shown a profile based on the actual myelopreservation data from the first line trial of trilaciclib + chemo and asked to allocate share
- Physicians were then shown a second profile with a three-month survival benefit added and asked to allocate share
- **Given physicians would treat a high percentage of patients based on the myelopreservation data alone, a survival benefit, if seen, would have a smaller incremental impact on share**

Preliminary price range where payers believe trilaciclib provides value and they will not restrict patient access



- Payers were shown the trilaciclib profile based on actual first-line data and asked about various pricing scenarios
- Given the multi-lineage benefits, initial payer research suggested a price premium above G-CSF, but below new therapeutics, would appropriately reflect the value trilaciclib provides patients
- The preliminary price range for trilaciclib is based on the value trilaciclib provides patients and is below the range where payors suggested significant access restrictions like step edits would be utilized
- Additional payer research and interactions are planned in 2019 and 2020

Trilaciclib does not add significant costs to the standard of care given the cost offsets trilaciclib provides

Cost Drivers with Trilaciclib

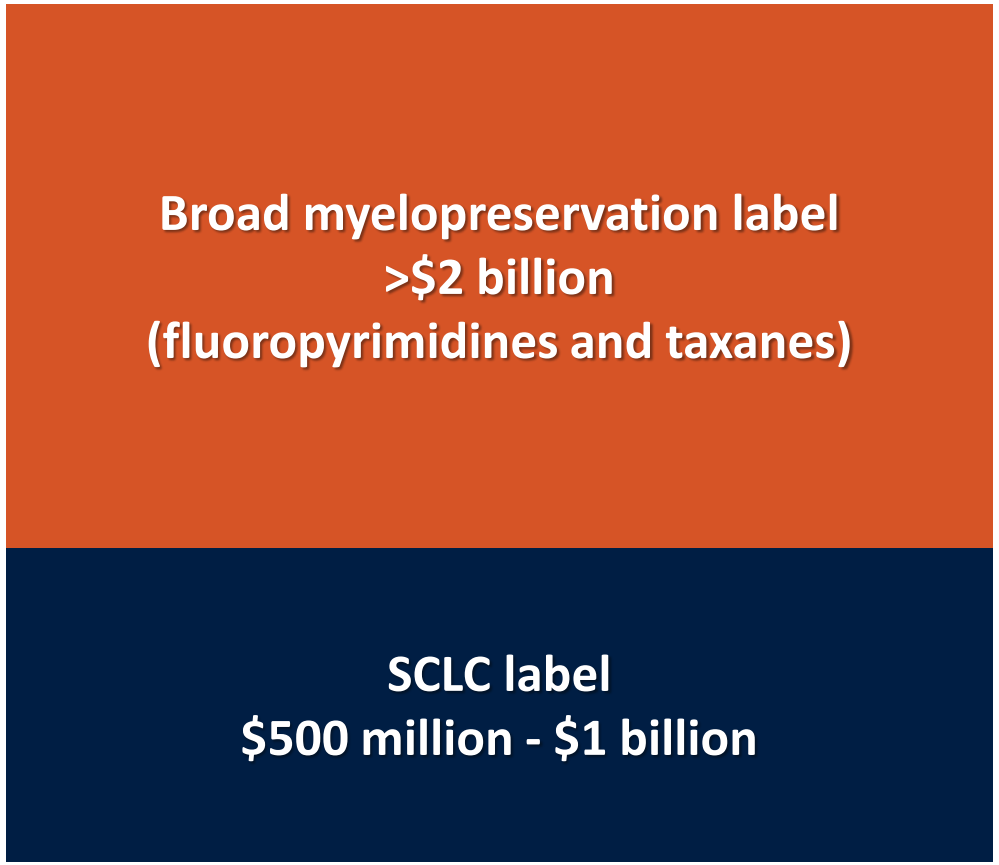
- Cost of trilaciclib
- Administration costs

Cost Savers with Trilaciclib

- Decrease in granulocyte colony stimulating factor (G-CSF) costs
- Decrease in hospitalizations
- Decrease in erythropoiesis stimulating agent (ESA) costs
- Decrease in red blood cell transfusion costs
- Decrease in platelet transfusion costs
- Decrease in incremental physician visits

- Payer research suggested the price range appropriately reflected trilaciclib's value, even before accounting for the cost offsets
- Given these cost offsets, the Budget Impact Model shows a de minimis impact on payer per member, per month (PMPM) drug costs

Trilaciclib market research highlights a substantial global commercial opportunity

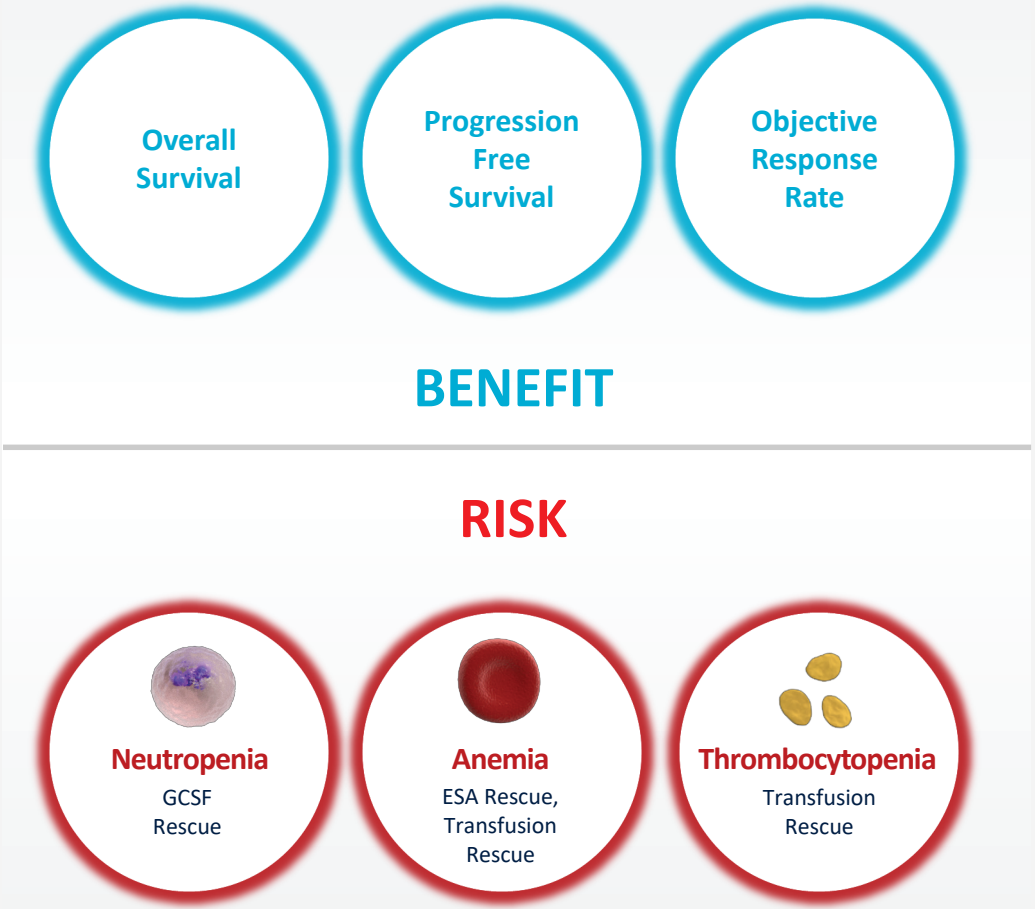


- Proactive myelopreservation is seen as a better approach for patients
- Physicians anticipate significant use of trilaciclib based on its myelopreservation benefits alone
- Payers see the multi-lineage benefits of trilaciclib as unique

Value Proposition

First and only therapy to improve Benefit/Risk Ratio by proactively reducing adverse events across all lineages

CURRENT



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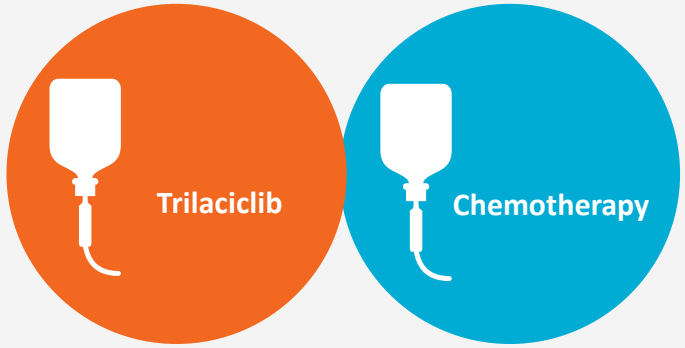
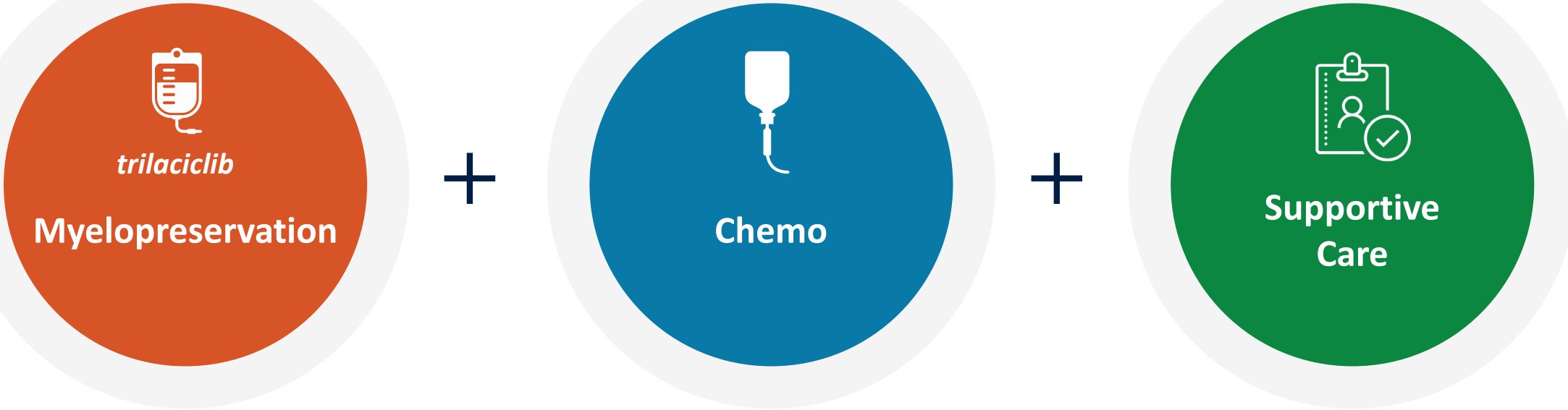
NEW



No detrimental impact on chemotherapy efficacy

Proactively reduces risk of AEs, rescue interventions, and chemotherapy dose delays/reductions

Trilaciclib optimally positioned as first-in-class, multi-lineage myelopreservation therapy that is complementary to SOC



Differentiating and motivating positioning

Proactively reduces myelotoxicity by preserving HSPC & immune system function

Does not compete with the current SOC

Potential for broad use across tumors/chemo

Convenient 30 min IV infusion prior to chemo

Rescue measures following chemotherapy

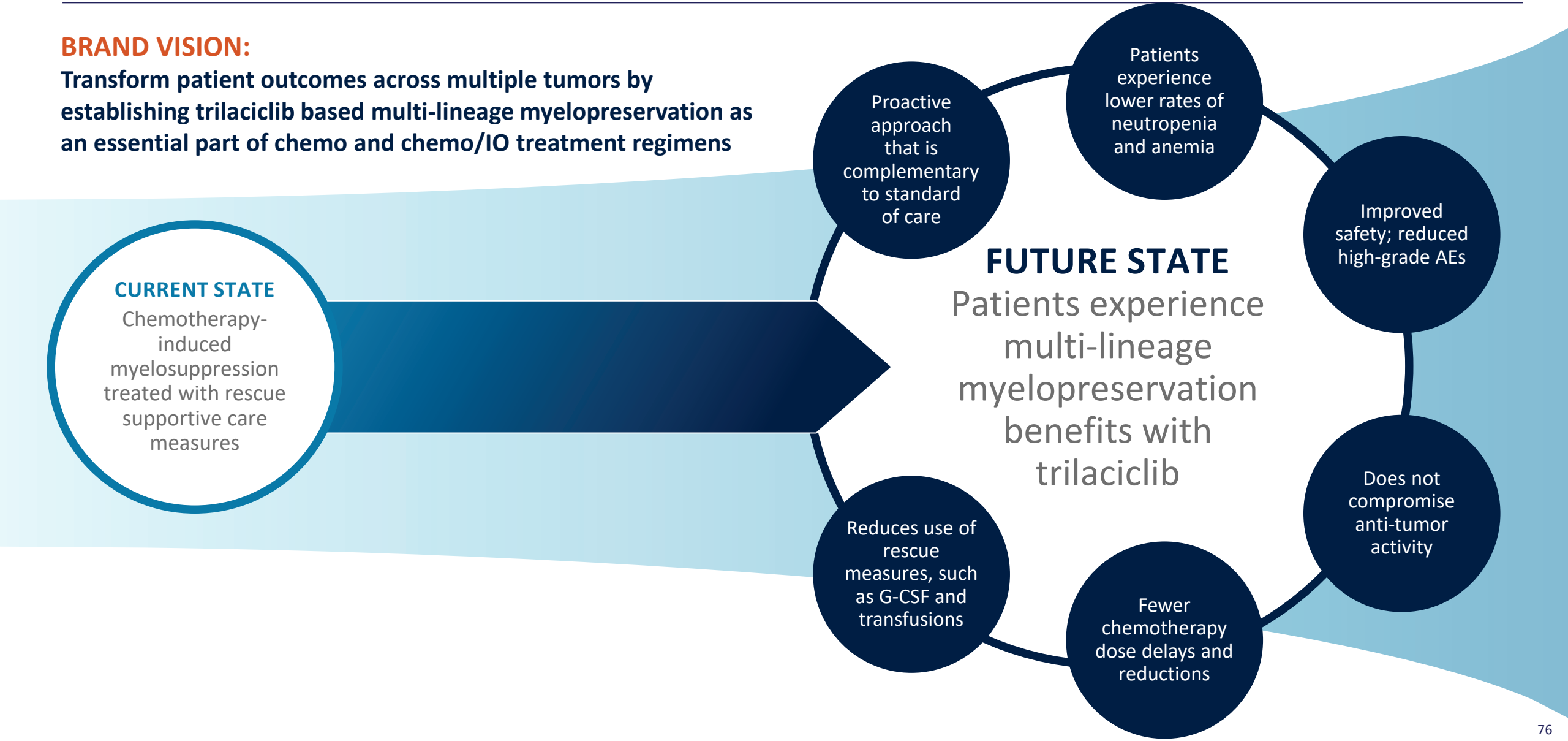
(G-CSF, ESAs and transfusions)

HSPC = hematopoietic stem and progenitor cell SOC = standard of care

Trilaciclib: first-in-class multi-lineage myelopreservation therapy

BRAND VISION:

Transform patient outcomes across multiple tumors by establishing trilaciclib based multi-lineage myelopreservation as an essential part of chemo and chemo/IO treatment regimens



Summary

Key takeaways for trilaciclib

1

There is still substantial unmet need for patients experiencing myelosuppression, despite the availability of rescue interventions like G-CSF, ESAs and transfusions

- no significant innovations for chemotherapy-induced myelosuppression
- chemotherapy will remain the backbone of treatment

2

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Q&A

LEROCICLIB and G1T48 DEVELOPMENT UPDATE

Raj Malik, M.D.
Chief Medical Officer and Senior Vice President, R&D

Tumor cell proliferation

Key takeaways

1

Lerociclib – potential in multiple tumor types

- ongoing trials in breast and non-small cell lung cancer
- differentiated profile = “partner of choice” in CDK4/6i combo regimens

2

G1T48 – oral SERD offers significant patient benefit

- oral delivery provides opportunity to move SERD into earlier lines of therapy
- monotherapy and combination regimens

3

Lerociclib + G1T48 – all-oral BC regimen

- all-oral regimen offers significant differentiation in crowded breast cancer space

Lerociclib development opportunities

Breast Cancer

- ✓ Differentiated profile
- ✓ Partnership opportunities to maximize value

Combined with G1T48

- ✓ Wholly-owned all-oral CDK4/6i + SERD combination in ER+ breast cancer

Beyond Breast Cancer

- ✓ Tolerability profile supports combination with other targeted agents
- ✓ Significant opportunity in multiple tumors

Lerociclib profile differentiated in CDK4/6 landscape

- Differentiated PK and tolerability profile vs. competitors
- Continuous dosing (no holiday) with fewer dose-limiting toxicities
- Highly potent and selective with demonstrated anti-tumor POC

	DOSE-LIMITING NEUTROPENIA	MONITORING REQUIREMENT	DOSING HOLIDAY	QT PROLONGATION	DILI	GRADE 3/4 DIARRHEA	VTE
lerociclib	–	Potential for less monitoring	–	–	–	–	–
Ibrance®	X	X	X	–	–	–	–
Kisqali®	X	X	X	X	X	–	–
Verzenio®	X	X	–	–	X	X	X

lerociclib data from ASCO 2018

Lerociclib differentiated safety and tolerability profile: CDK4/6i partner of choice for combo regimens

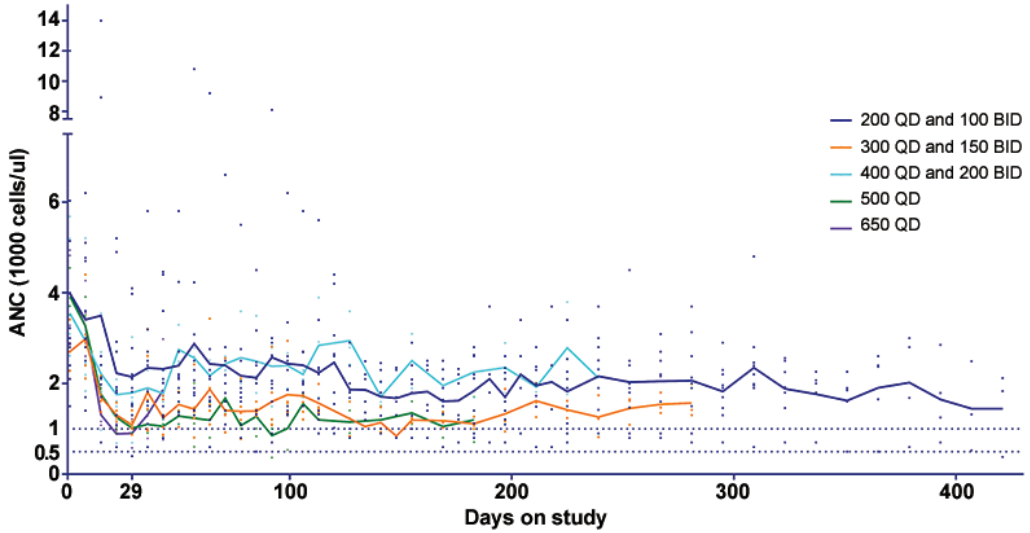
CANCER	INDICATION	lerociclib +	STATUS
BREAST	**ER+/HER2-**	**Faslodex® (fulvestrant)**	**Phase 2a enrolling; Phase 1b data update 4Q19**
	ER+/HER2-	**G1T48**	**Phase 1b/2 trial planned for 2019/2020**
LUNG	**EGFRm**	**Tagrisso**	**Phase 1b data in 3Q19**
PROSTATE	CRPC	AR-antagonist	Exploring
LYMPHOMA	Mantle Cell	BTKi	Exploring
BLADDER	Urothelial	FGFRi	Exploring
GI	Pancreatic	MAPKi	Exploring

ER+, HER2- breast cancer Faslodex® combination Phase 1b/2a trial

PRIMARY ENDPOINTS	• Assess safety, dose-limiting toxicities, and identify recommended Phase 2 dose/schedule
SECONDARY ENDPOINTS	• PK, PD • ORR, PFS and OS
DESIGN	• Open-label, single-arm; continuous dosing of lerociclib + Faslodex in ER+, HER2- breast cancer • Phase 1b: dose escalation (QD and BID schedules), 3+3 design • Phase 2a: dose expansion at RP2D/schedule
MILESTONE TIMING	• Phase 1b dose escalation completed; preliminary data presented at ASCO 2018 • Enrolling expansion phase to identify differentiated clinical profile • Anticipate reporting additional Phase 1b data in 4Q19

Continuously dosed lerociclib: promising early data

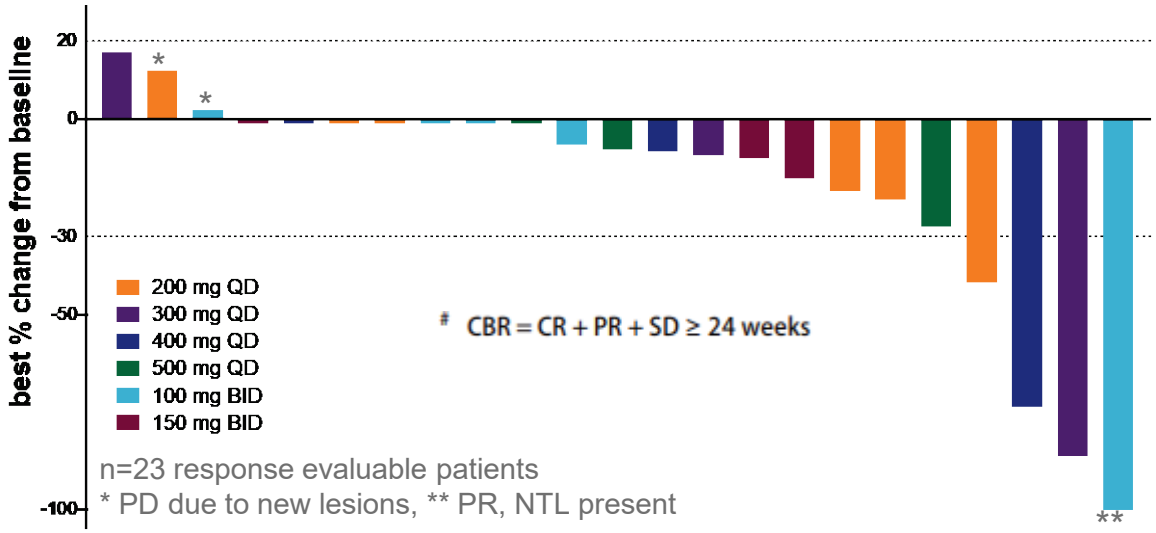
Continuous dosing with less Gr 4 neutropenia



Dose	N	Day 29 mean % change
200mg QD	6	-48%
300mg QD	3	-66%
400mg QD	3	-50%
500mg QD	4	-74%
650mg QD	6	-76%

ANC decreases ~50-60% for approved CDK4/6 inhibitors

Anti-tumor activity at all dose levels



n=23 response evaluable patients
* PD due to new lesions, ** PR, NTL present

Best Response lerociclib + fulvestrant (n=23)	
PR	4/23 (17%)
SD	16/23 (70%)
PD	3/23 (13%)
SD ≥ 24 weeks	11/23 (48%)
CBR 24	15/23 (65%)

data from ASCO 2018; efficacy data updated to account for one patient with non-measurable disease at baseline

CBR 24=CR+PR+SD≥24 weeks

EGFRm NSCLC Tagrisso combination
Phase 1b dose-finding/Phase 2 randomized trial

PRIMARY ENDPOINTS	• Assess safety, dose-limiting toxicities, and identify recommended Phase 2 dose • PFS
SECONDARY ENDPOINTS	• PK, PD • ORR and OS
DESIGN	• EGFRm NSCLC • Phase 1b: single-arm dose-finding, lerociclib + Tagrisso in 1st/2nd-line setting • Phase 2: lerociclib + Tagrisso, or Tagrisso; randomized (1:1)
MILESTONE TIMING	• Phase 1b enrollment ongoing • Preliminary Phase 1b data expected in 3Q19

Non-exclusive clinical trial collaboration with AstraZeneca

Strong rationale for lerociclib + Tagrisso

1

Goal is to extend time to resistance and improve PFS

2

In ER+ breast cancer, CDK4/6 inhibitors as a class extend PFS when added to endocrine therapy

- addition of lerociclib to osimertinib follows this same paradigm of adding a CDK4/6 inhibitor to a growth signaling inhibitor as a means to extend progression free survival

3

Lerociclib combined with osimertinib could extend time to resistance

- osimertinib resistance mechanisms include activating mutations in PIK3CA, BRAF and KRAS; amplifications in MET, HER2, FGFR1, CCND, CDK4/6; and EGFR C797S mutation, many of which are “upstream” of CDK4/6
- preclinical data demonstrated prolongation of time to resistance by the addition of lerociclib to EGFR TKIs (Sorrentino et al 2018)

ORAL SERD G1T48

Tumor cell proliferation

G1T48: strong strategic fit and patient need

1

Selective estrogen receptor degrader (SERD): validated approach for ER+ breast cancer

2

Faslodex (IM SERD): approved as monotherapy and in combination with CDK4/6i

- > $1B sales despite painful intramuscular (IM) injections; oral SERD addresses unmet patient need

3

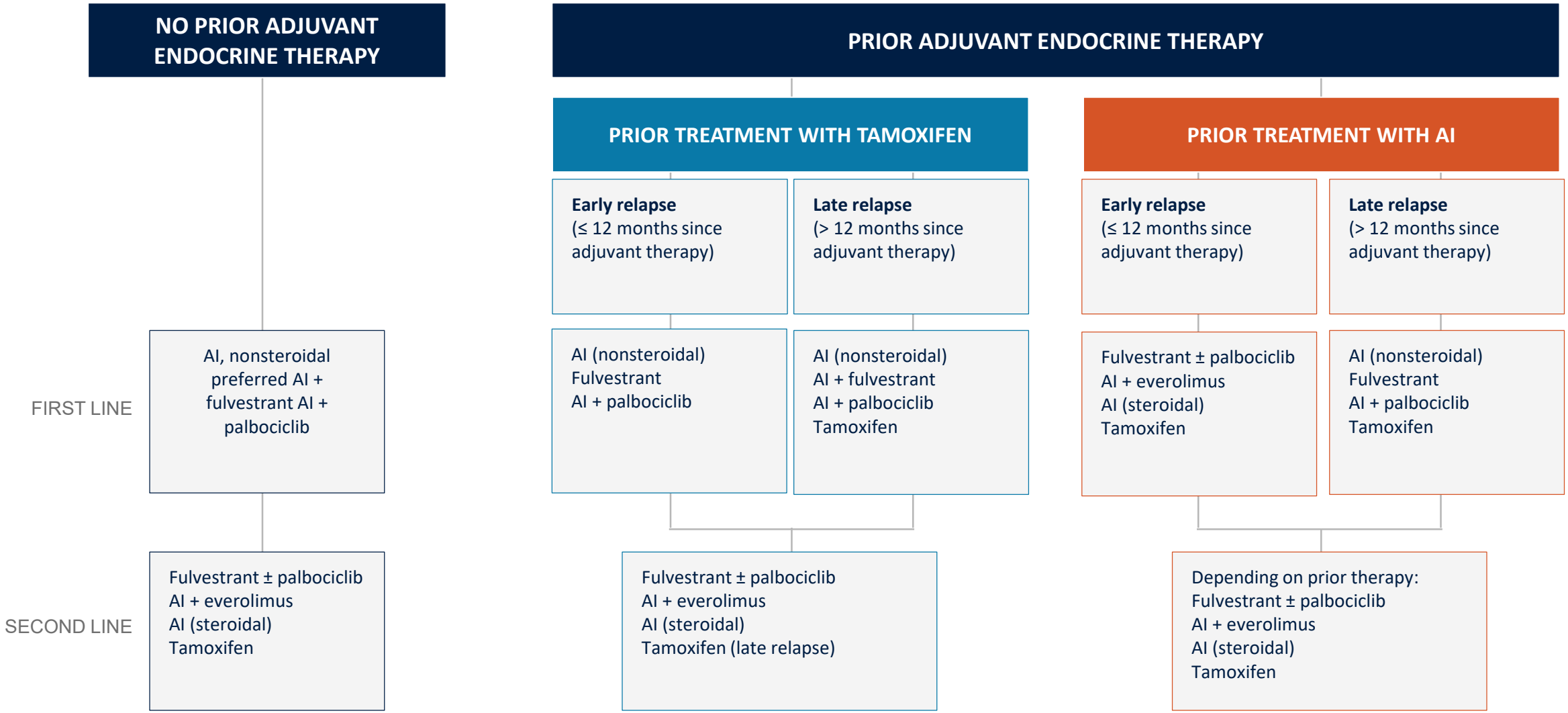
All-oral lerociclib/G1T48 combination regimen offers potential competitive advantages

4

Phase 1 trial in ER+ breast cancer (G1T48 monotherapy)

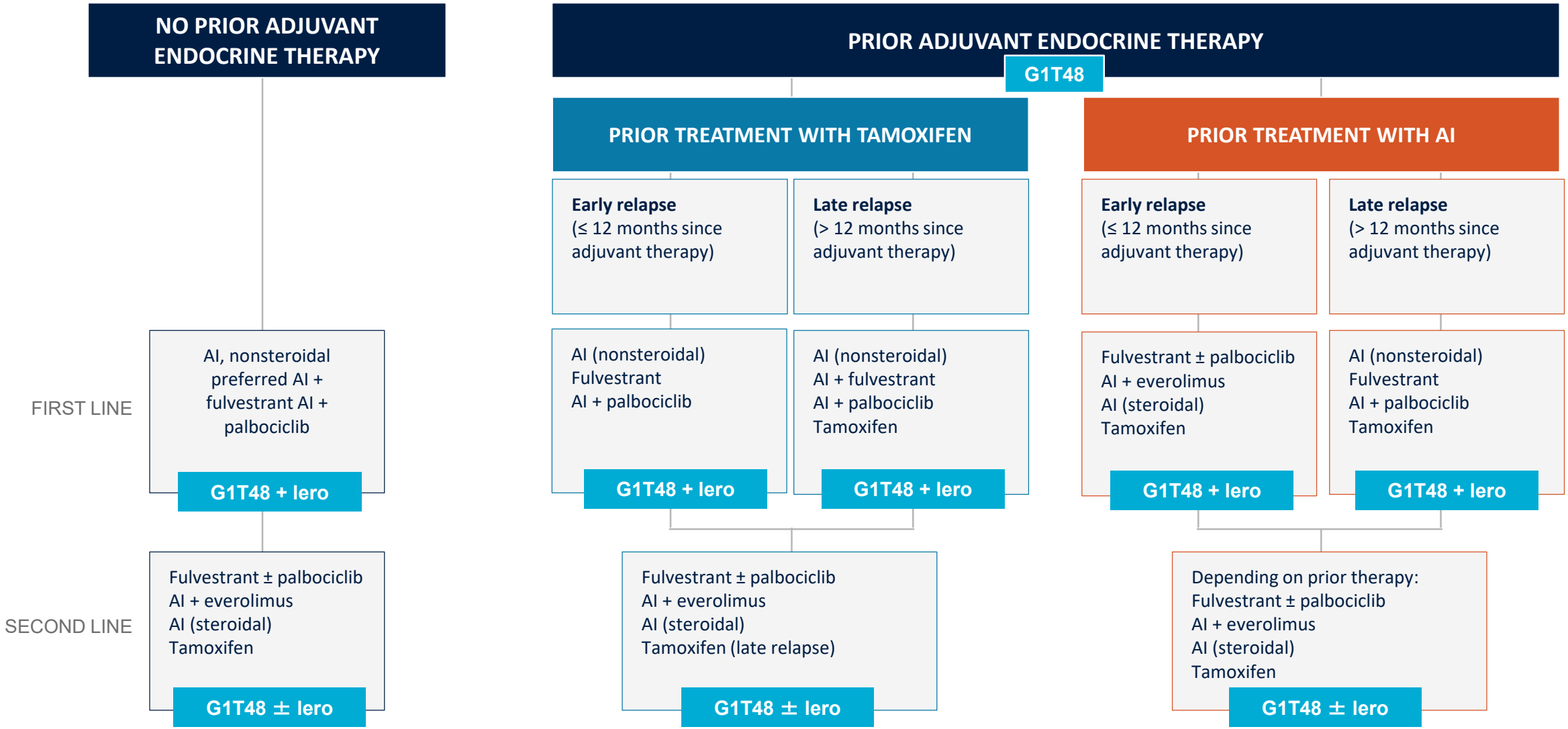
- initiated in 2018
- preliminary data expected in 4Q19
- plan to combine with lerociclib in 2019/2020, pending monotherapy trial results

ASCO guidelines for HR+ mBC: Oral SERD creates opportunity across lines of therapy



Adapted from Rugo et al JCO 2016

ASCO guidelines for HR+ mBC: Oral SERD creates opportunity across lines of therapy



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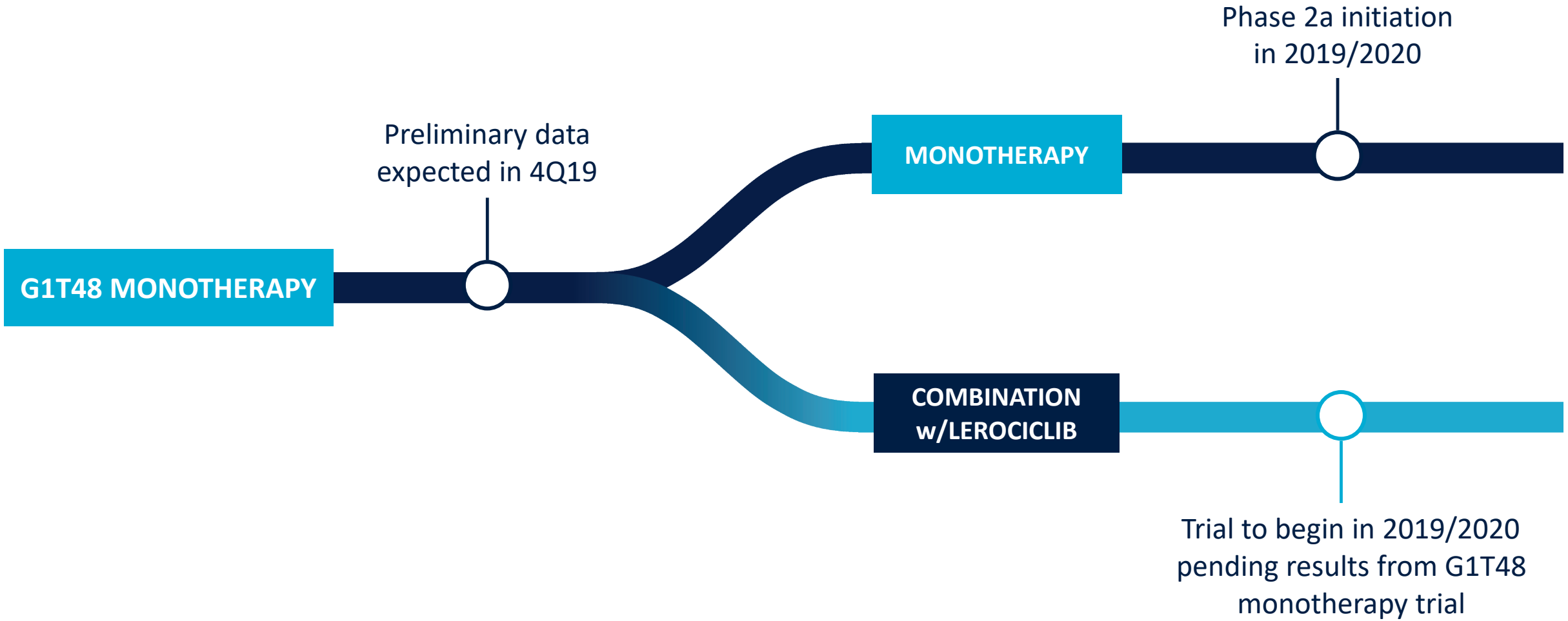
Development pathways



ER+, HER2- breast cancer
Phase 1/2a trial

PRIMARY ENDPOINTS	• Assess safety, dose-limiting toxicities, and identify recommended Phase 2 dose
SECONDARY ENDPOINTS	• PK, PD • ORR and OS • Food effect on bioavailability
DESIGN	• Open-label, ER+, HER2- breast cancer, enrolling up to 104 patients • Phase 1: dose-finding, G1T48 monotherapy in 2nd/3rd-line setting • Phase 2a: dose expansion at RP2D
MILESTONE TIMING	• Phase 1 enrollment ongoing • Report preliminary Phase 1 data in 4Q19

Key takeaways

1

Lerociclib – potential in multiple tumor types

- ongoing trials in breast and non-small cell lung cancer
- differentiated profile = “partner of choice” in CDK4/6i combo regimens

2

G1T48 – oral SERD offers significant patient benefit

- oral delivery provides opportunity to move SERD into earlier lines of therapy
- monotherapy and combination regimens

3

Lerociclib + G1T48 – all-oral BC regimen

- all-oral regimen offers significant differentiation in crowded breast cancer space

Q&A

Key anticipated milestones

	INDICATION/COMBO	2Q19		3Q19	4Q19
trilaciclib IV - CDK4/6i	1st-line SCLC (+ etop/carbo)	Present additional Phase 2 data	Provide regulatory update	Present additional Phase 2 data (pending mature OS)	
	1st-line SCLC (+ etop/carbo/Tecentriq)				
	2nd/3rd-line SCLC (+ topotecan)				
	Metastatic TNBC (+ gem/carbo)				Present additional Phase 2 data (pending mature PFS)
lerociclib Oral - CDK4/6i	ER+, HER2- BC (+ Faslodex)				Report additional Phase 1b data
	EGFRm NSCLC (+ Tagrisso)			Report preliminary Phase 1b data	
G1T48 Oral - SERD	ER+, HER2- BC (monotherapy)				Report preliminary Phase 1 data

Investor Day

March 6, 2019

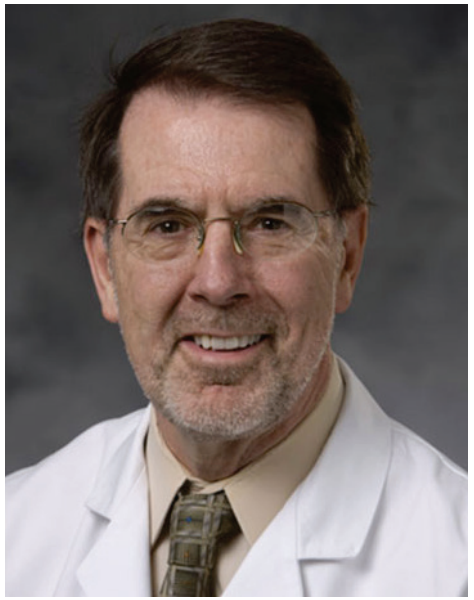
www.g1therapeutics.com

NASDAQ: GTHX

SPEAKER BIOS

Jeffrey Crawford, M.D.

Co-Director, Solid Tumor Therapeutics Program, Duke Cancer Institute



Jeffrey Crawford, M.D., is George Barth Geller Professor for Research in Cancer and Duke University Medical Center and Co-Director of the Solid Tumor Therapeutics Program in the Duke Cancer Institute (DCI) in Durham, North Carolina. He earned his medical degree from Ohio State University and completed his internship, residency and hematology/oncology fellowship at Duke University Medical Center. He is board certified in internal medicine, hematology and oncology.

Dr. Crawford is Principal Investigator for the National Clinical Trials Network Lead Academic Site Grant at Duke. He is a member of the executive committee for the Alliance and served as Chair of NCCN Myeloid Growth Factors Panel for 15 years.

Dr. Crawford's research interests include new treatment approaches to lung cancer, supportive care therapies, including hematopoietic growth factors, and agents that impact muscle wasting. He has published more than 180 manuscripts and chapters. As NCCN panel chair, he helped develop the guidelines for the first FDA approved biosimilar, filgrastim-Sndz. Due to his experience with growth factors and other biologics, Dr. Crawford has participated in the review process for several other biosimilars in development, as well as helped develop national educational programs for the incorporation of biosimilars into oncology.

Lowell Hart, M.D.

Scientific Director of Clinical Research, Florida Cancer Specialists

As the Scientific Director of Clinical Research, Lowell Hart, M.D. oversees the extensive clinical trial and research program at Florida Cancer Specialists & Research Institute. As a strategic research site for the largest community-based clinical trial organization in the nation, Florida Cancer Specialists & Research Institute can offer our patients more access to clinical trials than any other oncology practice, hospital, or academic medical center program in the state of Florida. Dr. Hart graduated with a B.A. from Columbia University in New York City, and completed his Internship and Residency at the University of Miami Hospitals.

His Fellowship in Hematology & Oncology was with Duke University Medical Center in Durham, N.C., and he later served at Duke as an Attending Physician in the Breast Oncology Clinic. Dr. Hart joined Florida Cancer Specialists in 1989 and was named Research Director in 2003. He has served as a sub-investigator or principal investigator in well over 100 clinical trials, and has extensively published the results of his research. He has served on the Board of Directors for the Florida Society of Clinical Oncology.

Since 2016, Dr. Hart has also been Associate Professor of Internal Medicine, Oncology and Hematology at Wake Forest School of Medicine and Co-Director of the Phase 1 Program at Wake Forest Baptist Comprehensive Cancer Center in North Carolina. He currently serves on the Young Investigator Award Review Committee for the American Society of Clinical Oncology.

Mark Velleca, M.D., Ph.D.

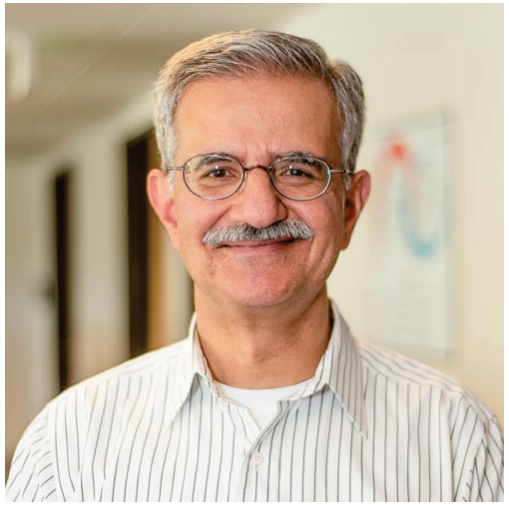
Chief Executive Officer

Mark Velleca, M.D., Ph.D., joined G1 as Chief Executive Officer in 2014. Previously, he was at CGI Pharmaceuticals, where he guided the company from its inception through its establishment as a drug discovery company that brought multiple drug candidates into clinical trials. After Gilead Sciences acquired CGI, Dr. Velleca served as a senior advisor to Gilead in R&D Strategy and Corporate Development. Earlier in his career, Dr. Velleca was an attending physician at Yale New Haven Hospital and on the faculty of the Yale University School of Medicine.

Dr. Velleca earned an M.D. and Ph.D. from Washington University in St. Louis, and a B.S. from Yale University. He has served on the boards of directors and scientific advisory boards of several biotechnology companies, including Intellikine, an oncology therapeutics company acquired by Takeda in 2011. He most recently served as Executive Vice President at the Leukemia & Lymphoma Society.

Raj Malik, M.D.

Chief Medical Officer and Senior Vice President, R&D



Raj Malik, M.D. joined G1 in 2014. In his role as Chief Medical Officer and Senior Vice President, R&D, he leads the company's clinical development, medical affairs, regulatory affairs, biometrics, translational medicine and preclinical teams. Previously, he served as Chief Medical Officer and management board member at Agennix AG, where he was responsible for research and development. Prior to Agennix AG, he served as Chief Medical Officer at Adherex Technologies, where he directed the company's global regulatory strategy and clinical development programs. Dr. Malik also served in oncology clinical development positions at EMD Pharmaceuticals and Bristol-Myers Squibb. Dr. Malik currently serves on the board of directors of Meryx, Inc.

Dr. Malik received his M.D. from the University of Sheffield Medical School in the UK. He completed his residency at Duke University Medical Center and fellowships at the Children's Hospital of Philadelphia and Duke University Medical Center. During his academic career, he was an assistant professor at the University of Virginia, where he conducted basic science and clinical research in addition to patient care and teaching.

John Demaree

Chief Commercial Officer

John Demaree joined G1 as Chief Commercial Officer in 2018. In this role, he is responsible for all aspects of product commercialization, including marketing, sales, market access and commercial operations. Previously, Mr. Demaree served as Vice President, Oncology Marketing for Astellas where he led several product launches. He has worked for more than 20 years in oncology including roles of increasing leadership at Eli Lilly, Novartis and Abbott.

Mr. Demaree brings to G1 a strong history of building commercial capabilities and leading multiple successful new product and new indication launches. His areas of expertise include strategy, commercialization, new product launches, market access and reimbursement strategy, internal and external collaboration leadership, and business development and licensing.

Mr. Demaree received his B.S. in Marketing and his MBA in Marketing & Finance from Indiana University.