Forward-Looking Statements

Except for the historical information contained herein, this presentation contains forward-looking statements made pursuant to the “safe harbor” provisions of the Private Securities Litigation Reform Act of 1995. Investors are cautioned that statements in this presentation regarding: (i) continued conduct by Janssen of IMbarkTM or IMergeTM; (ii) Janssen obtaining additional or more mature data from IMbark or IMerge; (iii) that Janssen will conduct any additional or further data reviews in IMbark or IMerge, and the timing of such data reviews; (iv) potential outcomes of any data reviews conducted by Janssen; (v) imetelstat having activity in MF, MDS or any other hematologic myeloid malignancies, including acute myeloid leukemia; (vi) imetelstat treatment suppressing the malignant clones underlying the disease in hematologic myeloid malignancies; (vii) the safety and efficacy of imetelstat; (viii) the potential receipt by Geron of additional payments up to a potential total of $900 million for the achievement of development, regulatory and commercial milestones, and royalties from sales of imetelstat under the collaboration agreement with Janssen; (ix) Geron’s desire to diversify; (x) plans for presentation of clinical data at future medical conferences; (xi) financial projections and expectations; and (xii) other statements that are not historical facts, constitute forward-looking statements. These statements involve risks and uncertainties that can cause actual results to differ materially from those in such forward-looking statements. These risks and uncertainties, include, without limitation, risks and uncertainties related to: (i) whether imetelstat will succeed in IMbark and IMerge by overcoming all of the clinical safety and efficacy, technical, scientific, manufacturing and regulatory challenges; (ii) whether health authorities permit IMbark or IMerge to continue to proceed; (iii) Janssen's ability to collect additional and more mature data from current clinical trials of imetelstat; (iv) whether Janssen continues to conduct IMbark or IMerge; (v) Geron’s total dependence on Janssen for the development, regulatory approval, manufacture and commercialization of imetelstat, including the risks that if Janssen were to breach or terminate the collaboration agreement or otherwise fail to successfully develop and commercialize imetelstat and in a timely manner, or at all, Geron would not obtain the anticipated financial and other benefits of the collaboration agreement with Janssen and the clinical development or commercialization of imetelstat could be delayed or terminated; (vi) whether imetelstat is safe and efficacious, and whether any future efficacy or safety results may cause the benefit/risk profile of imetelstat to become unacceptable; (vii) whether imetelstat can be applied to any or to multiple hematologic malignancies; (viii) the fact that Geron may not receive any or limited milestone, royalty or other payments from Janssen because Janssen may terminate the collaboration agreement for any reason or because imetelstat is unsuccessful developmentally or commercially; (ix) the ability of Geron and Janssen to protect and maintain intellectual property rights for imetelstat; (x) the need for future capital; and (xi) whether Geron is able to acquire any new product candidates, programs or companies to enable it to diversify. Additional information on the above risks and uncertainties and other factors that could cause actual results to differ materially from those in the forward-looking statements are contained in Geron’s periodic reports filed with the Securities and Exchange Commission under the heading “Risk Factors,” including Geron’s quarterly report on Form 10-Q for the quarter ended September 30, 2016. Undue reliance should not be placed on forward-looking statements, which speak only as of the date they are made, and the facts and assumptions underlying the forward-looking statements may change. Except as required by law, Geron disclaims any obligation to update these forward-looking statements to reflect future information, events or circumstances.
Geron Overview

I metelstat’s clinical activity suggests impact on underlying disease in hematologic myeloid malignancies
• Inhibiting telomerase activity impedes production of malignant cells
• First telomerase inhibitor in clinical development
• Molecular responses seen in essential thrombocythemeia (ET) and remissions observed in myelofibrosis (MF) indicate disease-modifying activity in hematologic malignancies
• Phase 2 clinical development being pursued in MF and MDS

Insufficient options for myelofibrosis patients when front-line JAK inhibitor therapy ineffective
• High discontinuation rate (~75%) due to suboptimal response or loss of therapeutic effect
• Short median survival (~7 months) after ruxolitinib failure or discontinuation
• Challenging patient populations: COMFORT I spleen volume response rates not replicated in subsequent front- or second-line studies with other JAKis
• New treatment approaches needed to alter underlying disease

Conventional treatments for lower-risk myelodysplastic syndromes inadequate
• No new drugs approved for > 10 years; many patients are dependent on transfusions to treat chronic anemia characteristic of this disease
• Transfusions shorten median survival and increase risk of transformation to AML

Ongoing clinical trials being conducted by Janssen
• IMbark (Phase 2 MF study) initiated in September 2015
• IMerge (Phase 2/3 MDS study) initiated in January 2016
• 1st internal data review of both studies conducted in Q3 2016
• 2nd internal data review of both studies planned by the end of Q2 2017 to enable assessment of further development plans

Financial position provides strategic business options
• ~$129 million in cash and investments as of December 31, 2016 (unaudited)
• Ability to act on potential imetelstat development opportunities
• Pipeline diversification through possible acquisitions of new oncology products, programs or companies
Telomerase enzyme

Comprised of an RNA template component (hTR) and a reverse transcriptase catalytic protein subunit (hTERT)

Binds to the 3’ strand of DNA and adds TTAGGG nucleotide repeats to offset the loss of telomeric DNA at the end of chromosomes that occurs with each replication cycle.
Telomerase transiently upregulated to support controlled proliferation; not active in somatic cells

Telomerase highly upregulated, enabling continued and uncontrolled proliferation
Imetelstat
A first-in-class telomerase inhibitor

- **Proprietary:** 13-mer thio-phosphoramidate oligonucleotide complementary to hTR, with covalently-bound lipid tail to increase cell permeability/tissue distribution

- **Long tissue residence time** in bone marrow, spleen, liver (0.19 – 0.51 µM observed in human bone marrow at 41 – 45 hours post 7.5 mg/kg dose)

- **Potent competitive inhibitor of telomerase:** IC50 = 0.5 – 10 nM (cell-free)

- **Target:** malignant progenitor cell proliferation

- **Clinical experience:** over 500 patients treated in Phase 1 and 2 trials; safety profile generally consistent
  - reported adverse events (AEs) and laboratory investigations include cytopenias, gastrointestinal symptoms, constitutional symptoms, hepatic biochemistry abnormalities
  - myelosuppression is dose-limiting toxicity observed (managed through dose holds and modification rules)
Hematologic Myeloid Malignancies
Arise from malignant progenitor cells in the bone marrow

- ~3,000 cases diagnosed per year in the US
- ~13,000 people in the US living with MF
  - ~70% of patients have high/intermediate-2 risk disease (population for most ongoing clinical trials)

- ~12,000 cases diagnosed per year in the US
- ~60,000 people in the US living with MDS
- ~70% of patients have lower risk disease

- ~20,000 cases diagnosed per year in the US
- ~27% of patients diagnosed are alive after 5 years

Mehta et al, Leuk Lymphoma 2014, 55:595-600
Gangat et al, J Clin Oncol 2011, 29:392-397
Fenaux et al, Blood 2013; 121:4280-4286
NCI SEER database: www.seer.cancer.gov
Targeting MF and MDS
Telomerase: A mechanism to address the underlying disease

**Myelofibrosis**
- **Disease targets:**
  - JAK/STAT signaling
  - cytokine/growth factor function
  - telomerase
- Phenotypic drivers:
  - JAK/STAT signaling pathway gain-of-function mutations (JAK2V617F, CALR, MPL)
  - Release of inflammatory cytokines and growth factors (interleukins, TGF-β)
  - Collagen and reticulin fibers (fibrosis)

**Myelodysplastic Syndromes**
- **Disease targets:**
  - erythropoiesis stimulation
  - growth factor function
  - telomerase
- Driver mutations unknown
- Excessive growth factor signaling/defects in cell maturation
- Anemia and other peripheral cytopenias
Proof-of-concept in essential thrombocythemia

- Molecular responses and allele burden reductions in patients with JAK2V617F, CALR or MPL mutations, and others
- Increases in average telomere length in patients’ granulocytes at time of best molecular response suggest recovery of normal hematopoiesis
- Ex vivo suppression of growth factor independent proliferation of megakaryocytes from patients

Pilot study in myelofibrosis

- Unprecedented complete and partial remissions – CR or PR: 21.2% (7/33) patients – median duration for CR 18 months (range 7 – 20+ at Dec, 2014)
- Molecular remissions in CR patients

Baerolley et al, NEJM 2015;373:920-928
Oppliger Leidengut et al, ASH 2015
Hauhitz et al, ASH 2016
Tefferi et al, NEJM 2015;373:908-919
Myelofibrosis
Disease characteristics

- **Malignant clonal proliferation** and atypical megakaryocytic hyperplasia leads to bone marrow fibrosis and impaired hematopoiesis
  - **Fibrosis** thought to be induced by inflammatory cytokines produced by megakaryocytes originating from the malignant progenitor cell clone
  - **Constitutional symptoms** (e.g., fever, weight loss, night sweats, pruritus) present in approximately 35% of patients also thought to be due to cytokines produced by malignant megakaryocytes
  - Impaired bone marrow hematopoiesis shifts blood production to spleen and liver (palpable **splenomegaly** in approximately 80% of patients)
- **Serious and life-threatening** illness
  - Leukemic transformation to AML (blast-phase MF)
  - Thrombohemorrhagic complications associated with dysfunctional hematopoiesis

Tefferi, JCO 2005; 23:8520-8530
Tefferi, Mayo Clin Proc 2012; 87:25-33
Gangat et al, JCO 2011; 29:392-397
Evolution of Therapy in High/Int-2 Risk MF
Treatment paradigm before ruxolitinib approval in Nov 2011

High or Intermediate-2 Risk MF

Conventional treatments
- drug therapies: e.g., hydroxyurea, steroids, immunomodulatory agents, androgens
- splenectomy, radiotherapy
- allogeneic hematopoietic cell transplantation (limited to very few eligible patients)

Clinical trials
- e.g., ruxolitinib (oral JAK1/JAK2 inhibitor)

COMFORT I
(vs. placebo at 24 weeks)
SVR: 42% vs. <1%
TSS: 46% vs. 5%
3-yr survival: 70% vs. 61%

COMFORT II
(vs. BAT incl. 47% HU, 16% steroids, 33% no therapy at 48 weeks)
SVR: 29% vs. 0%
3-yr survival: 79% vs. 59%

Regulatory co-primary endpoints established:
- ≥35% reduction in spleen volume (SVR) by imaging
- ≥50% improvement in Total Symptom Score (TSS)

Ruxolitinib (Jakafi®) approved
- High/Intermediate Risk MF
- November 2011
Evolution of Therapy in High/Int-2 Risk MF

In 2017 a high unmet medical need remains

Symptoms or splenomegaly
- treatment with ruxolitinib
- stay on drug as long as tolerated (conventional drugs viewed as ineffective, especially in advanced disease)

5-year discontinuation rate is ~75% (COMFORT I & II)
- major reasons:
  - suboptimal response
  - loss of therapeutic effect

After failure or discontinuation median survival is ~7 months (claims database analysis)

Harrison et al, ASH 2015
Gupta et al, ASCO 2016
Mehra et al, ASH 2016
Evolution of Therapy in High/Int-2 Risk MF
Defining patient populations in clinical trials

- **Frontline (JAK inhibitor naïve)**
  - No prior treatment with a JAK inhibitor

- **Second line (JAK inhibitor exposed)**
  - Prior treatment with a JAK inhibitor (e.g., for ≥28 days)

- **Relapsed/Refractory (JAK inhibitor failed)**
  - Disease progression during or after treatment with a JAK inhibitor
  - Never responded to JAK inhibitor treatment
Evolution of Therapy in High/Int-2 Risk MF
Additional JAK inhibitors with Phase 3 results reported

- **Frontline (JAK inhibitor naïve)**
  - **momelotinib** (Gilead)
    - JAK1/JAK2 inhibitor (oral)
  - **SIMPLIFY 1** *(vs. ruxolitinib)*
    - SVR: 26.5% vs. 29.0%
    - TSS: non-inferiority not achieved

- **Second line (JAK inhibitor exposed)**
  - **pacritinib** (CTI BioPharma)
    - JAK2/FLT3/IRAK1/CSF1R inhibitor (oral)
  - **PERSIST 1** *(vs. BAT no JAKi incl. ~38% no therapy)*
    - SVR: 19% vs. 5%
  - *TSS was a secondary endpoint

- **Relapsed/Refractory (JAK inhibitor failed)**

ClinicalTrials.gov
Mesa et al, ASCO 2015
Gilead press release, Nov 16, 2016
Evolution of Therapy in High/Int-2 Risk MF
Additional JAK inhibitors with Phase 3 results reported

**Frontline (JAK inhibitor naïve)**

**SIMPLIFY 1** (vs. ruxolitinib)

SVR: 26.5% vs. 29.0%

TSS: non-inferiority not achieved

**PERSIST 1** (vs. BAT no JAKi

incl. ~38% no therapy)

SVR: 19% vs. 5%

**Second line (JAK inhibitor exposed)**

**SIMPLIFY 2**: >28 days rux,

thrombocytopenic/anemic

(vs. BAT incl. ~88% rux)

SVR: 6.7% vs. 5.8%

TSS (2°): not reported*

**PERSIST 2**: ≤10⁴/µL plts

(vs. BAT incl. ~45% rux,

19% HU, 19% no therapy)

SVR: 18% vs. 3%

TSS: 25% vs. 14%

(~44% received prior rux; remainder naïve)

**Relapsed/Refractory (JAK inhibitor failed)**

**PAC203**

(Patients who have failed prior rux therapy)

Differences in favor of momelotinib were observed for the pre-specified secondary endpoints of TSS and one of the three anemia-related endpoints (transfusion independence), however, formal sequential statistical testing was not undertaken because the primary superiority endpoint was not achieved.

* Differences in favor of momelotinib were observed for the pre-specified secondary endpoints of TSS and one of the three anemia-related endpoints (transfusion independence), however, formal sequential statistical testing was not undertaken because the primary superiority endpoint was not achieved.

** To start in Q2 2017; definition of “failed” not disclosed

**momelotinib**

(Gilead)

JAK1/JAK2 inhibitor

(oral)

**pacritinib**

(CTI BioPharma)

JAK2/FLT3/IRAK1/CSF1R inhibitor

(oral)

ClinicalTrials.gov

Mesa et al, ASCO 2015

Mascarenhas et al, ASH 2016

Gilead press release, Nov 16, 2016

CTI Biopharma press release, Jan 5, 2017
Evolution of Therapy in High/Int-2 Risk MF
Focusing imetelstat on relapsed or refractory patients

**Mayo Pilot Study**
(All-comers High/Int-2 Risk MF incl. 48% JAKi exposed)
- CR or PR: 21.2% (7/33)
- Median duration for CR: 18 months (range 7 to 20+)
- Spleen responses (by palpation): 34.8% (8/23)
- Spleen responses in JAKi exposed subgroup: 27.3% (3/11)

- Unprecedented complete and partial remissions suggested disease-modifying activity

**IMbark**
Phase 2 clinical trial being conducted by Janssen
(First patient: Sep 2015)

Define dosing and confirm activity in rigorously defined R/R population using established regulatory co-primary SVR/TSS endpoints

Tefferi et al, NEJM 2015;373:908-919
Tefferi, et al. ASH 2014
**IMbark**
Original study design

**A multi-center, open label study being conducted by Janssen**

- **Objectives:**
  - **Define proper dosing by evaluating potential therapeutic range of the drug**
    - 4.7 mg/kg q3w: lowest dose where telomerase inhibition is predicted
    - 9.4 mg/kg q3w: max dosing regimen derived from the Mayo Pilot Study
  - **Confirm efficacy in high unmet need population using current established regulatory endpoints**
- **First patient dosed in September 2015; over 100 patients enrolled as of October 2016**

**Randomize (1:1)**

- **Intermediate-2 or high risk myelofibrosis patients relapsed/refractory to JAK inhibitor Treatment (N = ~200)**

- **Imetelstat 9.4 mg/kg every 3 weeks**
  - **Co-1° Efficacy:**
    - Spleen response rate* and symptom response rate**
  - **2° Efficacy:**
    - CR/PR and CI+, anemia response per 2013 IWG-MRT criteria, duration of responses, overall survival
  - **Exploratory:**
    - Cytogenetic and molecular responses, leukemia free survival

- **Imetelstat 4.7 mg/kg every 3 weeks**

* ≥35% reduction in spleen volume at Week 24 from baseline by imaging scans
** ≥50% reduction in Total Symptom Score at Week 24 from baseline by the modified Myelofibrosis Symptom Assessment Form (MFSAF) version 2.0 diary
+ Complete remission (CR) or partial remission (PR), and clinical improvement (CI) per modified 2013 IWG-MRT criteria

More information can be found on ClinicalTrials.gov (NCT02426086).
Current Status of IMbark
Trial ongoing to evaluate more mature MF data

First planned internal data review completed by Janssen in September 2016
• 20 patients from each dosing arm who had been followed for at least 12 weeks

Safety profile was consistent with previous imetelstat clinical trials in hematologic myeloid malignancies
• No new safety signals were identified

Activity in the 4.7 mg/kg dosing arm did not warrant further investigation of that dose
• Arm closed to new patient enrollment
• Patients may continue to receive imetelstat at 4.7 mg/kg
• Protocol amendment submitted to health authorities to allow dose increase to 9.4 mg/kg per investigator discretion

Encouraging trends in efficacy data observed in the 9.4 mg/kg dosing arm warrant further investigation
• Dosing continues at 9.4 mg/kg to obtain additional and more mature data, including longer follow-up at 24 weeks
• Meanwhile new patient enrollment suspended because insufficient number of patients met protocol defined interim criteria at 12 weeks to confirm dose

Second internal data review planned by the end of 2Q 2017
• Over 100 patients enrolled across both dosing arms as of October 2016
• Timing: allows for 24-week follow up of some more recently enrolled patients at the 9.4 mg/kg starting dose
• Objective: to assess whether 9.4 mg/kg is the appropriate starting dose for this patient population
  - enrollment at 9.4 mg/kg starting dose could resume to reach a total of ~100 patients for primary analysis, or
  - a new dosing arm could be added, or
  - the trial could be closed
Myelodysplastic Syndromes
Disease characteristics

- Diverse group of clonal hematologic malignancies with disordered and ineffective production of the myeloid lineage in the bone marrow characterized by abnormal cell morphology and counts (anemia and other cytopenias)
- Median age at diagnosis is ~70 years
- Up to 30% of patients progress to acute leukemia (AML)
- Chronic anemia is predominant clinical problem in lower risk MDS and many patients become dependent on transfusions
- Transfusion dependency may lead to iron overload and is associated with shorter survival (2 units red blood cells per month may reduce life expectancy by 50%) and increased risk of transformation to AML

Sekeres, Natl Compr Canc Netw 2011; 9:57-63
Malcovati et al, JCO 2007; 25:3503-3510
Greenberg et al, Blood 1997; 89:2079-2028
Bejar & Steensma, Blood 2014; 124:2793-2803
Treatment Paradigm for Lower Risk MDS
No new drugs approved for >10 years

Approved or conventional treatments

Del 5q cytogenetic abnormality (5-20% patients)

Lenalidomide
• 55% to 65% RBC-TI (≥8 wk)
• median duration: 2-2.5 years

Erythropoiesis stimulating agents (ESAs)
• improvement in anemia in ~50% of patients
• median duration: ~2 years

Lower Risk MDS
Median survival: 3.5 - 5.7 years
Non-del 5q

HMAs
• mostly reserved for higher risk disease

Red blood cell transfusion dependent

Clinical trials
• e.g., imetelstat, luspatercept

Fenaux and Adès, Blood 2013; 121:4280-4286
Clinical Development in Lower Risk MDS
Chronic anemia remains an unmet need

imetelstat
telomerase inhibitor

Mayo Pilot Study:
IPSS int-1/int-2 RARS
3/8 (37.5%) transfusion dependent patients
achieved RBC-TI ≥8 weeks
Median duration 28 weeks;
9, 28 and 37 weeks respectively

IMerge Phase 2/3:
Being conducted by Janssen
(First patient: Jan 2016)

Luspatercept
(Acceleron/Celgene)
ligand trap inhibiting
TGF-β superfamily

PACE Phase 2:
IPSS low/int-1
11/22 (50%) patients
transfused prior to study
achieved RBC-TI ≥8 weeks;
range: 9-80+ weeks
(Higher response rates in RS+ patients)

MEDALIST Phase 3:
(vs. placebo)
Ongoing

Ringed sideroblast positive
(~20% of lower risk MDS patients)

Ringed sideroblast negative
(~80% of lower risk MDS patients)

ClinicalTrials.gov: NCT02598661 (IMerge), NCT02631070 (MEDALIST)
Platzbecker et al, EHA 2016; Platzbecker et al, ASH 2016
A multi-center, two part study being conducted by Janssen

- Objectives:
  - Part 1 (Phase 2) to evaluate safety and efficacy of imetelstat to advance to Part 2 based on positive assessment of benefit-risk profile in significant unmet medical need population
  - Part 2 (Phase 3) to compare imetelstat to placebo using an established regulatory endpoint
- Dosing regimen used in Mayo Clinic Pilot Study MDS-RARS cohort
- First patient dosed in January 2016; Part 1 fully enrolled

Supportive care permitted in both arms: RBC transfusions, myeloid growth factors per investigator discretion as clinically needed and local guidelines

More information on ClinicalTrials.gov (NCT02598661)
Current Status of IMerge
Trial ongoing to evaluate more mature data

First internal data review completed by Janssen in September 2016
• Subset of patients in Part 1
• Emerging safety and efficacy consistent with data reported from Mayo Clinic Pilot Study MDS-RARS cohort
• Part 1 of the trial continues unmodified

Second internal data review planned by the end of 2Q 2017
• Timing: allows longer follow-up of all Part 1 patients
• Objective: to assess the benefit-risk profile of Part 1 for decision to move forward to Part 2
  - If Janssen moves forward with Part 2, the Phase 3 trial may be open for enrollment in mid-2017

Clinical data from Part 1 expected to be submitted for presentation at a future medical conference
Key Milestones for IMbark and IMerge
Expectations for 2017

1H 2017

Internal data review
- to include patients from 9.4 mg/kg arm with at least 24 weeks follow-up

2H 2017

Possible outcomes:
- enrollment is resumed at 9.4 mg/kg to reach a total of ~100 patients for primary analysis, or
- new dosing arm is added, or
- trial is closed

Note: Clinical data are expected to be presented at a medical conference to be determined in the future. Projected timelines are dependent on patient enrollment rates in clinical studies and study continuation.
Partnership with Janssen
Exclusive worldwide collaboration for imetelstat

First Stage

**IMbark (Phase 2 MF study)**
- Janssen conducting IMbark and IMerge
- Janssen to provide Continuation Decision following primary analysis of IMbark**

**IMerge (Phase 2/3 MDS study)**

Continuation Stage

**Phase 3: MF, MDS**
**Phase 2: Additional exploratory indications**

- Geron has Opt-In right to share further US development and promotion costs
- Under Opt-In, Geron may provide 20% of US selling effort with sales force personnel, in lieu of funding 20% of US promotion costs

First Stage Economics

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<th>Upfront</th>
<th>Cost Share</th>
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<td><strong>IMbark</strong> (Phase 2 MF study)</td>
<td>$35M</td>
<td>50% Geron 50% Janssen</td>
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Continuation Stage Economics

<table>
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<th>Opt-Out</th>
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<td><strong>Primary analysis</strong>*</td>
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<tr>
<td><strong>Phase 2: Additional exploratory indications</strong></td>
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<td>Cost Share</td>
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<td>100% Janssen</td>
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<td>Continuation/US Rights Fee</td>
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<td>$135M</td>
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<td>Dev/Reg Milestones</td>
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<td>Royalty % Tier***</td>
<td>Mid-teens to low twenties</td>
<td>Double digit to mid-teens</td>
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* Due to the current suspension of enrollment in IMbark, the timing of the protocol-specified primary analysis for the trial is uncertain and may be substantially delayed
** If IMbark is terminated early, or placed on clinical hold or suspended by a health authority for an extended period, then Janssen must instead notify Geron of their Continuation Decision ~24 months after the initiation of IMerge

*** Calculated on worldwide net sales in any countries where regulatory exclusivity exists or there are valid claims under patent rights covering composition of matter or methods of use exclusively licensed to Janssen
Thank you

If you have any questions, please contact us:
investor@geron.com