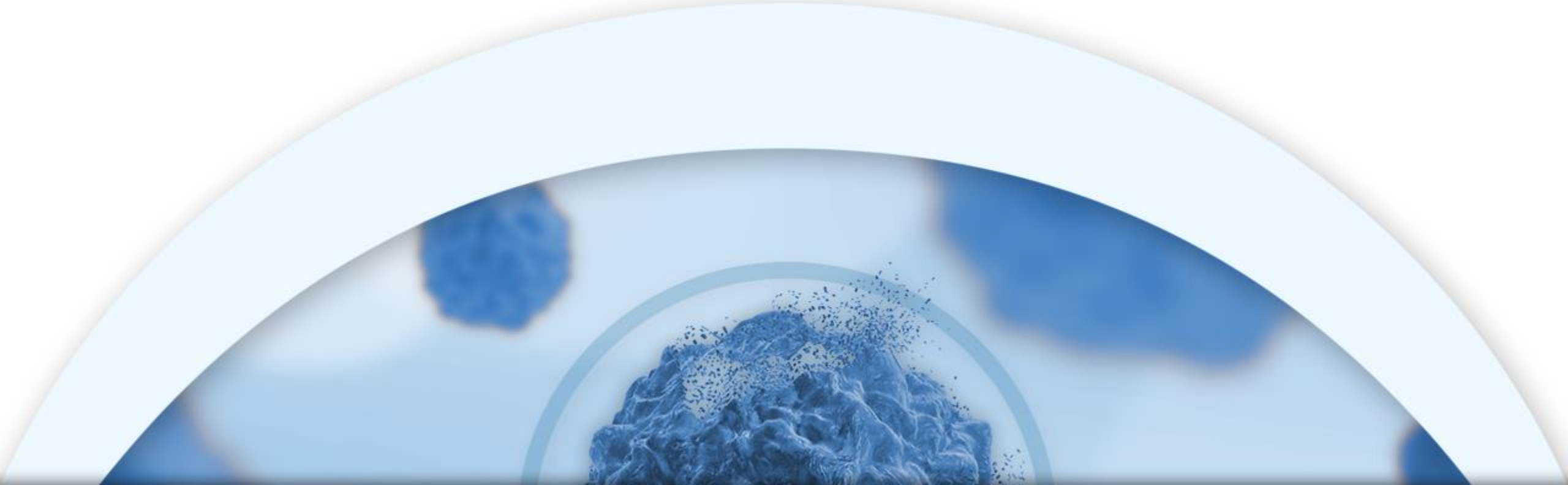
TakeAim Leukemia Update

December 12, 2022

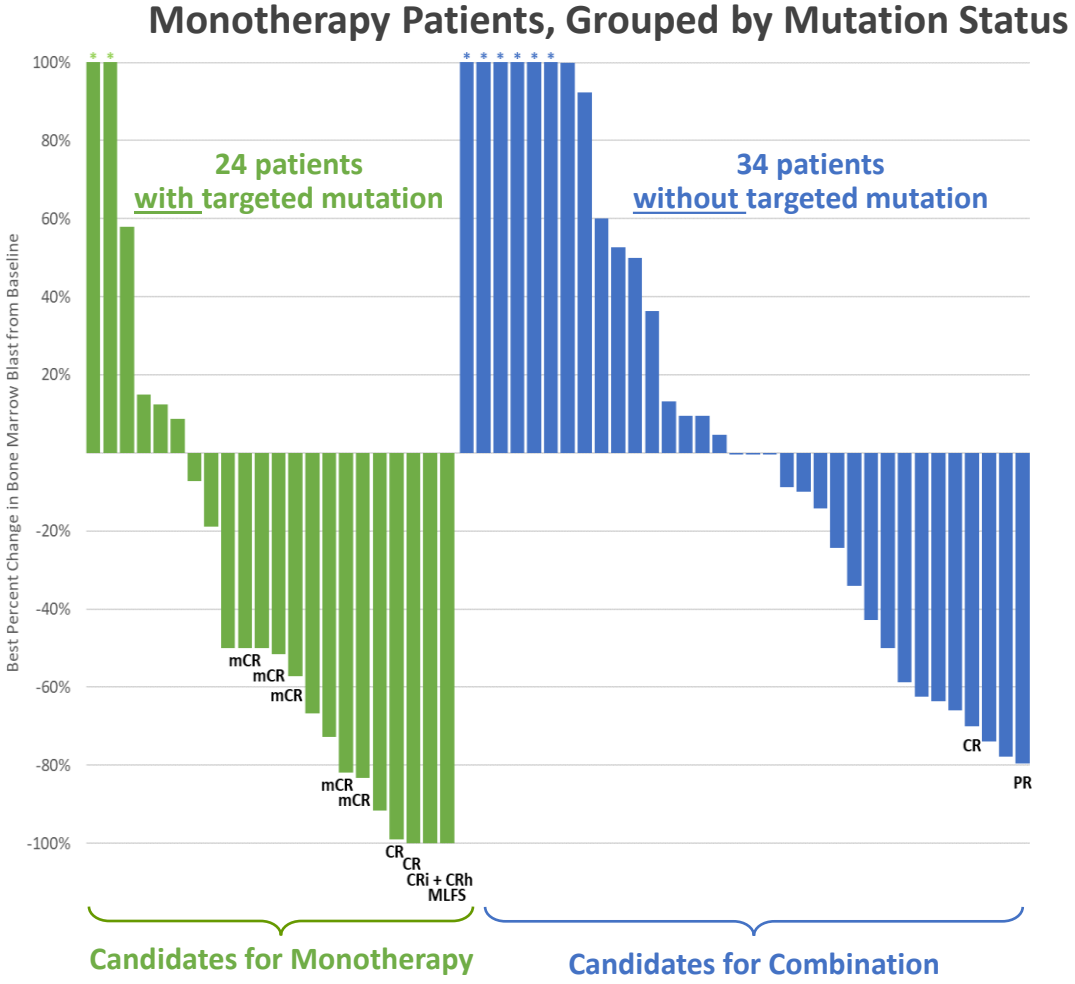


Cautionary Note Regarding Forward Looking Statements

This presentation contains certain forward-looking statements about Curis, Inc. (“we,” “us,” or the “Company”) within the meaning of the Private Securities Litigation Reform Act of 1995, as amended. Words such as “expect(s),” “believe(s),” “will,” “may,” “anticipate(s),” “focus(es),” “plans,” “mission,” “strategy,” “potential,” “estimate(s)”, "intend," "project," "seek," "should," "would" and similar expressions are intended to identify forward-looking statements. Forward-looking statements are statements that are not historical facts, reflect management’s expectations as of the date of this presentation, and involve important risks and uncertainties. Forward-looking statements herein include, but are not limited to, statements with respect to the timing and results of future clinical and pre-clinical milestones; the timing of future preclinical studies and clinical trials and results of these studies and trials; the clinical and therapeutic potential of our drug candidates; the proposed focus on emavusertib and management’s ability to successfully achieve its goals. These forward-looking statements are based on our current expectations and may differ materially from actual results due to a variety of important factors including, without limitation, risks relating to: whether and when the U.S. Food and Drug Administration may remove the partial clinical hold on the combination therapy phase (Phase 1b) and the expansion phase (Phase 2a) of the Phase 1/2 TakeAim Leukemia trial, or may take further regulatory action with regard to this trial, whether any of our drug candidates will advance further in the clinical development process and whether and when, if at all, they will receive approval from the FDA or equivalent foreign regulatory agencies; whether historical preclinical results will be predictive of future clinical trial results; whether historical clinical trial results will be predictive of future trial results; whether any of our drug candidate discovery and development efforts will be successful; whether any of our drug candidates will be successfully marketed if approved; our ability to achieve the benefits contemplated by our collaboration agreements; management’s ability to successfully achieve its goals; the sufficiency of our cash resources; our ability to raise additional capital to fund our operations on terms acceptable to us or the use of proceeds of any offering of securities or other financing; general economic conditions; competition; and the other risk factors contained in our periodic and interim reports filed with the Securities and Exchange Commission which are available on the SEC website at www.sec.gov. You are cautioned not to place undue reliance on these forward-looking statements that speak only as of the date hereof, and we do not undertake any obligation to revise and disseminate forward-looking statements to reflect events or circumstances after the date hereof, or to reflect the occurrence of or non-occurrence of any events, except as required by law.

Emavusertib Monotherapy Activity Reinforced in Updated Data

New data roughly doubles the targeted patient population (FLT3 or Spliceosome mutation)



Consistent, deep anticancer activity with a single agent

In Targeted Patients

Specific genetic mutations (FLT3, U2AF1, SF3B1) are the primary drivers of disease in this population; new data continue to show deep and durable responses

In Non-Targeted Patients

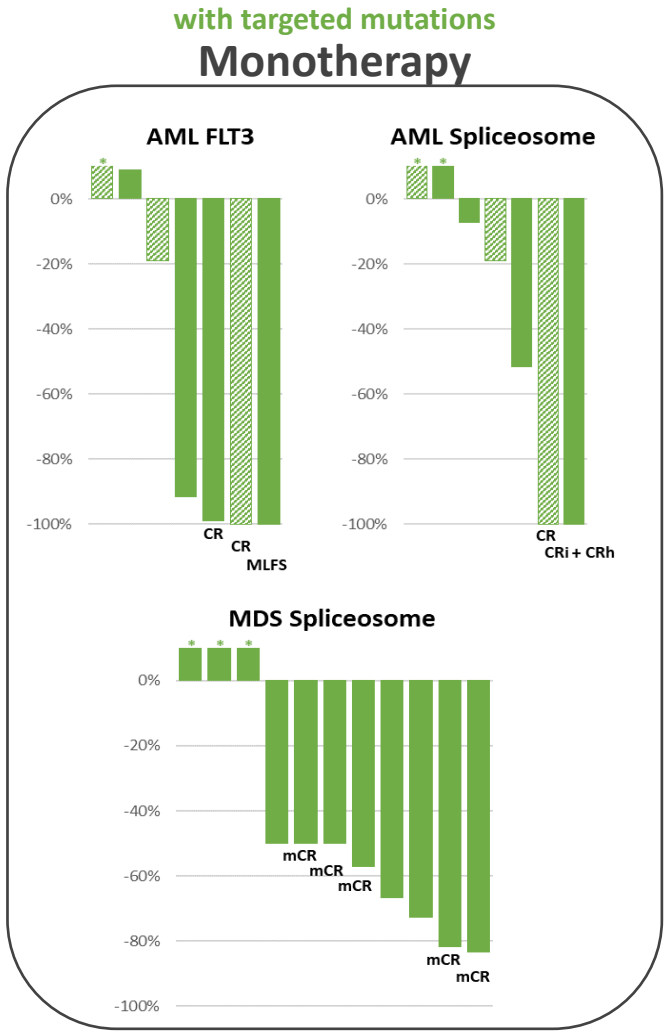
The majority of patients in this population have disease that harbors excess IRAK4-L; updated data continue to demonstrate emavusertib's monotherapy anticancer effects, suggesting potential to contribute independent anti-cancer activity when combined with other agents

** Indicates the graphic cutoff as 100%*
2 additional AML patients with spliceosome mutation had no post-treatment bone marrow assessment, but reported progressive disease and were considered as response evaluable

CURIS

Emavusertib Monotherapy Activity Reinforced in Updated Data

New data show consistent, deep anticancer activity with a single agent



In Patients with FLT3 Mutation

In this population, IRAK4 is a key driver of resistance to FLT3 inhibition; updated data show multiple deep and durable objective responses

Concomitant targeting of IRAK and FLT3 is the most effective means to overcome adaptive resistance incurred when targeting FLT3[1]

In Patients with Spliceosome Mutation

In this population, the primary driver of disease is a splicing factor mutation which causes excessive production of IRAK4-L; this population also represents a particularly high unmet need, as there are no approved therapies for R/R hrMDS

* *Indicates the graphic cutoff as 10%*

3 patients have both a FLT3 and spliceosome mutation and are included in both populations

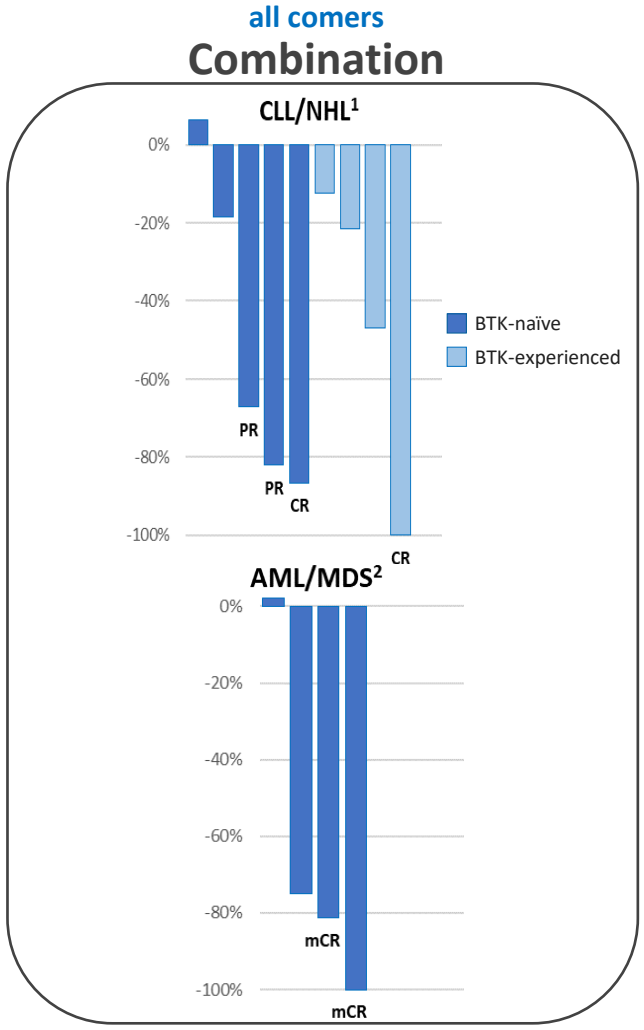
2 additional AML patients with spliceosome mutation had no post-treatment bone marrow assessment, but reported progressive disease and were considered as response evaluable

[1] *Melgar, Sci Transl Med. 2019*

CURIS

Initial Data Show Emavusertib is Highly Active in Combination

Initial combination data in AML/MDS are consistent with data seen in NHL/CLL



[1] *in combination with ibrutinib*
[2] *in combination with venetoclax*

Combination in NHL/CLL

Blocking both of the two pathways that drive overactivity of NFκB (BCR pathway and TLR pathway) achieves strong anti-cancer activity, including in patients previously treated with ibrutinib

BTKi targets BCR pathway
IRAK4i targets TLR pathway

Combination in AML/hrMDS

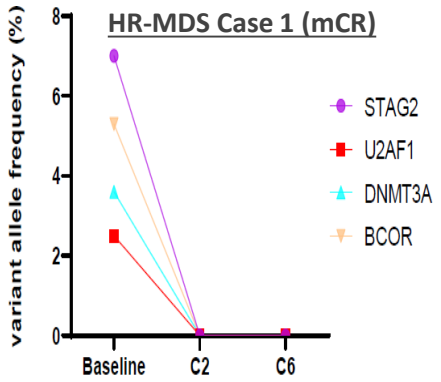
In AML/hrMDS, treatment resistance is dependent upon expression of anti-apoptotic factors such as MCL1 and BCL2; in initial data, combining emavusertib with venetoclax induced strong anti-cancer effect in patients

venetoclax targets BCL2
IRAK4i reduces MCL1

CURIS

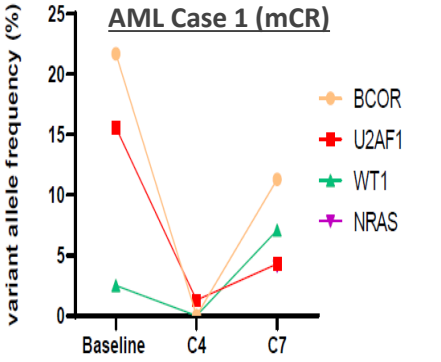
Emavusertib Induced Molecular Responses

Disease modifying activity in spliceosome-, FLT3- and dual-mutated disease



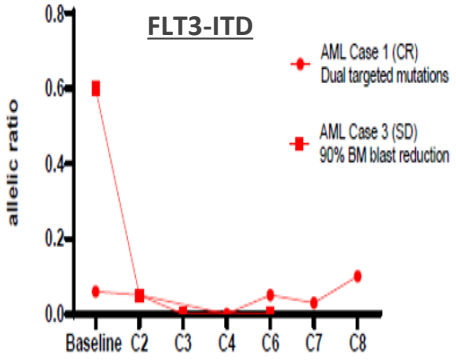
Patient with hrMDS harboring U2AF1 mutation

Demonstrates loss of not only the mutated splicing factor U2AF1, but also STAG2, DNMT3A and BCOR



Patient with AML harboring dual mutation (U2AF1 and FLT3)

Patient achieved CR with complete hematologic recovery, as well as loss of BCOR, U2AF1, WT1 and NRAS



FLT3 mutations by PCR

Demonstrates rapid loss of the FLT3-mutated clone in patient with dual mutation above, and a patient without targeted spliceosome mutation

CURIS