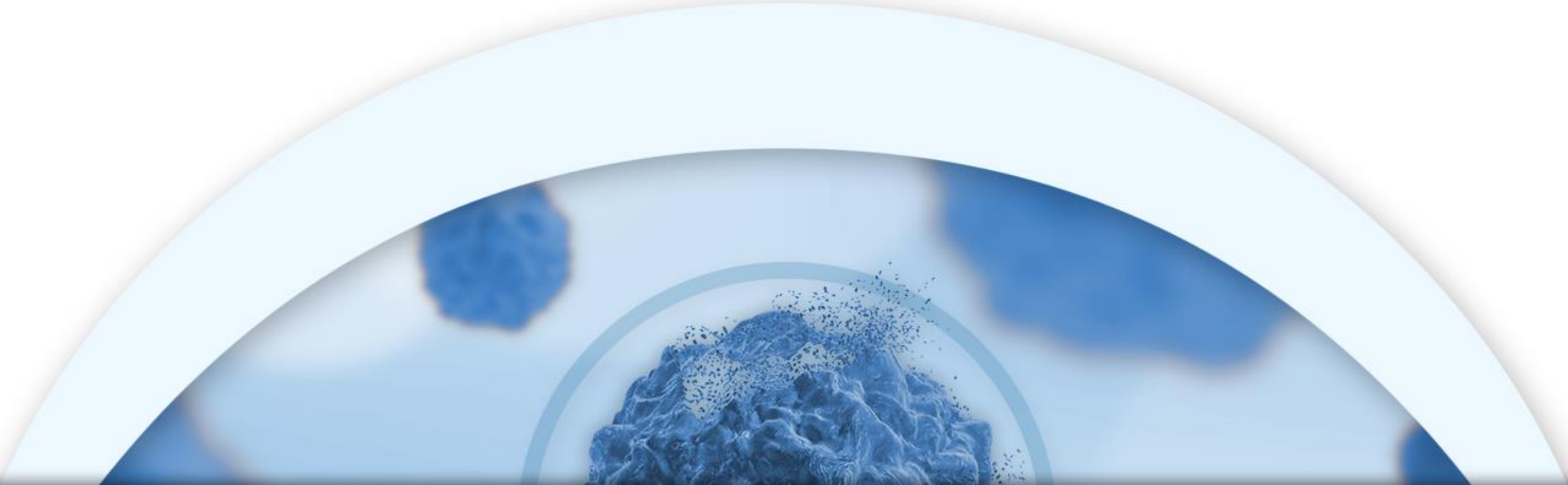
TakeAim Leukemia Update

December 12, 2022



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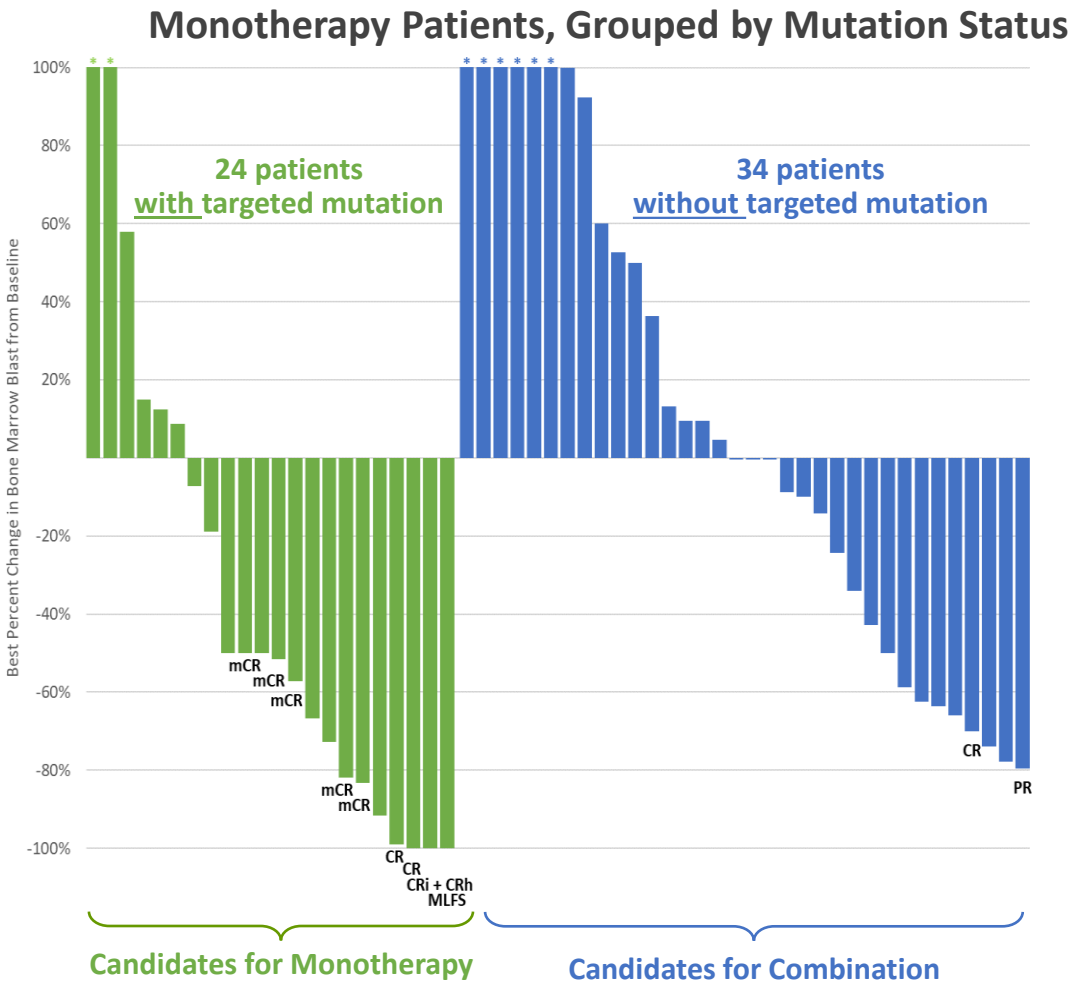
Emavusertib Monotherapy Activity Reinforced in Updated Data

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%*
22 AML patients with spliceosome mutation were response evaluable with baseline and post-treatment bone marrow assessments at data cutoff;
2 additional AML patients with spliceosome mutation had no post-treatment bone marrow assessment, but reported progressive disease and were also considered as response evaluable

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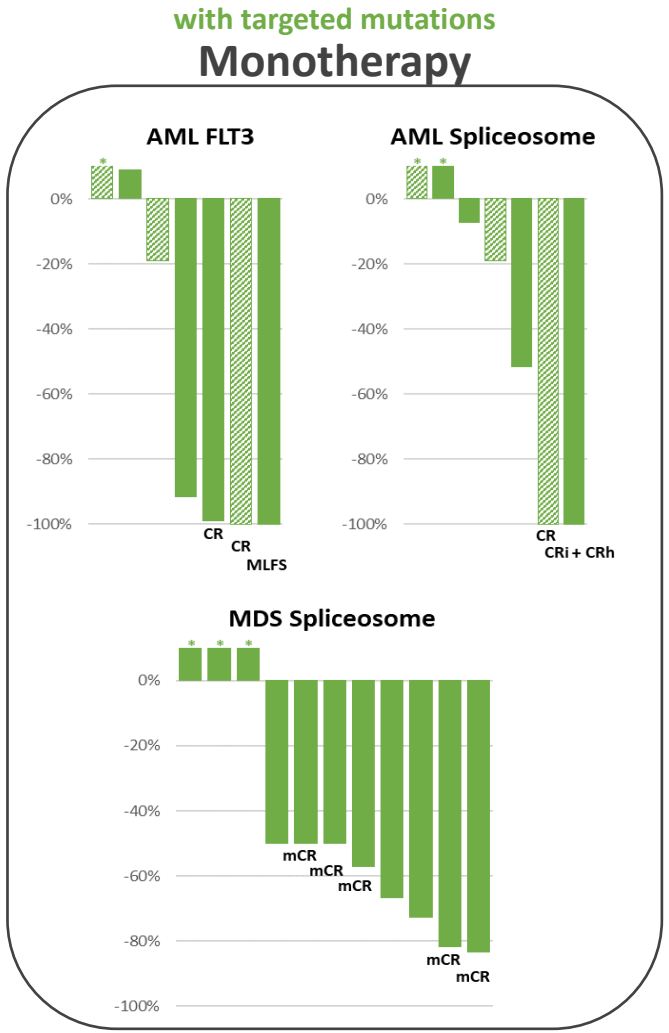
Emavusertib Monotherapy Activity Reinforced in Updated Data

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

7 AML patients with spliceosome mutation were response evaluable with baseline and post-treatment bone marrow assessments at data cutoff;

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

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

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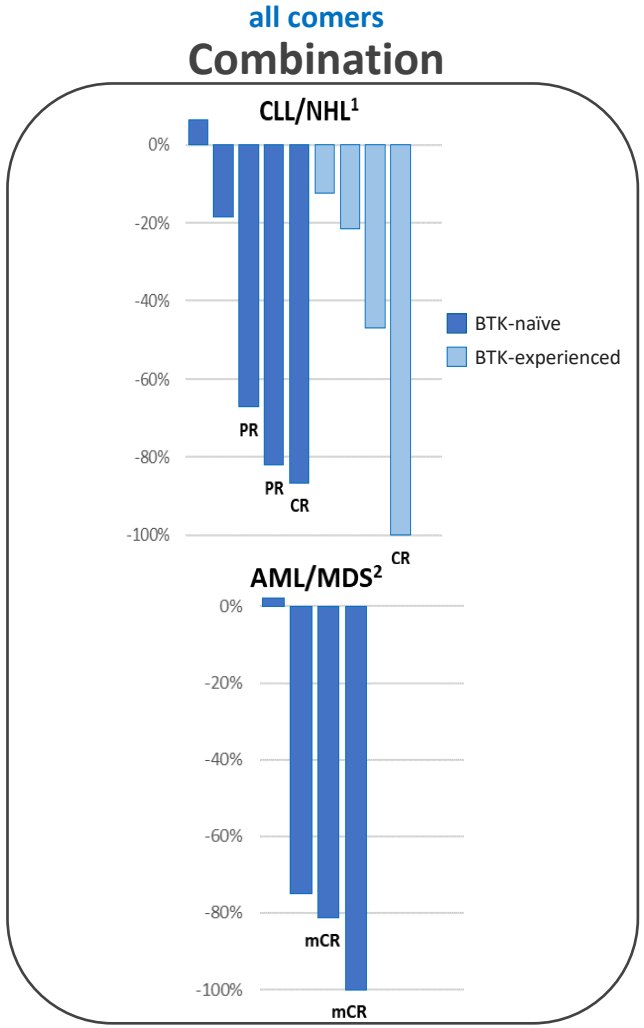
Initial Data Show Emavusertib is Highly Active in Combination

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

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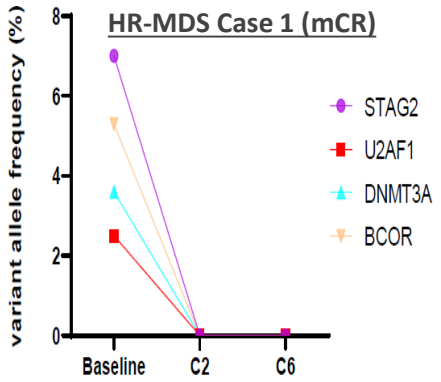
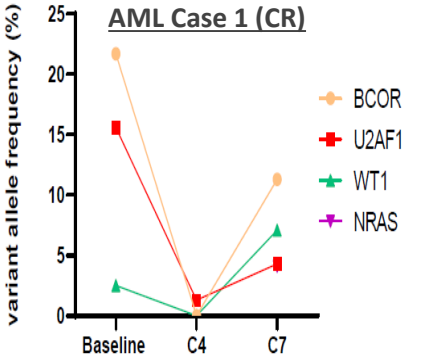
Emavusertib Induced Molecular Responses

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



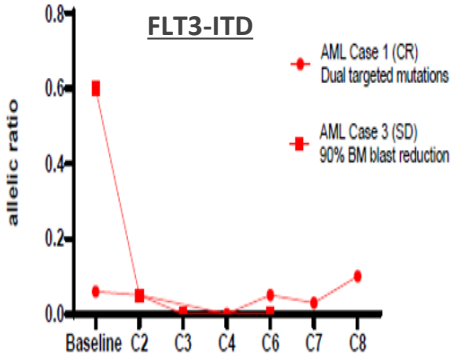
Patient with hrMDS harboring U2AF1 mutation

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

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