How effective is elranatamab as a treatment for multiple myeloma compared to other standard treatments?

The full title of this abstract is: An indirect comparison of elranatamab's progression-free survival, duration of response, and overall survival from MagnetisMM-3 versus real-world external control arms in triple-class refractory multiple myeloma

VIEW ABSTRACT

View Scientific Abstract >

Date of summary:
November 2024

Please note this summary only contains information from the scientific abstract. See a summary of the final poster presentation here.

Study number: NCT04649359

Study start date:
February 2021

Study end date:
December 2025

For more information on this study, go to:
https://www.clinicaltrials.gov/study/NCT04649359

KEY TAKEAWAY

What are the key takeaways from this study?

- People with multiple myeloma who received ELREXFIO™ **(elranatamab)** in the global **MagnetisMM-3** clinical study may have had better results from treatment than people who received standard treatments, based on information from a US electronic health record database (large collection of information about people receiving healthcare)
  - People who received **elranatamab** may have a longer response to treatment
  - People who received **elranatamab** may have had a lower risk of their disease getting worse
  - People who received **elranatamab** may have had a better chance of living longer

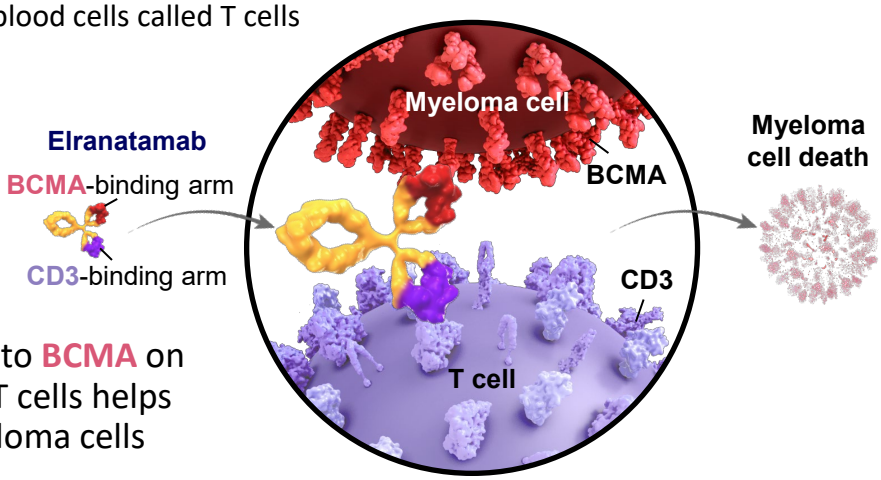
INTRODUCTION

What is elranatamab?

- **Elranatamab** is a medication being studied in patients with multiple myeloma who have received 3 other types of therapy including:
  - a proteasome inhibitor (such as bortezomib or carfilzomib)
  - an immunomodulatory medicine (such as lenalidomide or pomalidomide)
  - an anti-CD38 antibody (such as daratumumab or elotuzumab)
- In August of 2023, **elranatamab** gained **accelerated approval** from the US FDA for the treatment of adult patients with myeloma who have received at least 4 prior lines of therapy, including 1 of each of the therapy types listed above

INTRODUCTION

- **Elranatamab** works by attaching to 2 proteins known as BCMA and CD3
  - BCMA is found on plasma cells, including myeloma cells
  - CD3 is found on white blood cells called T cells



The binding of **elranatamab** to **BCMA** on myeloma cells and **CD3** on T cells helps activate T cells to kill myeloma cells

What does this summary describe?

- This summary describes an analysis that was done to compare how much myeloma improved after people received either **elranatamab** in the **MagnetisMM-3** study or other treatments commonly given to treat myeloma

Researchers wanted to find out…

- Does a person's myeloma improve when they receive **elranatamab**?
- Does a person's myeloma improve more when they receive **elranatamab** than when they receive other treatments commonly given in the US to treat myeloma?

STUDY DETAILS

Who took part in this study?

This study looked at people whose multiple myeloma was previously treated with and may have stopped responding to 3 types of medicines for myeloma:

- **Anti-CD38 antibodies**, which help the body's defense system (the immune system) to recognize and kill myeloma cells
- **Proteasome inhibitors**, which cause proteins to build up within the myeloma cells and cause these cells to die
- **Immunomodulatory medicines**, which stimulate or suppress the immune system to help the body fight cancer

These people have **triple-class exposed/refractory multiple myeloma**

Who took part in this study?

123 people with refractory or relapsed myeloma received **elranatamab** in the **MagnetisMM-3** clinical study. People were included or excluded from the study as described below:

Reasons people were included	Reasons for excluding people
Multiple myeloma diagnosis	Plasma cell leukemia
Able to take care of themselves	Smoldering multiple myeloma
Triple-class exposed/refractory multiple myeloma	Previous treatment with BCMA-directed therapies

These reasons for including or excluding people were applied to a US electronic health record database, called the **COTA database**, to get a comparable population (group of people similar enough to compare the results of treatment)

Patient records from the US **COTA database** were studied to see which treatments people received for **triple-class exposed/refractory multiple myeloma**

COTA database

Researchers then compared how well **elranatamab** worked in **MagnetisMM-3** to other standard treatments from the **COTA database** for **triple-class exposed/refractory multiple myeloma**

MagnetisMM-3 (123 people)

COTA database (577 people)

STUDY DETAILS

Statistical models were used to account for differences or imbalances in the patient populations between those in **MagnetisMM-3** and the **COTA database**

RESULTS

What were the results of this study?

Here are some of the similarities and differences between people in the **MagnetisMM-3** study and people in the US from the **COTA database**

	MagnetisMM-3 (123 people)	COTA database (577 people)
Age	67 years	67 years
Previous therapies	5 prior lines	5 prior lines
Male	55%	54%
Ability to take care of themselves	37%	31%
High-risk cytogenetics	25%	29%
Advanced disease	23%	29%
Extramedullary disease	31%	17%

RESULTS

What were the results of this study?

After adjusting for population differences, people treated with **elranatamab** were **more likely** to respond to their treatment and **less likely** to have their disease get worse than people who received treatments commonly used in the US

84%
less likely to have their myeloma stop responding to treatment

Duration of response
Myeloma in people who received **elranatamab** responded to treatment for a significantly longer time than myeloma in people who received **standard treatments**

62%
less likely to have their myeloma get worse

Progression-free survival
People who received **elranatamab** had a significantly longer time without their myeloma getting worse than people who received **standard treatments**

42%
lower risk of death while on treatment

Overall survival
People with multiple myeloma who received **elranatamab** lived for a significantly longer time than people who received **standard treatments**

CONCLUSIONS

What were the main conclusions of this study?

- **Elranatamab** may be better than the usual treatments used in the US for multiple myeloma
- People treated with **elranatamab** may have responded to treatment longer, lived longer without their disease getting worse, and may have lived longer overall

MORE INFORMATION

Who sponsored the study?

This study was sponsored by Pfizer Inc.
Pfizer Inc.
66 Hudson Blvd E
New York, NY 10001
Phone (United States): +1 212-733-2323
The sponsor thanks everyone who took part in this study.

- The information reported in this summary is based on the results of a single study
  - The results of this study may differ from those of other studies
  - Health professionals should make treatment decisions based on all available evidence, not just on the results of a single study
  - This study described is still ongoing; therefore, the final outcomes of this study may differ from the outcomes described in this summary

Where can I find more information?

For more information on this study, please visit:

View Scientific Abstract >

MagnetisMM-3: https://www.clinicaltrials.gov/study/NCT04649359

For more information on clinical trials in general, please visit:
https://www.clinicaltrials.gov/ct2/about-studies/learn
http://www.cancerresearchuk.org/about-cancer/find-a-clinical-trial/what-clinical-trials-are

Please note this summary only contains information from the scientific abstract. See a summary of the final poster presentation here.

Medical writing support for this summary was provided by Brittany Woodby, PhD, of Nucleus Global, and was funded by Pfizer.

PHONETICS

Find out how to say medical terms used in this summary

Antibody
<AN-tee-BAH-dee>

Cytogenetics
<SY-toh-jeh-NEH-tix>

Elranatamab
<ehl-RA-na-ta-mab>

Immunomodulatory
<IH-myoo-noh-MOD-you-lay-tory>

MagnetisMM
<MAG-nuh-ti-zuhm>

Myeloma
<MY-eh-LOH-muh>

Proteasome inhibitor
<PROH-tee-us-some in-HIH-bih-ter>

Refractory
<reh-FRAK-tor-ee>

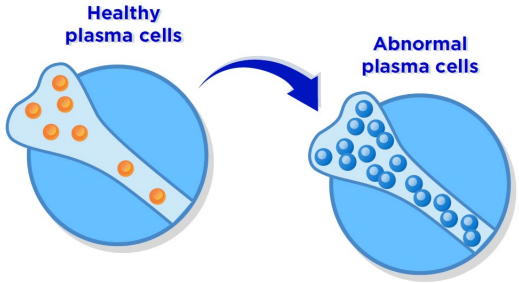
Relapsed
<REE-lapst>

GLOSSARY

accelerated approval: an official process that allows a new drug to be approved for use in the US
antibody: a protein the body's immune system makes to help fight infections
anti-CD38 antibodies: an immunotherapy protein that binds to the CD38 protein, which is abundant on the myeloma cell surface, and kills myeloma cells both directly and with the help of the immune system
BCMA: B-cell maturation antigen. A protein found on the surface of myeloma cells
BCMA-directed bispecific antibody: a monoclonal antibody that is engineered to simultaneously bind to two different cell surface proteins, one of which is BCMA
bone marrow: the soft, spongy tissue that is in most bones. This is where blood cells develop before moving into the bloodstream
CD3: a protein on the surface of bone marrow and blood cells that plays a key role in the immune system's response
cytogenetic risk: the risk of myeloma getting worse based on chromosomes (which contain DNA or genetic information)
elranatamab: a medicine being studied by researchers as a treatment for people with multiple myeloma. This medicine is a BCMA-directed bispecific antibody
extramedullary disease: the presence of myeloma cells outside of the bone marrow
immune system: the body's defense system. It helps fight infections and cancer
immunomodulatory medicines: medications that help the immune system fight diseases, such as cancer
multiple myeloma: a type of blood cancer that begins in the plasma cells
plasma cell: a type of white blood cell that makes large amounts of antibodies
proteasome inhibitors: medications that interfere with the cell's ability to break down and recycle unwanted proteins that cancer cells often accumulate excessively
refractory multiple myeloma: the state in which multiple myeloma does not respond or stops responding to treatment
relapsed multiple myeloma: the state in which the signs and symptoms of multiple myeloma reappear after a period of responding to therapy
standard of care: treatment that is accepted and widely used
triple-class exposed or refractory multiple myeloma: multiple myeloma that no longer responds to 3 different types of medicine for multiple myeloma, including **anti-CD38 antibodies, proteasome inhibitors**, and immunomodulatory medicines
T cell: a type of immune cell. T cells are part of the immune system and develop from stem cells in the bone marrow. They help protect the body from infection and may help fight cancer

What is multiple myeloma?

- Multiple myeloma is a blood cancer that affects a type of white blood cell known as a plasma cell in the bone marrow
  - Healthy plasma cells make proteins called antibodies that help fight infections
- Multiple myeloma leads to the buildup of abnormal plasma cells in the bone marrow, which can:
  - Stop the body from making normal numbers of healthy blood cells, often causing anemia (low red blood cells)
  - Make abnormal antibodies (also called M proteins)
  - Interfere with the normal function of kidneys and affect bone health



- At this time, multiple myeloma is not considered curable, but current treatments can often put the disease in remission for years
- Myeloma treatments can significantly reduce the number of myeloma cells, but in most patients, eventually it will start to grow again. When this happens, we say the disease has **relapsed** after treatment
- In some people with multiple myeloma, the cancer does not respond to treatment at all
  - This is known as **refractory** multiple myeloma