About Chronic Myeloid Leukemia

Overview of CML

If you have been diagnosed with chronic myeloid leukemia or are worried about it, you likely have a lot of questions. Learning some basics is a good place to start.

- What Is Chronic Myeloid Leukemia?

Research and Statistics

See the latest estimates for new cases of chronic myeloid leukemia and deaths in the US and what research is currently being done.

- What Are the Key Statistics About Chronic Myeloid Leukemia?
- What's New in Chronic Myeloid Leukemia Research and Treatment?

What Is Chronic Myeloid Leukemia?

Cancer starts when cells in the body begin to grow out of control. Cells in nearly any part of the body can become cancer, and can spread to other areas of the body. To learn more about how cancers start and spread, see What Is Cancer?

Chronic myeloid leukemia (CML), also known as chronic myelogenous leukemia, is a type of cancer that starts in certain blood-forming cells of the bone marrow. In CML, a genetic change takes place in an early (immature) version of myeloid cells - the cells that make red blood cells, platelets, and most types of white blood cells (except lymphocytes). This change forms an abnormal gene called BCR-ABL, which turns the cell into a CML cell. The leukemia cells grow and divide, building up in the bone marrow and spilling over into the blood. In time, the cells can also settle in other parts of the body, including the spleen. CML is a fairly slow growing leukemia, but it can also change into a fast-growing acute leukemia that is hard to treat.
Most cases of CML occur in adults, but very rarely it occurs in children, too. In general, their treatment is the same as for adults.

**What is leukemia?**

Leukemia is a cancer that starts in the blood-forming cells of the bone marrow. When one of these cells changes and becomes a leukemia cell, it no longer matures normally. Often, it divides to make new cells faster than normal. Leukemia cells also don't die when they should. This allows them to build up in the bone marrow, crowding out normal cells. At some point, leukemia cells leave the bone marrow and spill into the bloodstream, often causing the number of white blood cells in the blood to increase. Once in the blood, leukemia cells can spread to other organs, where they can prevent other cells in the body from functioning normally.

Leukemia is different from other types of cancer that start in organs such as the lungs, colon, or breast and then spread to the bone marrow. Cancers that start elsewhere and then spread to the bone marrow are not leukemia.

Not all leukemias are the same. Knowing the specific type of leukemia helps doctors better predict each patient’s prognosis (outlook) and select the best treatment.

**What is a chronic leukemia?**

Whether leukemia is *acute* or *chronic* depends on whether most of the abnormal cells are immature (and are more like stem cells) or mature (and are more like normal white blood cells).

In chronic leukemia, the cells can mature partly but not completely. These cells may look fairly normal, but they are not. They generally do not fight infection as well as normal white blood cells do. The leukemia cells survive longer than normal cells, and build up, crowding out normal cells in the bone marrow. Chronic leukemias can take a long time before they cause problems, and most people can live for many years. But chronic leukemias are generally harder to cure than acute leukemias.

**What is a myeloid leukemia?**

Whether leukemia is *myeloid* or *lymphocytic* depends on which bone marrow cells the cancer starts in.
Myeloid leukemias (also known as *myelocytic, myelogenous, or non-lymphocytic* leukemias) start in early myeloid cells -- the cells that become white blood cells (other than lymphocytes), red blood cells, or platelet-making cells (megakaryocytes).

**What are the other types of leukemia?**

There are 4 main types of leukemia, based on whether they are acute or chronic, and myeloid or lymphocytic:

- **Acute myeloid (or myelogenous) leukemia (AML)**
- **Chronic myeloid (or myelogenous) leukemia (CML)**
- **Acute lymphocytic (or lymphoblastic) leukemia (ALL)**
- **Chronic lymphocytic leukemia (CLL)**

In *acute* leukemias, the bone marrow cells cannot mature the way they should. These immature cells continue to reproduce and build up. Without treatment, most people with acute leukemia would only live a few months. Some types of acute leukemia respond well to treatment, and many patients can be cured. Other types of acute leukemia have a less favorable outlook. *Lymphocytic* leukemias (also known as lymphoid or lymphoblastic leukemia) start in the cells that become lymphocytes. Lymphomas are also cancers that start in those cells. The main difference between lymphocytic leukemias and lymphomas is that in leukemia, the cancer cell is mainly in the bone marrow and blood, while in lymphoma it tends to be in lymph nodes and other tissues.

Chronic myelomonocytic leukemia (CMML) is another chronic leukemia that starts in myeloid cells. For more information on this type of cancer, see [Chronic Myelomonocytic Leukemia](#).

**The rest of this information is only about chronic myeloid leukemia (CML).**

- **References**

  See all references for Chronic Myeloid Leukemia

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Normal Bone Marrow and Blood

Different types of leukemia are formed from different types of cells. To understand the different types of leukemia, it helps to have some basic knowledge about the blood and lymph systems. The information which follows is quite complex. It may prove helpful, but you don't need to understand all of it to learn more about your leukemia.

Bone marrow

Bone marrow is the soft inner part of some bones such as the skull, shoulder blades, ribs, pelvis, and backbones. Bone marrow is made up of a small number of blood stem cells, more mature blood-forming cells, fat cells, and supporting tissues that help cells grow.

Inside the bone marrow, blood stem cells develop into new blood cells. During this process, the cells become either lymphocytes (a kind of white blood cell) or other blood-forming cells. These blood-forming cells can develop red blood cells, white blood cells (other than lymphocytes), or platelets.

Types of Blood cells

Red blood cells carry oxygen from the lungs to all other tissues in the body, and take carbon dioxide back to the lungs to be removed. Having too few red blood cells in the body (anemia) can make you feel tired, weak, and short of breath because your body tissues are not getting enough oxygen.

Platelets are actually cell fragments made by a type of bone marrow cell called the megakaryocyte. Platelets are important in plugging up holes in blood vessels caused by cuts or bruises. Having too few platelets (thrombocytopenia) may cause you to bleed or bruise easily.

White blood cells help the body fight infections. Having too few white blood cells (neutropenia) lowers your immune system and can make you more likely to get an infection.

Types of white blood cells

Lymphocytes are mature, infection-fighting cells that develop from lymphoblasts, a
type of blood stem cell in the bone marrow. Lymphocytes are the main cells that make up lymphoid tissue, a major part of the immune system. Lymphoid tissue is found in lymph nodes, the thymus gland, the spleen, the tonsils and adenoids, and is scattered throughout the digestive and respiratory systems and the bone marrow. The 2 major types of lymphocytes are known as B lymphocytes (B cells) and T lymphocytes (T cells). Lymphocytes help protect the body from germs. Some types of lymphocytes help regulate the immune system.

**Granulocytes** are mature, infection-fighting cells that develop from *myeloblasts*, a type of blood forming cell in the bone marrow. Granulocytes have granules that show up as spots under the microscope. These granules contain enzymes and other substances that can destroy germs, such as bacteria. The 3 types of granulocytes -- *neutrophils*, *basophils*, and *eosinophils* -- are distinguished under the microscope by the size and color of their granules. Neutrophils are the most common type of granulocyte in the blood. They are essential in destroying bacteria that have invaded the blood.

**Monocytes** develop from blood-forming *monoblasts* in the bone marrow and are related to granulocytes. After circulating in the bloodstream for about a day, monocytes enter body tissues to become *macrophages*, which can destroy some germs by surrounding and digesting them. Macrophages also help lymphocytes recognize germs and start making antibodies to fight them.

- **References**

  See all references for Chronic Myeloid Leukemia

What Are the Key Statistics About Chronic Myeloid Leukemia?

The American Cancer Society's estimates for chronic myeloid leukemia (CML) in the United States for 2017 are:

- About 8,950 new cases will be diagnosed with CML (5,230 in men and 3,720 in...
• About 1,080 people will die of CML (610 men and 470 women).

A little over 10% of all new cases of leukemia are chronic myeloid leukemia. About 1 person in 555 will get CML in their lifetime in the United States.

The average age at diagnosis of CML is around 64 years. Almost half of cases are diagnosed in people 65 and older. This type of leukemia mainly affects adults, and is only rarely seen in children.

Visit the American Cancer Society’s Cancer Statistics Center for more key statistics.

• References
See all references for Chronic Myeloid Leukemia

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What's New in Chronic Myeloid Leukemia Research and Treatment?

Studies of chronic myeloid leukemia (CML) are being done in labs and in clinical trials around the world.

Genetics of chronic myeloid leukemia

Scientists are making great progress in understanding how changes in a person's DNA can cause normal bone marrow cells to develop into leukemia cells. Learning about changes in the genes (regions of the DNA) involved in CML is providing insight into why these cells grow too quickly, live too long, and fail to develop into normal blood cells. The explosion of knowledge in recent years is being used to develop many new drugs.

Treatment
Combining the targeted drugs with other treatments

Imatinib and other drugs that target the BCR-ABL protein have proven to be very effective, but by themselves these drugs don't help everyone. Studies are now in progress to see if combining these drugs with other treatments, such as chemotherapy, interferon, or cancer vaccines (see below) might be better than either one alone. One study showed that giving interferon with imatinib worked better than giving imatinib alone. The 2 drugs together had more side effects, though. It is also not clear if this combination is better than treatment with other tyrosine kinase inhibitors (TKIs), such as dasatinib and nilotinib. A study going on now is looking at combing interferon with nilotinib.

Other studies are looking at combining other drugs, such as cyclosporine or hydroxychloroquine, with a TKI.

New drugs for CML

Because researchers now know the main cause of CML (the BCR-ABL gene and its protein), they have been able to develop many new drugs that might work against it.

In some cases, CML cells develop a change in the BCR-ABL oncogene known as a T315I mutation, which makes them resistant to many of the current targeted therapies (imatinib, dasatinib, and nilotinib). Ponatinib is the only TKI that can work against T315I mutant cells. More drugs aimed at this mutation are now being tested.

Other drugs called farnesyl transferase inhibitors, such as lonafarnib and tipifarnib, seem to have some activity against CML and patients may respond when these drugs are combined with imatinib. These drugs are being studied further.

Other drugs being studied in CML include the histone deacetylase inhibitor panobinostat and the proteasome inhibitor bortezomib (Velcade).

Cancer vaccines

Cancer cells are different from normal cells, so it is sometimes possible to get the body's immune system to react against them. One way to do this is to use a cancer vaccine -- a substance injected into the body that boosts the immune system and causes it to attack certain cells. Several vaccines are now being studied for use against CML. For instance, in one small study, a vaccine called CMLVAX100 was given along with imatinib and seemed to increase its effectiveness. Research into this and other vaccines is continuing.