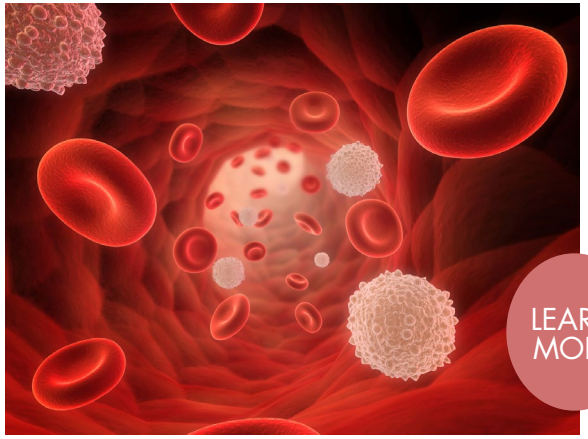
Chronic Myeloid Leukemia



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is a cancer that occurs when the blood-forming cells of the bone marrow (the soft, spongy tissue in the center of bones) make too many white blood cells, including immature ones.[3]

What happens every...

Once a devastating diagnosis, deaths from CML have declined sharply over the past decades.[4,5] While the number of people diagnosed each year stays relatively constant, more and more people are now living with the disease due to advances in treatment.[2]

1975

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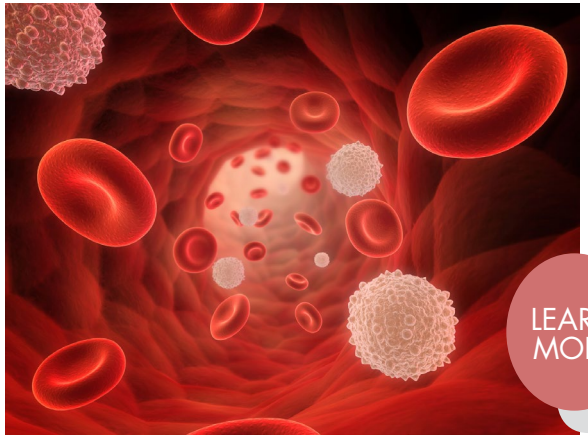

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LEARN MORE

Ph+ CML is caused by a genetic abnormality that produces an abnormal chromosome in bone marrow stem cells—the Philadelphia chromosome.[3]

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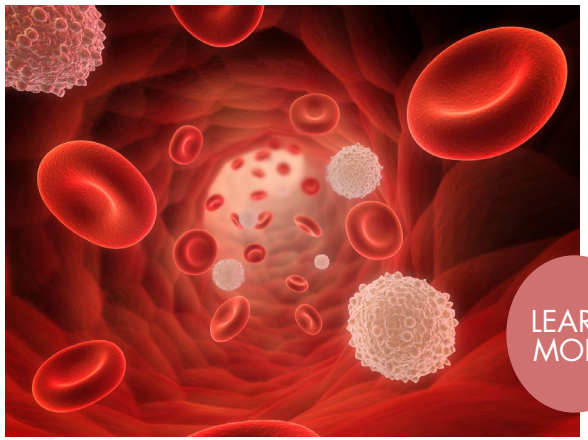

LEARN MORE

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What happens every...

Around the world, 3 people are diagnosed with and 2 people die from a blood cancer every 2 minutes.[1]

Click

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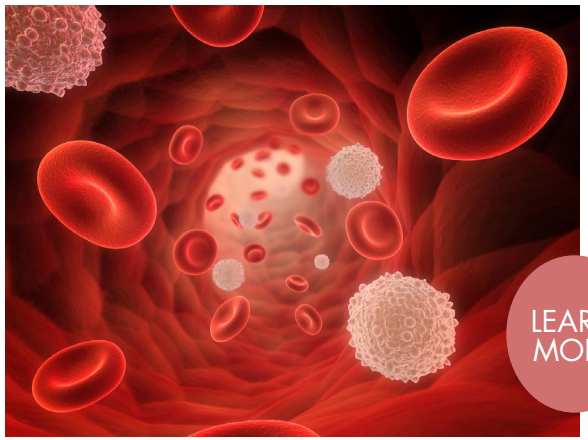

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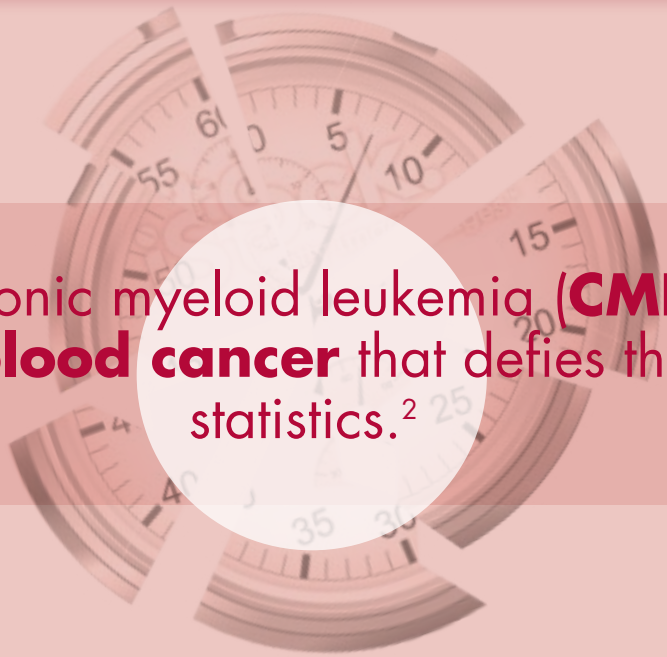

Chronic myeloid leukemia (**CML**) is a **blood cancer** that defies these statistics.[2]

Once a devastating diagnosis, deaths from CML have declined sharply over the past decades.[4,5] While the number of people diagnosed each year stays relatively constant, more and more people are now living with the disease due to advances in treatment.[2]

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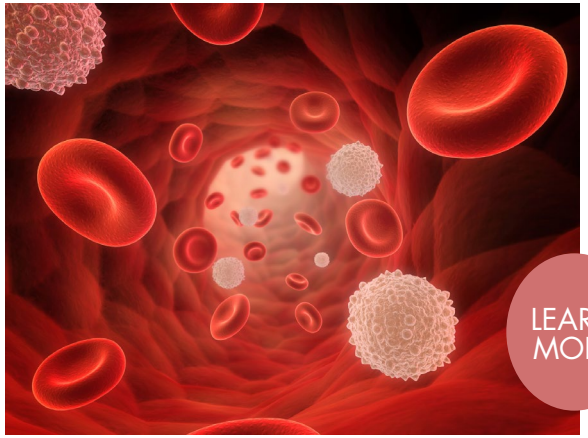

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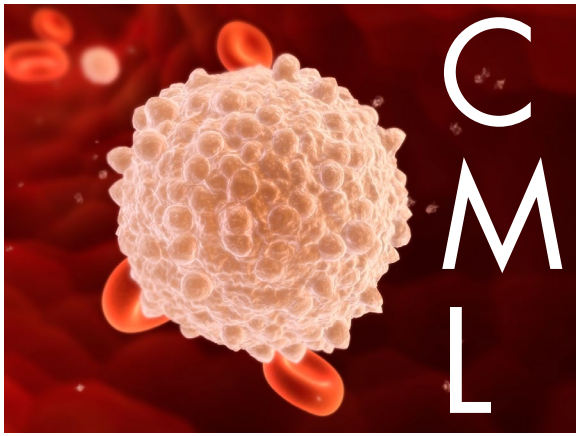
In The Emperor of All Maladies: A Biography of Cancer, Dr. Siddhartha Mukherjee, 2011 Pulitzer Prize winner, wrote, "[In 10 years] each of us, on average, will know one person with this leukemia [chronic myeloid leukemia] who is being kept alive by a targeted anticancer drug."[2]

1. Ferlay J, et al. Int J of Cancer. 2010;127:2893-2917. 2. Mukherjee, Siddhartha. The Emperor of All Maladies: A Biography of Cancer. New York: Scribner, 2010. Print. 3. National Cancer Institute. General Information About Chronic Myelogenous Leukemia. National Institutes of Health. http://www.cancer.gov/cancertopics/pdq/treatment/CML/Patient/page1. Accessed May 2014. 4. Centers for Disease Control and Prevention. Vital Statistics Data Available Online: Mortality Multiple Cause. http://www.cdc.gov/nchs/data_access/vitalstatsonline.htm. Accessed May 2014. 5. Surveillance Epidemiology and End Results. SEER Stat Fact Sheets: Chronic Myeloid Leukemia. National Cancer Institute. http://archive.is/zNlo. Accessed May 2014. 6. American Cancer Society. Cancer Facts & Figures 2014. http://www.cancer.org/acs/groups/content/@research/documents/webcontent/acspc-042151.pdf. Accessed May 2014.



Chronic Myeloid Leukemia

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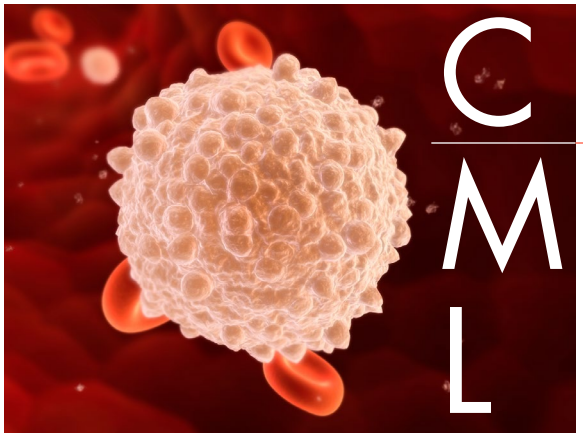

Chronic Myeloid Leukemia

Chronic means a relatively slower-growing cancer that may take years to progress.[1]

DID YOU KNOW ?

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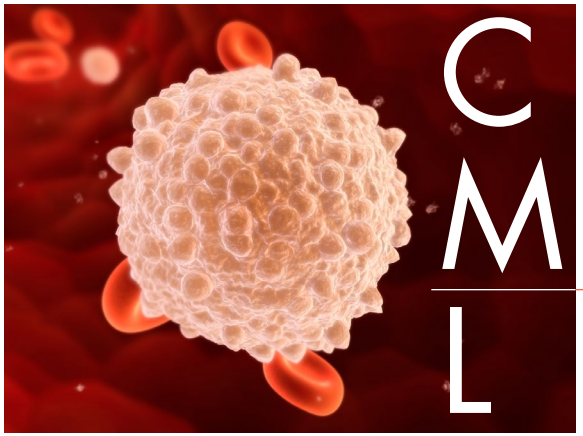

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Myeloid (or myelogenous) refers to the type of white blood cell being overproduced.[1]

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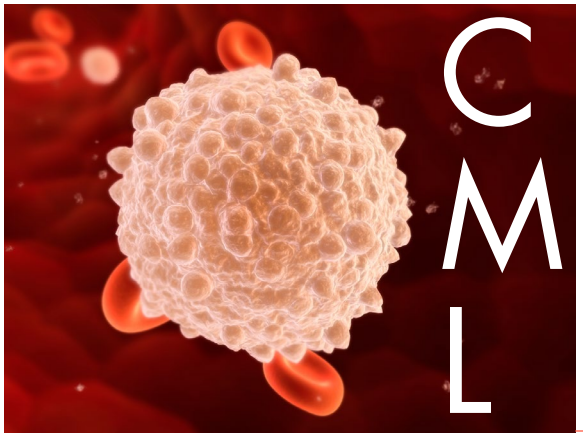

Chronic Myeloid Leukemia

Leukemia is a cancer of the blood and bone marrow. With leukemia, bone marrow stem cells are abnormal, or defective, which ultimately leads to excessive amounts (overproduction) of abnormal white blood cells.[1]

DID YOU KNOW ?

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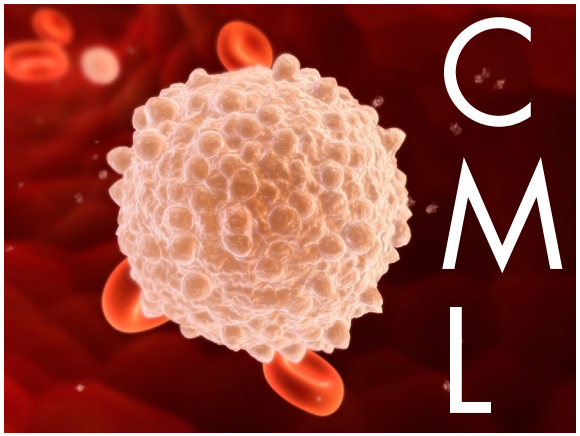
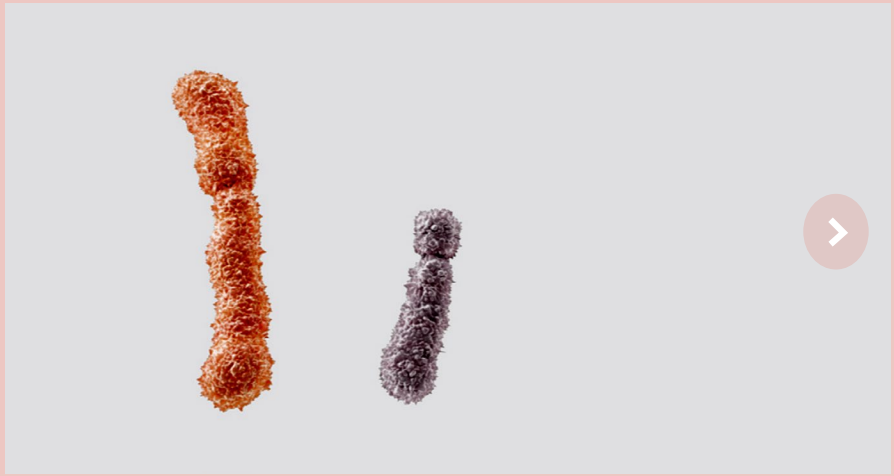


Chronic Myeloid Leukemia

DID YOU KNOW ?

CML is characterized in three phases: **chronic phase** (CP), **accelerated phase** (AP) and a **terminal blast phase** (BP).[2] Most patients find out they have CML in the early, chronic phase and will remain in this phase for a number of years without progressing to a more advanced phase.[2,3] If CML is left untreated, progression from CP to BP usually occurs in three to five years.[4]

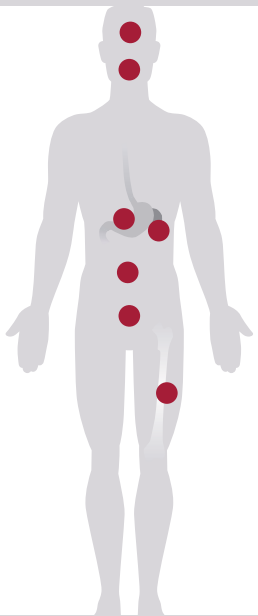
1. National Cancer Institute. General Information About Chronic Myelogenous Leukemia. National Institutes of Health. http://www.cancer.gov/cancertopics/pdq/treatment/CML/Patient/page1. Accessed May 2014. 2. American Cancer Society. Detailed Guide: CML. http://www.cancer.org/acs/groups/cid/documents/webcontent/003112-pdf.pdf. Accessed May 2014. 3. The NCCN Chronic Myelogenous Leukemia Clinical Practice Guidelines in Oncology (Version 3.2014). © 2014 National Comprehensive Cancer Network, Inc. http://www.nccn.org. Accessed May 2014. 4. Goldberg S, et al. Community Oncology. 2009;6(3):113-125. 5. Genetics Home Reference. How many chromosomes do people have? National Institutes of Health. http://ghr.nlm.nih.gov/handbook/basics/howmanychromosomes. Accessed May 2014. 6. Moore FR, et al. Methods Mol Biol. 2013;999:1-23.



Your DNA has 46 chromosomes.[5] In Ph+ CML, pieces of chromosomes 9 and 22 have broken off and switched places.[1] This creates a new abnormal chromosome called the Philadelphia chromosome, abbreviated "Ph chromosome" or simply "Ph."[1]

During the early stages of Ph+ CML, some patients show no signs or symptoms of the disease, sometimes for many years. When symptoms do develop, they may include:[2]

Click dots to reveal symptoms



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The Ph chromosome carries a gene called BCR-ABL, which produces a protein called BCR-ABL.[6] The Bcr-Abl protein triggers bone marrow to keep making abnormal versions of white blood cells, which are the leukemia cells.[6] The BCR-ABL gene and Bcr-Abl protein are the key causes of Ph+ CML in 95% of patients.[6]

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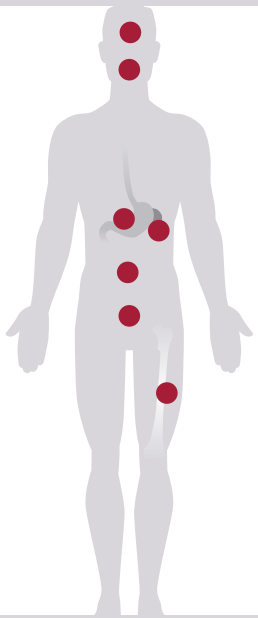

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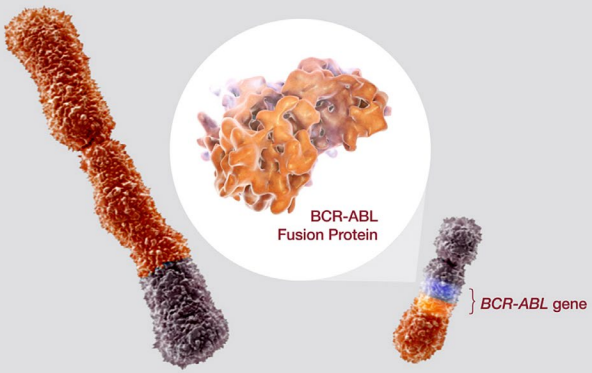

The resulting uncontrolled growth of these white blood cells causes a large increase in their concentration in the blood.[1] Over time, these white blood cells crowd out healthy red blood cells and platelets, as well as normal white blood cells, which can have negative effects on a Ph+ CML patient's health.[1]

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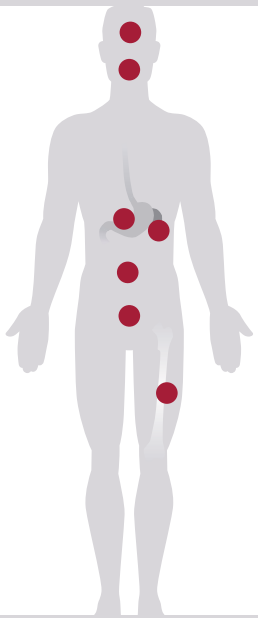

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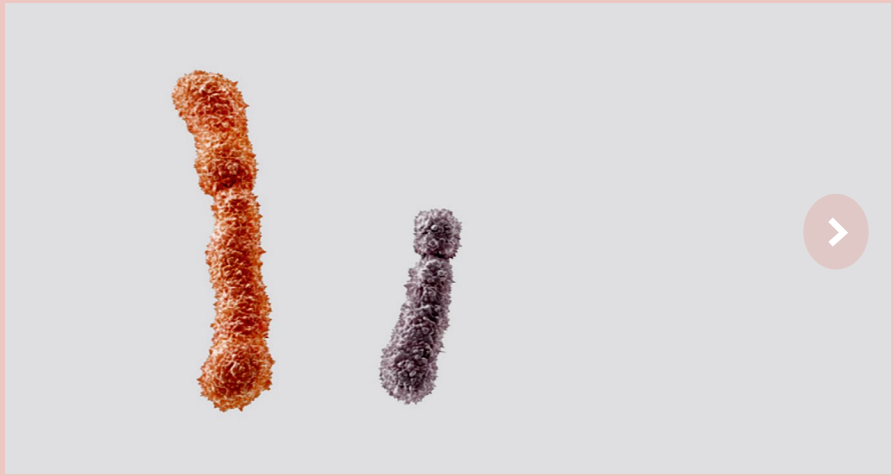

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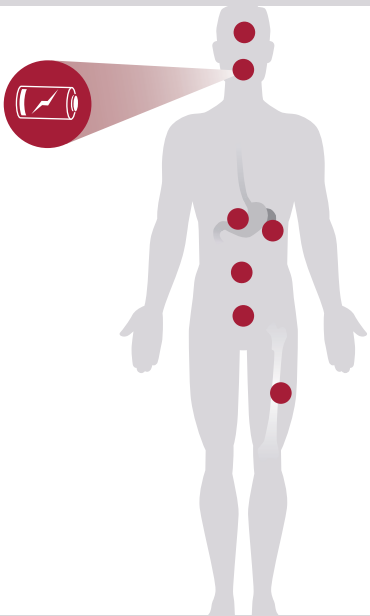
Tiredness or fatigue that will not go away due to low red blood cell counts



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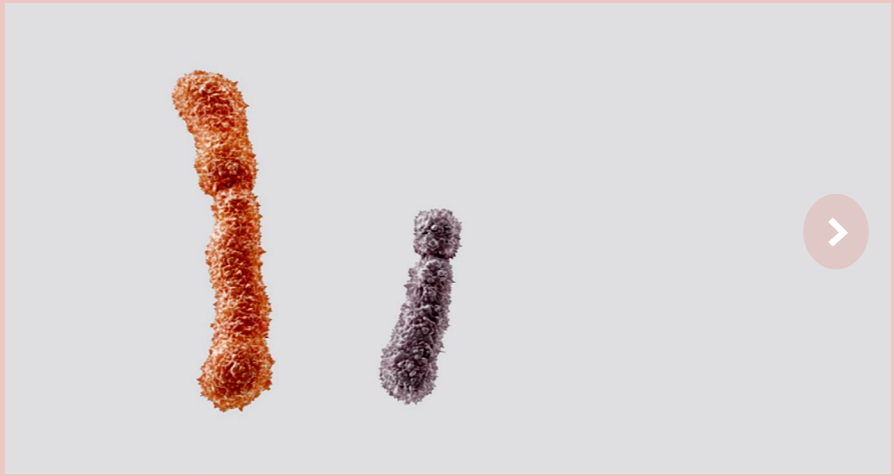

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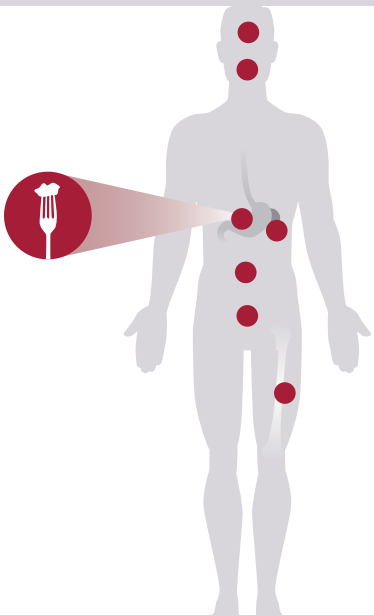
Feeling full after eating even a small amount of food



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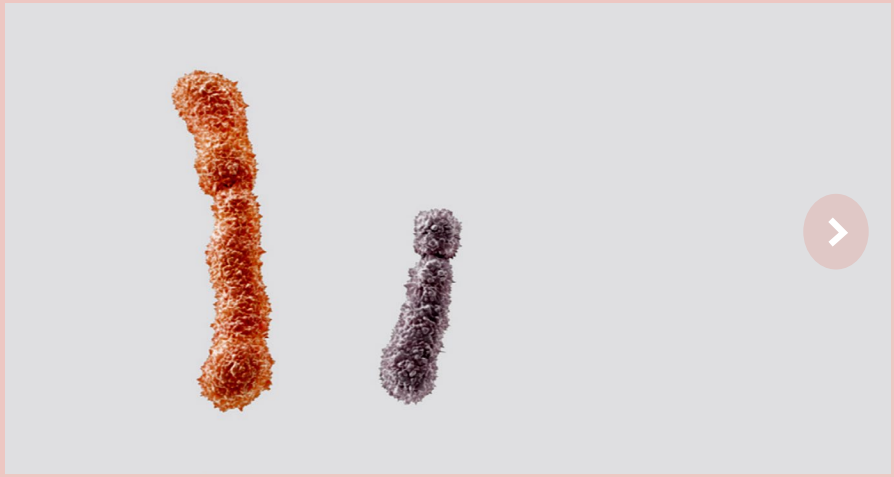

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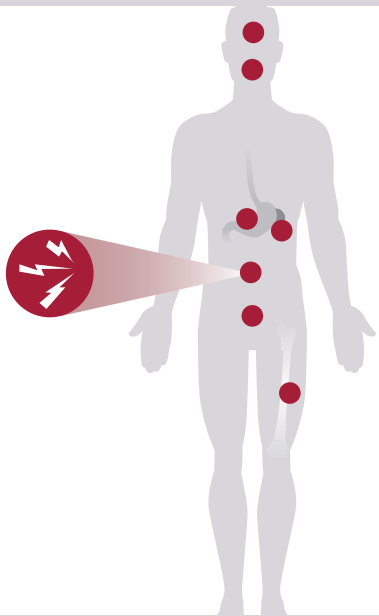
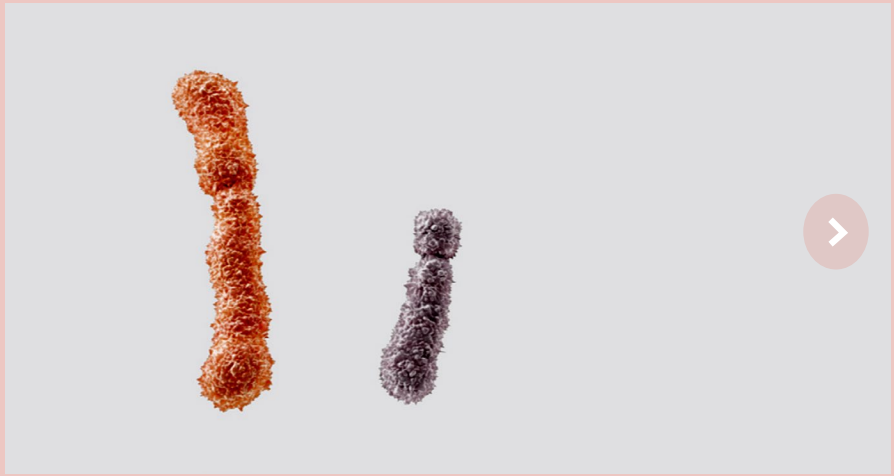
Click dots to reveal symptoms

Pain or a sense of "fullness" in the belly



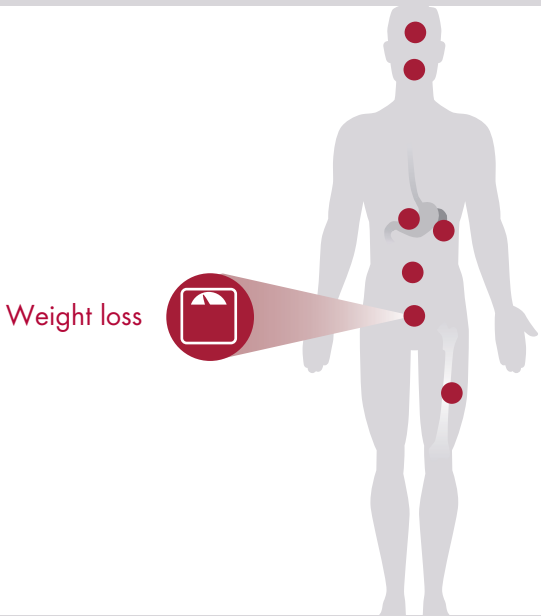
1. National Cancer Institute. General Information About Chronic Myelogenous Leukemia. National Institutes of Health. http://www.cancer.gov/cancertopics/pdq/treatment/CML/Patient/page1. Accessed May 2014. 2. American Cancer Society. Detailed Guide: CML. http://www.cancer.org/acs/groups/cid/documents/webcontent/003112-pdf.pdf. Accessed May 2014. 3. The NCCN Chronic Myelogenous Leukemia Clinical Practice Guidelines in Oncology (Version 3.2014). © 2014 National Comprehensive Cancer Network, Inc. http://www.nccn.org. Accessed May 2014. 4. Goldberg S, et al. Community Oncology. 2009;6(3):113-125. 5. Genetics Home Reference. How many chromosomes do people have? National Institutes of Health. http://ghr.nlm.nih.gov/handbook/basics/howmanychromosomes. Accessed May 2014. 6. Moore FR, et al. Methods Mol Biol. 2013;999:1-23.



Your DNA has 46 chromosomes.[5] In Ph+ CML, pieces of chromosomes 9 and 22 have broken off and switched places.[1] This creates a new abnormal chromosome called the Philadelphia chromosome, abbreviated "Ph chromosome" or simply "Ph."[1]

During the early stages of Ph+ CML, some patients show no signs or symptoms of the disease, sometimes for many years. When symptoms do develop, they may include:[2]

Click dots to reveal symptoms



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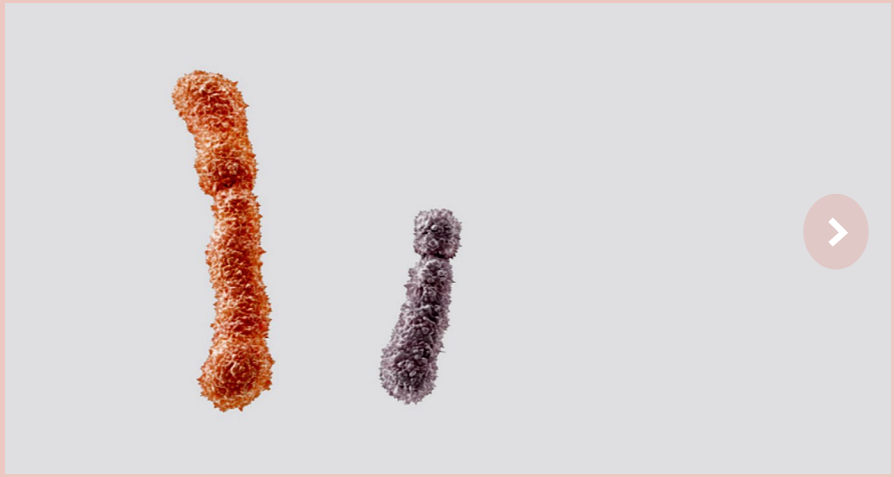

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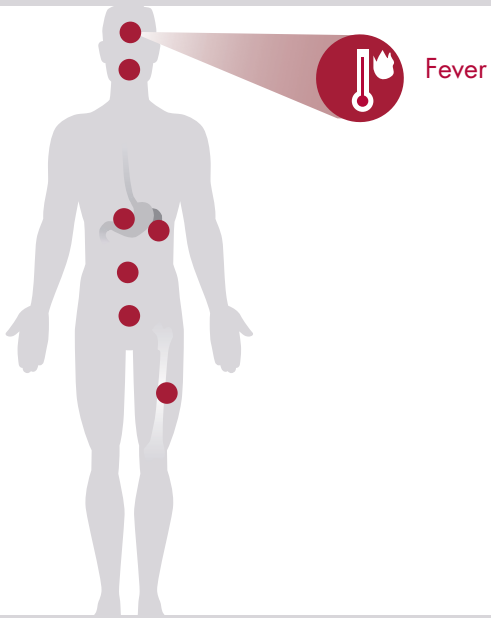

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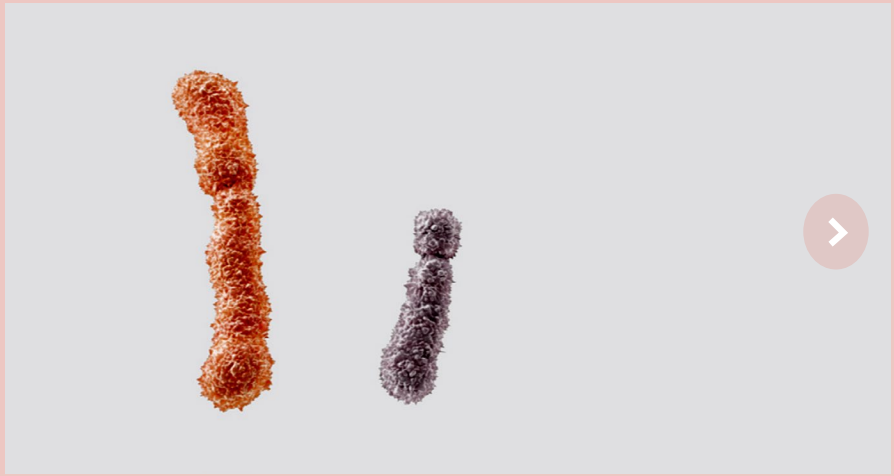

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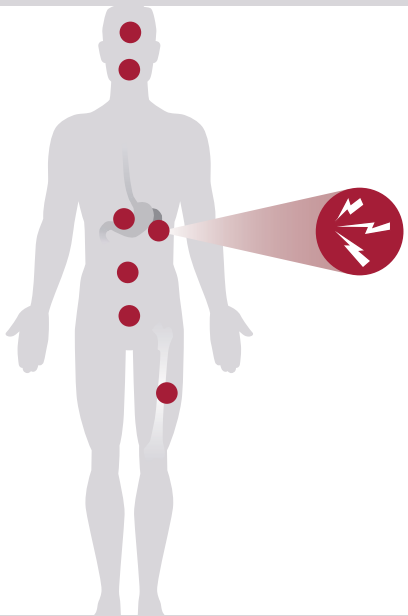

Pain under the left ribs from enlarged spleen (felt as a mass under the left side of the ribcage)

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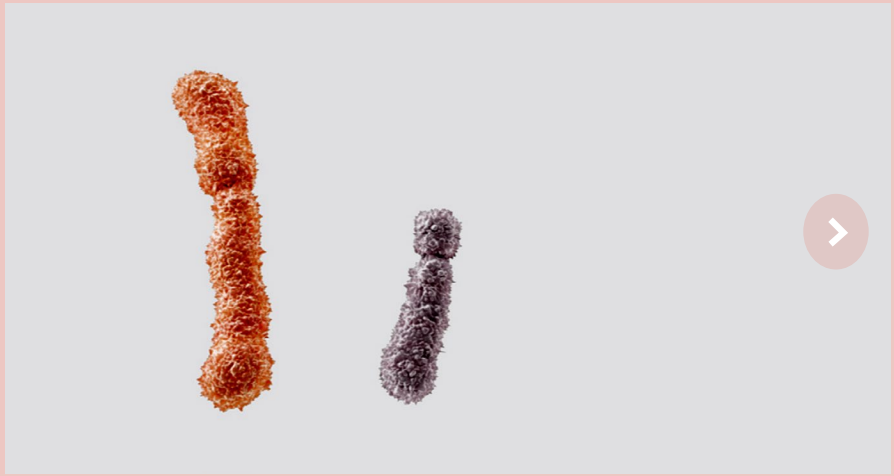

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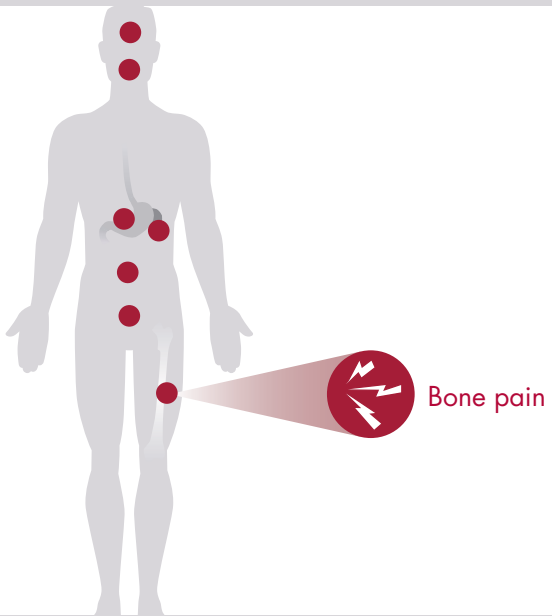

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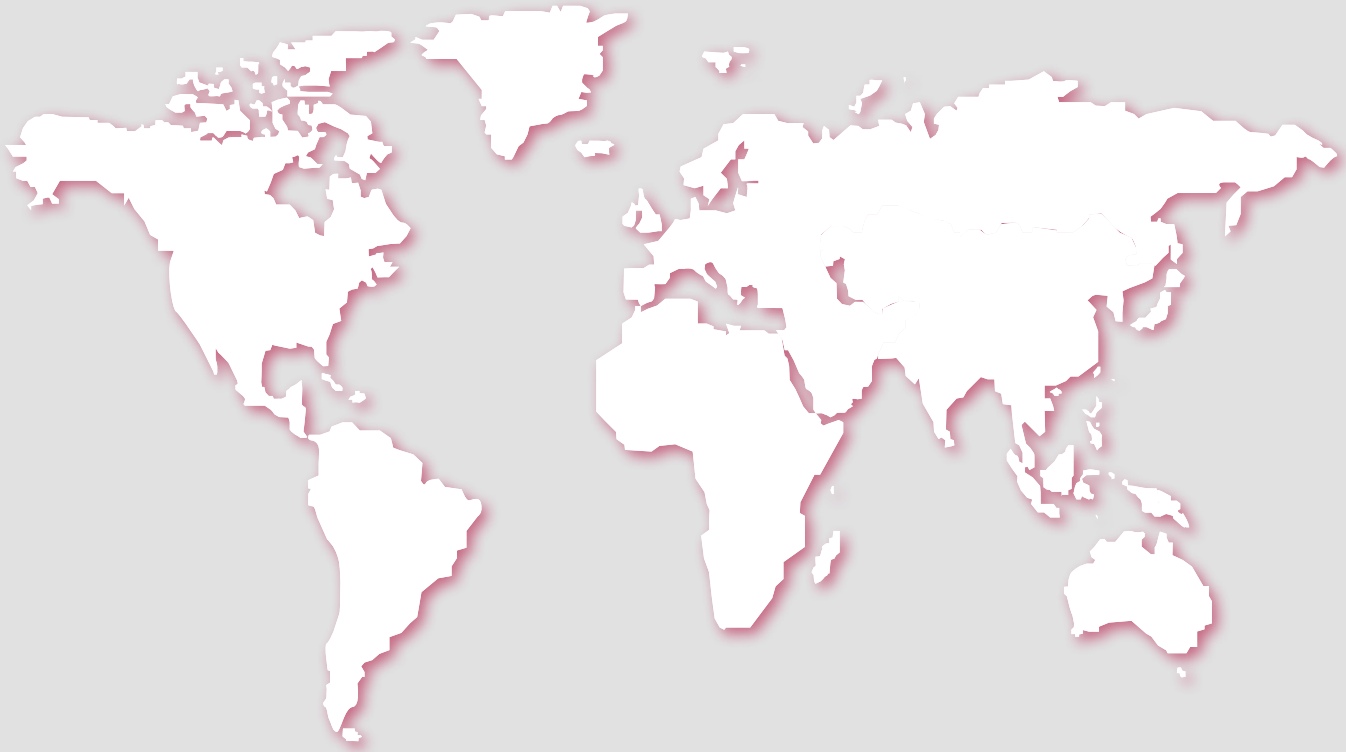
Estimated total Ph+ CML patients worldwide

2014

2015

2025

2040



1. American Cancer Society. Detailed Guide: CML. http://www.cancer.org/acs/groups/cid/documents/webcontent/003112-pdf.pdf. Accessed May 2014. 2. Experts in Chronic Myeloid Leukemia. Blood. 2013;121:4439-4442 3. Hughes T, et al. Blood. 2010;116:3758-3765 4. Data on file. Novartis Pharma AG. Basel, Switzerland.




CML is slightly more prevalent in men. The reasons for this are unknown.[1]

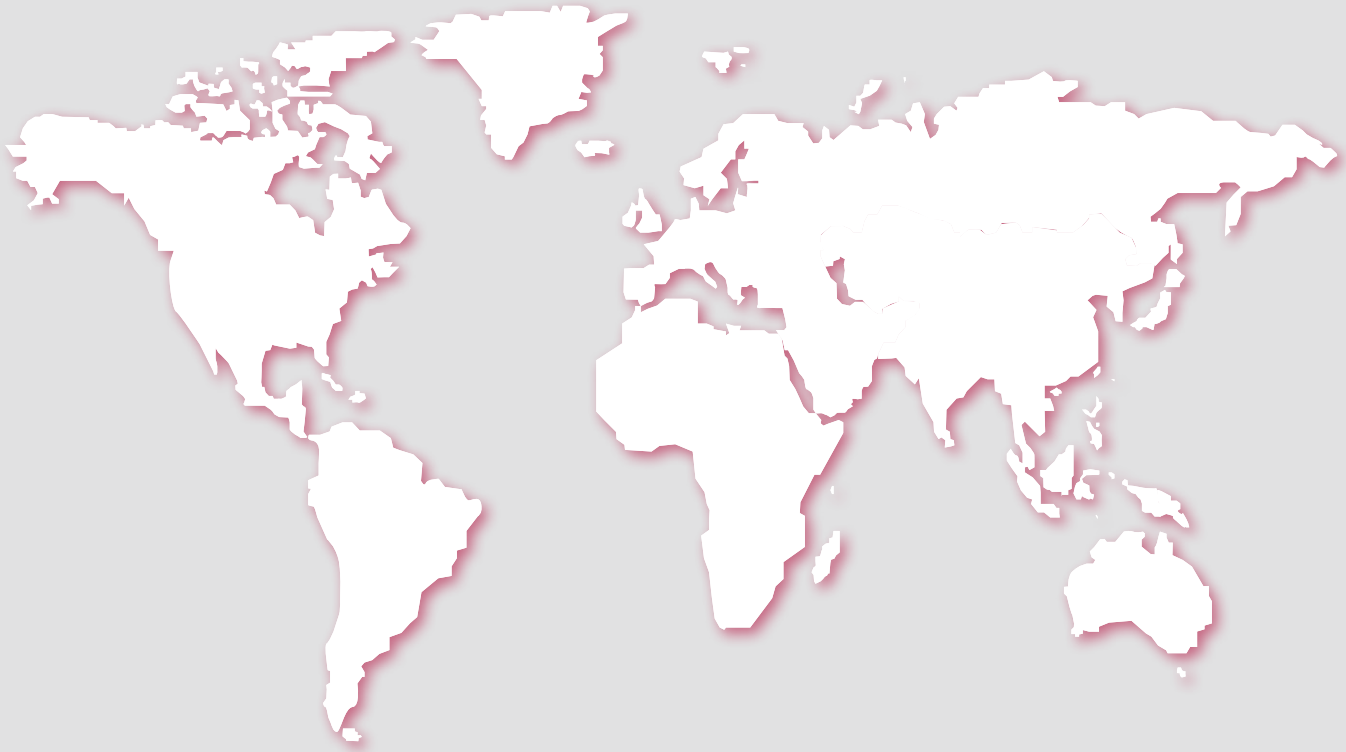
Estimated total Ph+ CML patients worldwide

2014

2015

2025

2040



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CML is rarely seen in children. In fact, over half of CML cases are diagnosed in people 65 and older, with the average age of diagnosis being around 64.[1]

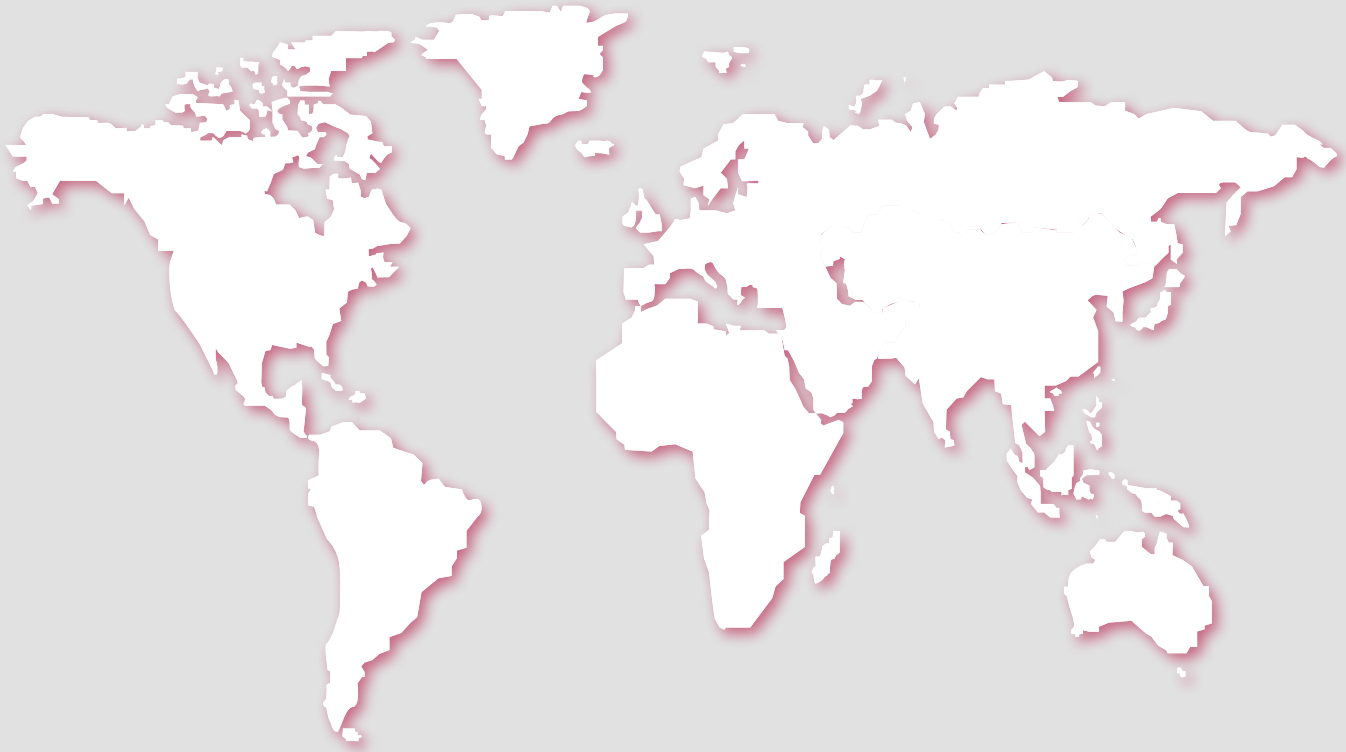
Estimated total Ph+ CML patients worldwide

2014

2015

2025

2040



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CML accounts for about 15% of all adult cases of leukemia.[1]

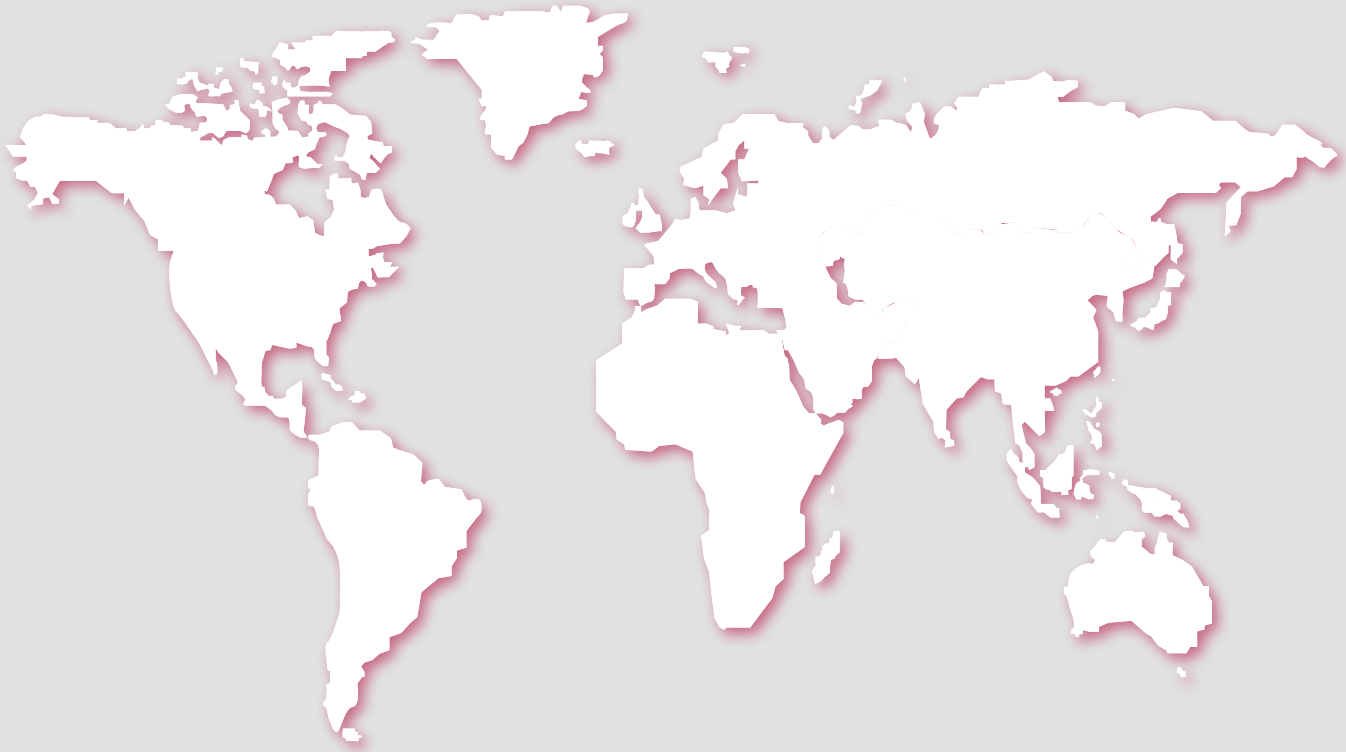
Estimated total Ph+ CML patients worldwide

2014

2015

2025

2040



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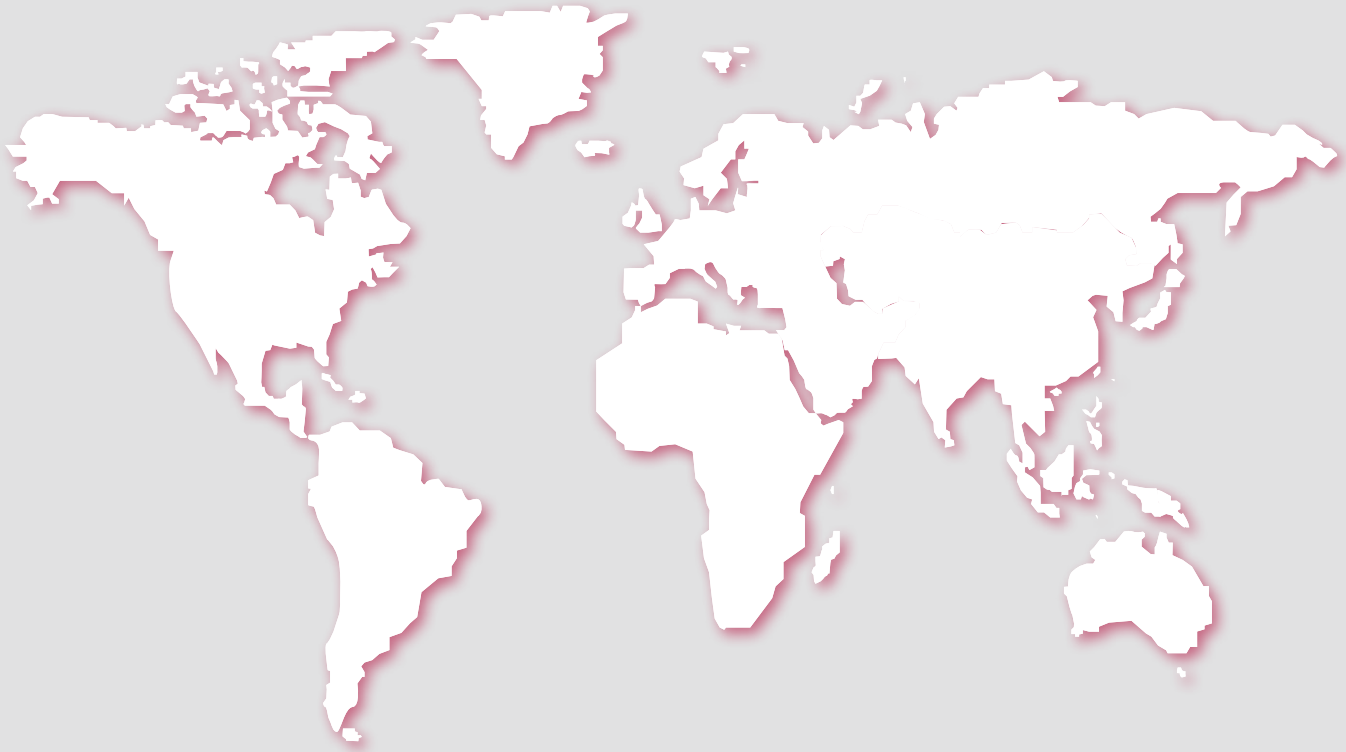
Worldwide, 1.2 to 1.5 million people are currently living with CML,[2] and due to advances in treatment the number of people living with CML is growing.[3,4]

Estimated total Ph+ CML patients worldwide

- **2014**
- **2015**
- **2025**
- **2040**



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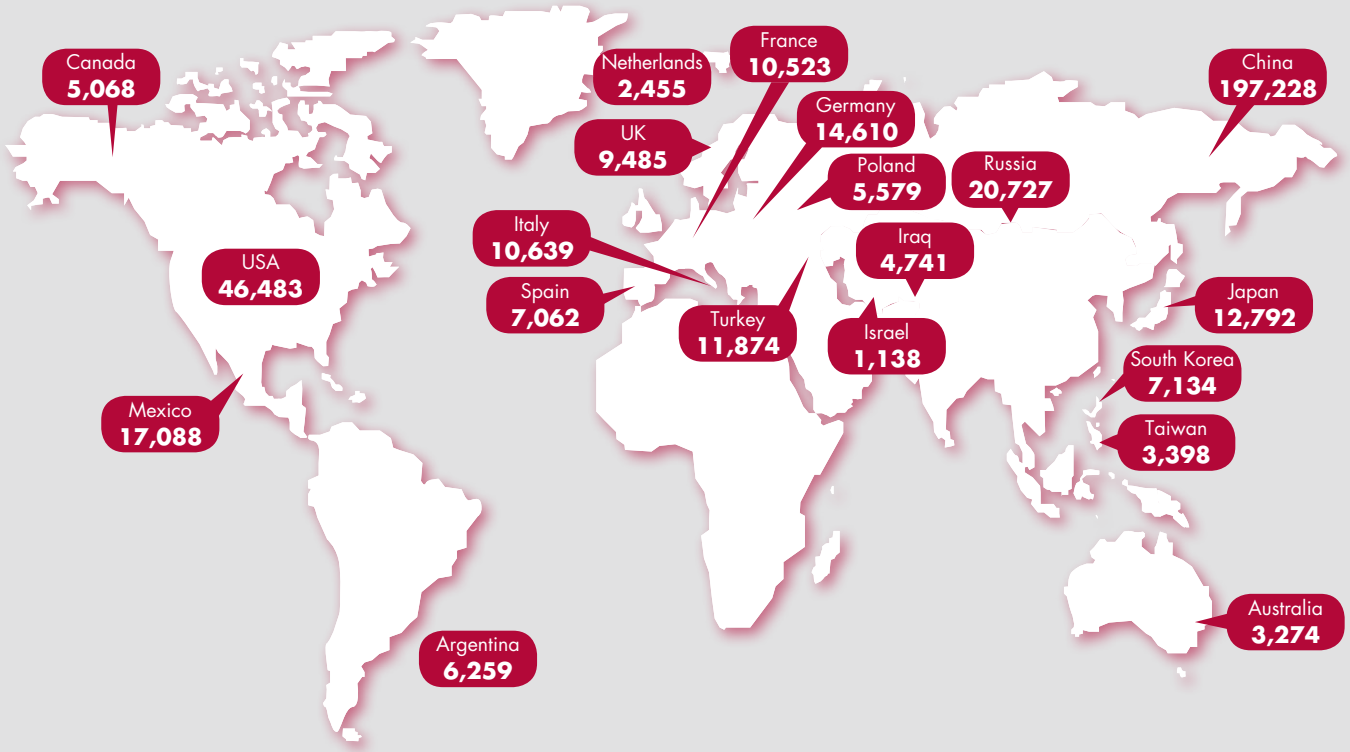
Estimated total Ph+ CML patients worldwide

2014 - **914,420**

2015

2025

2040



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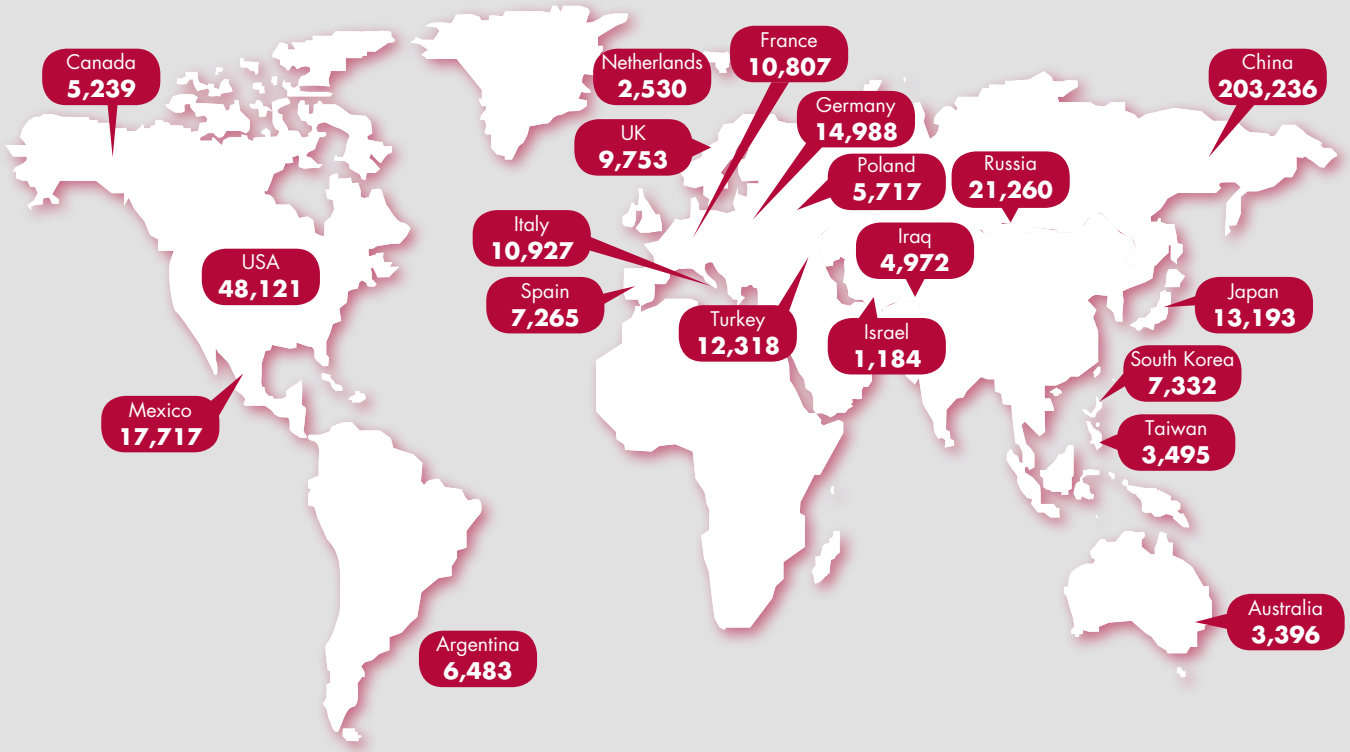
Estimated total Ph+ CML patients worldwide

- 2014
- **2015** - 946,638
- 2025
- 2040



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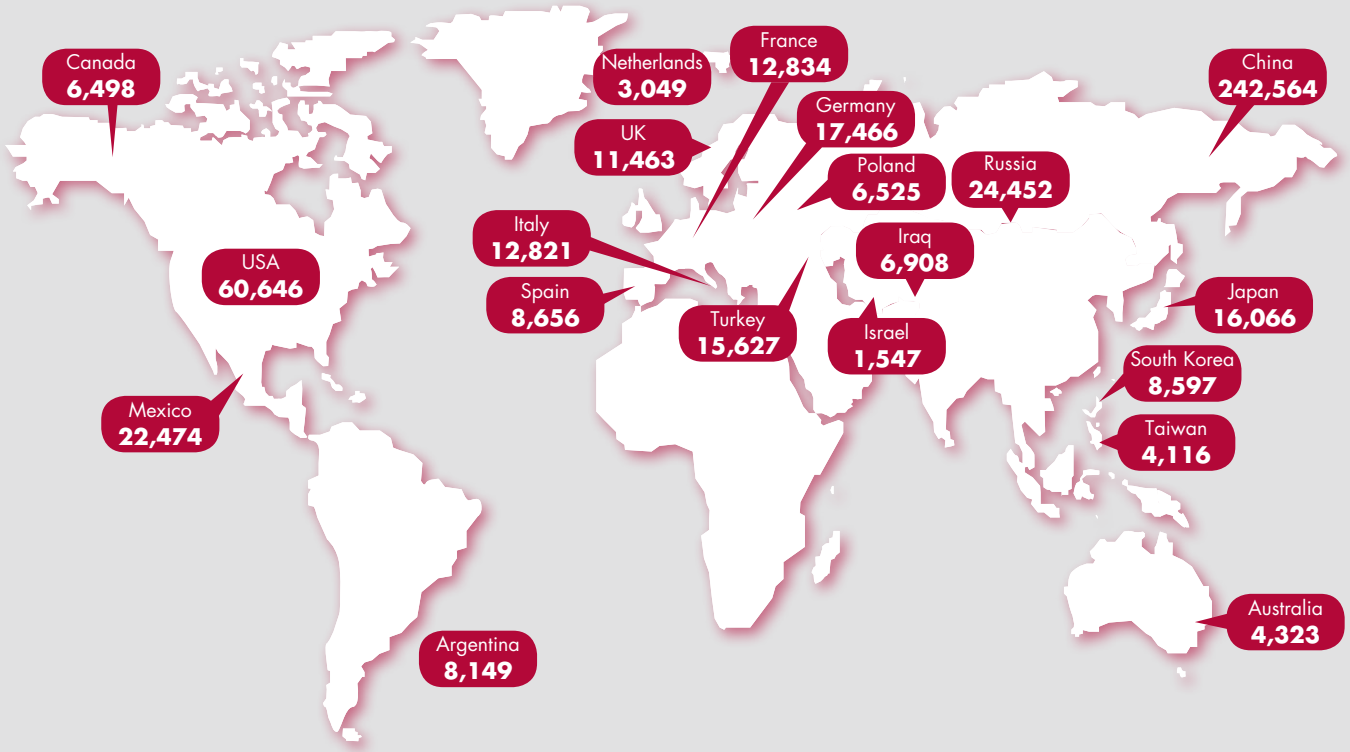
Estimated total Ph+ CML patients worldwide

2014

2015

2025 - 1,185,171

2040



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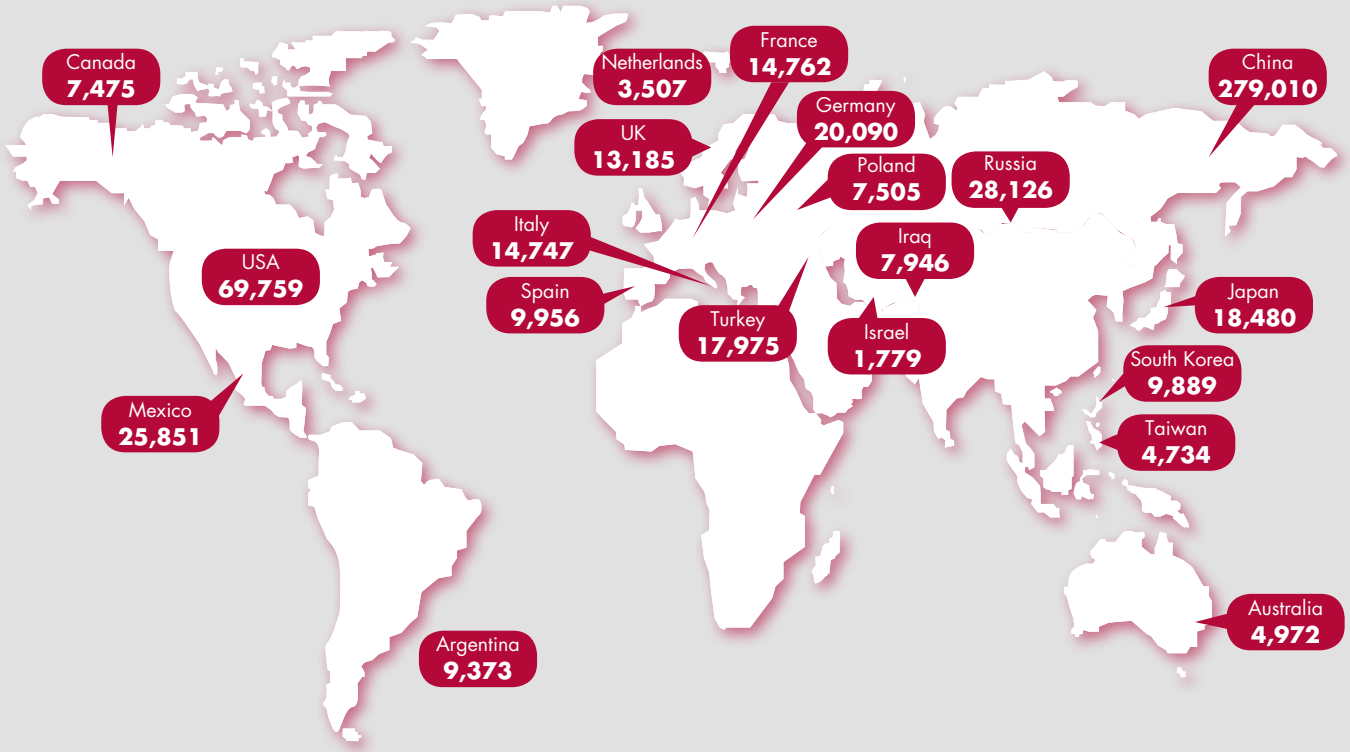
Estimated total Ph+ CML patients worldwide

2014

2015

2025

2040 - 1,363,249



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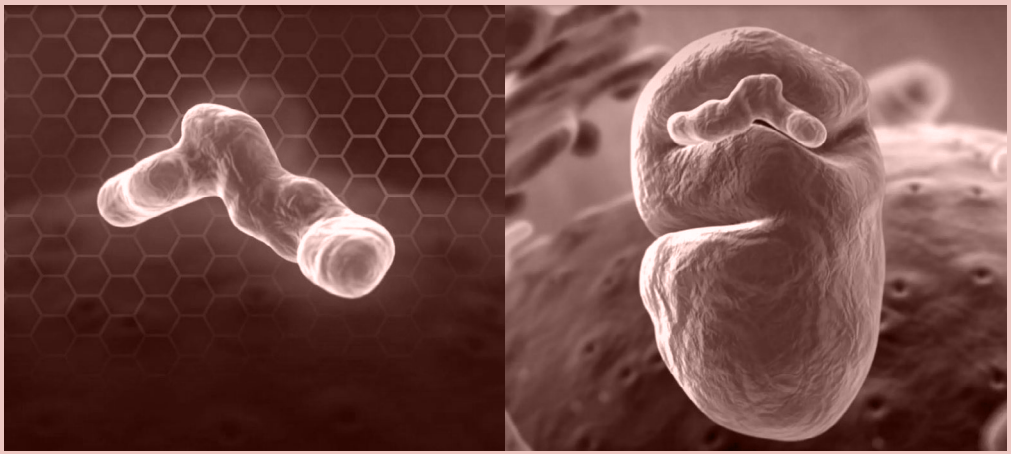

Targeted Ph+ CML therapies have been developed to slow the reproduction of leukemia cells.[1] These therapies work to reduce the levels of cancer-causing proteins and Ph+ CML cells.[1] Some patients who respond exceptionally well to treatment may achieve a level of leukemic cells that is nearly undetectable.

The introduction of BCR-ABL tyrosine kinase inhibitor (TKI) therapy more than a decade ago helped transform Ph+ CML from a life-threatening disease to, in most cases, a chronic condition when managed with appropriate treatments.[2] TKI therapy results in significant BCR-ABL reductions for the majority of patients.[1]

The goal of Ph+ CML treatment is clear: fewer leukemia cells in the body as early on in treatment as possible.[1]

There are several different tests patients will need to have throughout their Ph+ CML treatment journey, and they are taken at different times.[1] Testing occurs most frequently in the first year of treatment, and becomes less frequent – though still important – thereafter.[1]

1. The NCCN Chronic Myelogenous Leukemia Clinical Practice Guidelines in Oncology (Version 3.2014). © 2014 National Comprehensive Cancer Network, Inc. http://www.nccn.org. Accessed May 2014. 2. Rea D, et al. Curr Hematol Malig Rep. 2012;7:103-8. 3. Baccarani M, et al. Blood. 2013;122(6):872-884. 4. Radich J. Blood. 2009;114:3376-3381.

For Ph+ CML there are widely recognized levels of response to treatment: hematologic, cytogenetic and molecular.[1] Routine monitoring of the level of leukemic cells in the body (at a frequency of every three months) is a critical component of Ph+ CML management, using some or all of the laboratory tests available.[1]

Complete Blood Count (CBC):

Cytogenetic Test:

Standardized PCR Test:

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Complete Blood Count (CBC):

A simple blood test that counts the number of white blood cells, red blood cells, and platelets,[1] and should be conducted every 15 days until complete hematologic response (CHR) is achieved, then every 3 months.[3]

Cytogenetic Test:

Standardized PCR Test:

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Complete Blood Count (CBC):

Cytogenetic Test:

A test of the blood or bone marrow that reveals the organization of chromosomes. This helps assist doctors to identify the Philadelphia chromosome,[1] and should be conducted at 3 months, 6 months and 12 months until complete cytogenetic response (CCyR) is achieved, then every 12 months.[3]

Standardized PCR Test:

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Complete Blood Count (CBC):

Cytogenetic Test:

Standardized PCR Test:

A very sensitive test using peripheral blood or bone marrow cells that measures the number of cells that have the BCR-ABL gene (the key cause of Ph+ CML),[1] and should be conducted every 3 months until major molecular response (MMR) is achieved, then every 6 months.[3]

• IS RQ-PCR stands for International Scale Real-time Quantitative Polymerase Chain Reaction. A PCR test measures deep levels of response and is sensitive enough to find one cell with the BCR-ABL gene out of 1,000,000 normal cells.[4]

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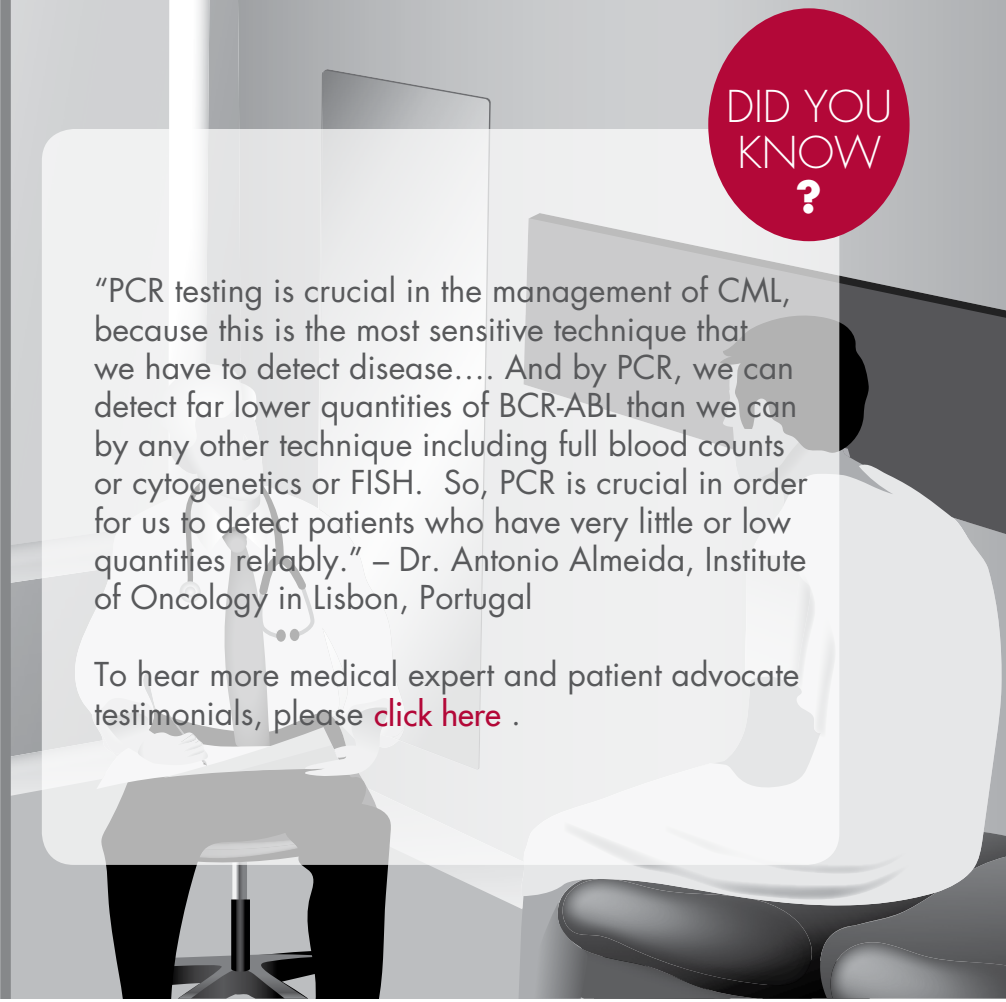

DID YOU KNOW ?

"PCR testing is crucial in the management of CML, because this is the most sensitive technique that we have to detect disease…. And by PCR, we can detect far lower quantities of BCR-ABL than we can by any other technique including full blood counts or cytogenetics or FISH. So, PCR is crucial in order for us to detect patients who have very little or low quantities reliably." – Dr. Antonio Almeida, Institute of Oncology in Lisbon, Portugal

To hear more medical expert and patient advocate testimonials, please click here .

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As Ph+ CML treatment has advanced, so has monitoring of BCR-ABL levels. Greater reductions in BCR-ABL require more precise and sensitive monitoring techniques, and the International Scale Real-time Quantitative Polymerase Chain Reaction, also called IS RQ-PCR, is the most sensitive and accurate test that can be used to identify Ph+ CML in the blood or bone marrow.

More

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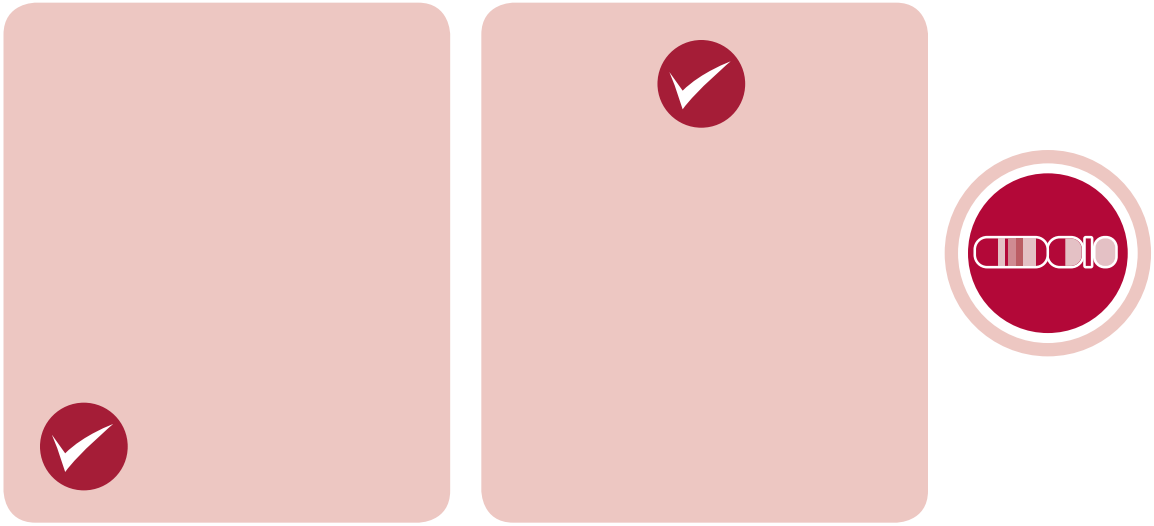

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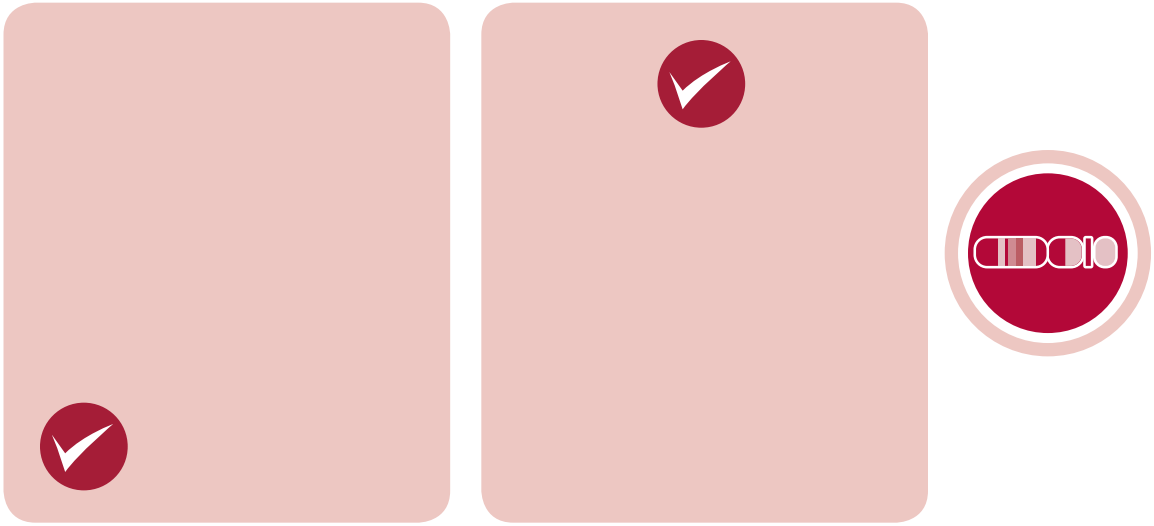
The PCR test is simple, and requires only a blood draw.[1]



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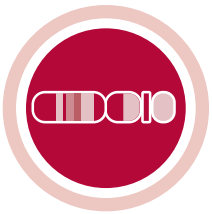
More

+

The fewer leukemia cells a patient has, the deeper the level of response to treatment, and the harder it is to detect the amount of BCR-ABL that remains. PCR is used to monitor the achievement of treatment milestones over time.



1. Radich J. Blood. 2009;114:3376-3381. 2. Akard LP, et al. Clinical Lymphoma, Myeloma & Leukemia. 2011;11(5):385-395. 3. The NCCN Chronic Myelogenous Leukemia Clinical Practice Guidelines in Oncology (Version 3.2014).

As Ph+ CML treatment has advanced, so has monitoring of BCR-ABL levels. Greater reductions in BCR-ABL require more precise and sensitive monitoring techniques, and the International Scale Real-time Quantitative Polymerase Chain Reaction, also called IS RQ-PCR, is the most sensitive and accurate test that can be used to identify Ph+ CML in the blood or bone marrow.

More +

Early and deep response to tyrosine kinase inhibitor therapy, along with routine IS RQ-PCR monitoring, are fundamental to successful management of Ph+ CML.[3]

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Only routine IS RQ-PCR can:[1,2]

- Confirm that deep levels of response, such as MMR and MR4.5, are achieved.
  - MMR (Major molecular response) means that there is 0.1% BCR-ABL detected in the patient's blood, or a 1,000 times fewer BCR-ABL leukemic cells since diagnosis. MMR can also be expressed as molecular response of 3.0. MR4.5 (Molecular response of 4.5) means that the BCR-ABL gene is reduced to 0.0032% from the level at baseline
- Detect early response trends and signs of resistance to CML treatment.
- Provide consistent information about how a patient is responding to treatment, which may drive clinical decisions, such as the need to change therapy.

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DID YOU KNOW ?

Patients with Ph+ CML may always have the leukemic cells in their blood, but the goal of treatment is to prevent reproduction of the leukemic cells and to reduce the overall level of these cells to a level that is very difficult to detect.[1]

The BCR-ABL protein is the sole cause and driver of Ph+ CML in 95-100% of patients.[2] Suppression of BCR-ABL – stopping the overproduction of leukemic white blood cells – is central to achieving undetectable disease.[3] Continuous suppression of BCR-ABL:[4]

- Helps control the disease and achieve deep levels of response (exponentially fewer Bcr-Abl proteins or leukemic cells)
- Reduces the risk of progression to advanced stages of the disease

Meet the milestones that matter

Click body >

Here is a simplified way to understand CML treatment milestones. Think of the dots shown in the body as the amount of leukemic cells in the blood.

At Diagnosis

The level of BCR-ABL in the body is different for every patient at diagnosis, as measured on the International Scale (IS).

1. American Cancer Society. Detailed Guide: CML. http://www.cancer.org/acs/groups/cid/documents/webcontent/003112-pdf.pdf. Accessed May 2014. 2. Moore FR, et al. Methods Mol Biol. 2013;999:1-23. 3. Radich J. Blood. 2009;114:3376-3381. 4. The NCCN Chronic Myelogenous Leukemia Clinical Practice Guidelines in Oncology (Version 3.2014). © 2014 National Comprehensive Cancer Network, Inc. http://www.nccn.org. Accessed May 2014. 5. Baccarani M, et al. Blood. 2013;122(6):872-884. 6. Marin D, et al. J Clin Oncol. 2012;30(3):232-238. 7. Hanfstein B, et al. ASH Annual Meeting 2011. Abstract 783. 8. Hehlmann R, et al. J Clin Oncol. 2014;32(5):415-423. 9. Kantarjian HM, et al. Lancet Oncol. 2011;12(9)841-851. 10. Kantarjian HM, et al. Blood. 2012;119(5):1123-1129.

DID YOU KNOW ?

Physicians use tests to help manage Ph+ CML and decide if any treatment changes are necessary, particularly if a patient is not achieving the recommended milestones according to guidelines (reduction in BCR-ABL within a specific timeframe).[1]

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Early Molecular Response (EMR)

An EMR means BCR-ABL ≤10% when measured on the International Scale (IS). Early molecular response helps to predict future major molecular response and MR4.5 as well as higher progression free and overall survival.[5,6,7]

1. American Cancer Society. Detailed Guide: CML. http://www.cancer.org/acs/groups/cid/documents/webcontent/003112-pdf.pdf. Accessed May 2014. 2. Moore FR, et al. Methods Mol Biol. 2013;999:1-23. 3. Radich J. Blood. 2009;114:3376-3381. 4. The NCCN Chronic Myelogenous Leukemia Clinical Practice Guidelines in Oncology (Version 3.2014). © 2014 National Comprehensive Cancer Network, Inc. http://www.nccn.org. Accessed May 2014. 5. Baccarani M, et al. Blood. 2013;122(6):872-884. 6. Marin D, et al. J Clin Oncol. 2012;30(3):232-238. 7. Hanfstein B, et al. ASH Annual Meeting 2011. Abstract 783. 8. Hehlmann R, et al. J Clin Oncol. 2014;32(5):415-423. 9. Kantarjian HM, et al. Lancet Oncol. 2011;12(9)841-851. 10. Kantarjian HM, et al. Blood. 2012;119(5):1123-1129.

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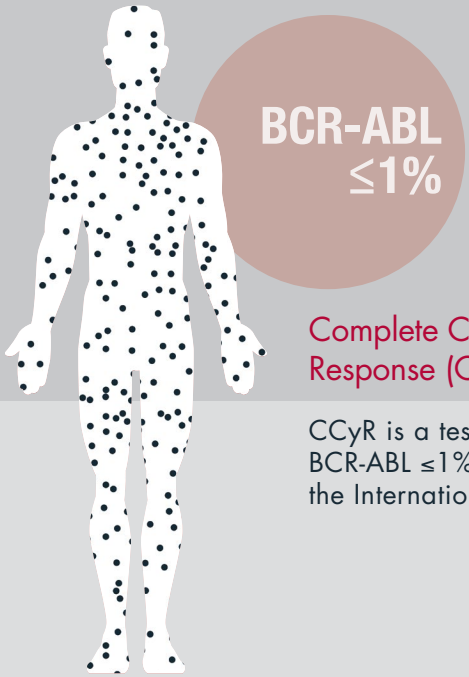

Here is a simplified way to understand CML treatment milestones. Think of the dots shown in the body as the amount of leukemic cells in the blood.

Complete Cytogenetic Response (CCyR)

CCyR is a test result equivalent to BCR-ABL ≤1% when measured on the International Scale (IS).[5]

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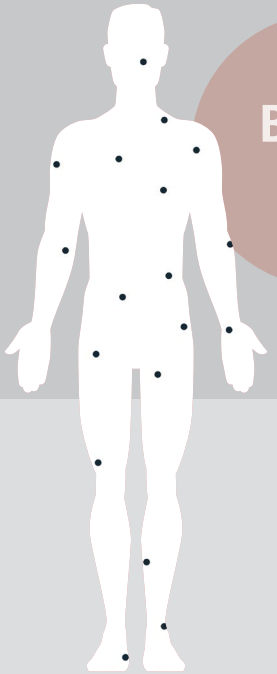

BCR-ABL ≤0.1%

Major Molecular Response (MMR)

An MMR means BCR-ABL ≤0.1% when measured on the International Scale (IS).[5]

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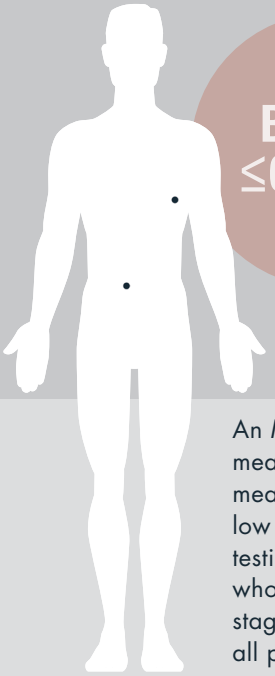


BCR-ABL ≤0.0032%

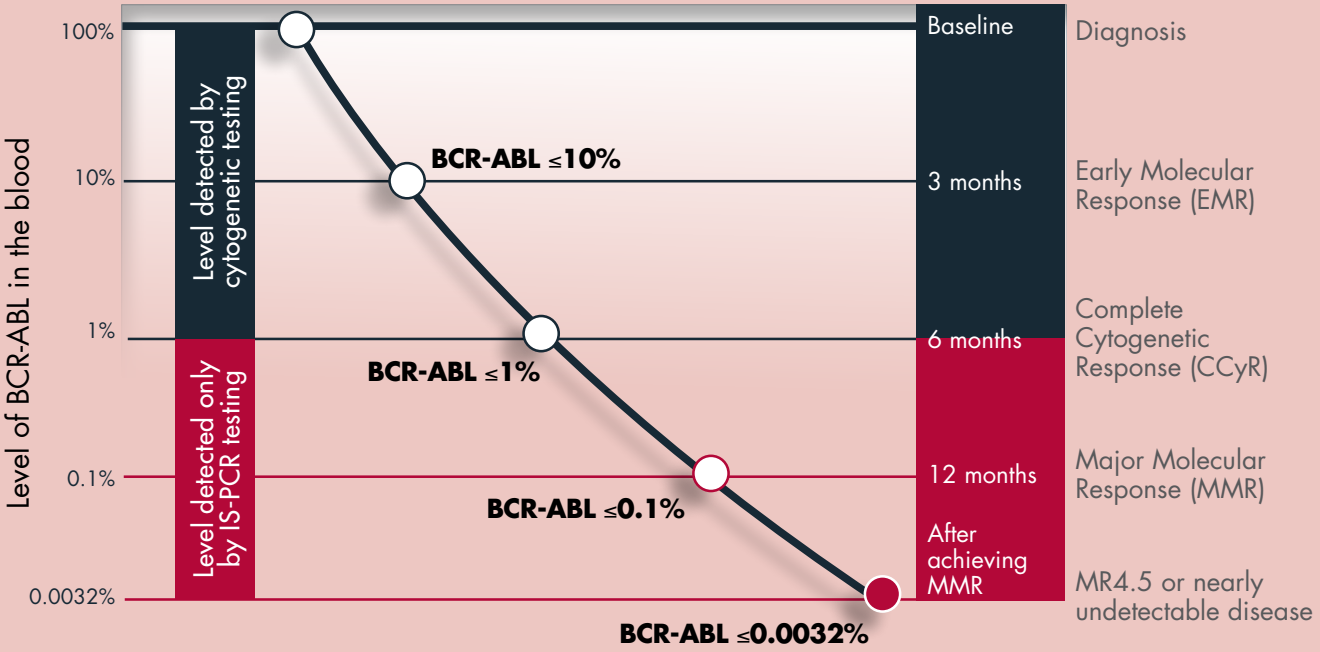
Here is a simplified way to understand CML treatment milestones. Think of the dots shown in the body as the amount of leukemic cells in the blood.

MR4.5: Deeper Response

An MR4.5 means BCR-ABL ≤0.0032% when measured on the International Scale (IS). It also means the amount of leukemic cells is extremely low or nearly undetectable by the most sensitive testing methods currently available. No patients who achieved MR4.5 progressed to advanced stages of the disease in clinical trials, but not all patients may reach this milestone and some patients may reach goals earlier than others.[8,9,10]

1. American Cancer Society. Detailed Guide: CML. http://www.cancer.org/acs/groups/cid/documents/webcontent/003112-pdf.pdf. Accessed May 2014. 2. Moore FR, et al. Methods Mol Biol. 2013;999:1-23. 3. Radich J. Blood. 2009;114:3376-3381. 4. The NCCN Chronic Myelogenous Leukemia Clinical Practice Guidelines in Oncology (Version 3.2014). © 2014 National Comprehensive Cancer Network, Inc. http://www.nccn.org. Accessed May 2014. 5. Baccarani M, et al. Blood. 2013;122(6):872-884. 6. Marin D, et al. J Clin Oncol. 2012;30(3):232-238. 7. Hanfstein B, et al. ASH Annual Meeting 2011. Abstract 783. 8. Hehlmann R, et al. J Clin Oncol. 2014;32(5):415-423. 9. Kantarjian HM, et al. Lancet Oncol. 2011;12(9)841-851. 10. Kantarjian HM, et al. Blood. 2012;119(5):1123-1129.

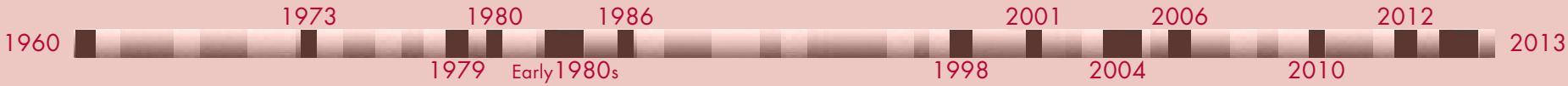
With each treatment milestone, the amount of leukemia in the body is reduced.



What patients can do to help reach their treatment milestones:[1]

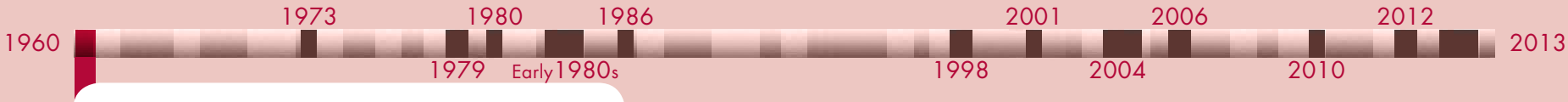
- Take their medicine exactly as prescribed
- Monitor their progress with frequent testing
- Keep appointments with their doctor
- If just starting treatment, discuss the importance of early molecular response with their doctor
- Talk to their doctor if they are experiencing side effects
- Talk to their doctor if they are not reaching their milestones to determine if they may need to switch treatment to get to a deeper level of response

1. American Cancer Society. Detailed Guide: CML. http://www.cancer.org/acs/groups/cid/documents/webcontent/003112-pdf.pdf. Accessed May 2014. 2. Moore FR, et al. Methods Mol Biol. 2013;999:1-23. 3. Radich J. Blood. 2009;114:3376-3381. 4. The NCCN Chronic Myelogenous Leukemia Clinical Practice Guidelines in Oncology (Version 3.2014). © 2014 National Comprehensive Cancer Network, Inc. http://www.nccn.org. Accessed May 2014. 5. Baccarani M, et al. Blood. 2013;122(6):872-884. 6. Marin D, et al. J Clin Oncol. 2012;30(3):232-238. 7. Hanfstein B, et al. ASH Annual Meeting 2011. Abstract 783. 8. Hehlmann R, et al. J Clin Oncol. 2014;32(5):415-423. 9. Kantarjian HM, et al. Lancet Oncol. 2011;12(9)841-851. 10. Kantarjian HM, et al. Blood. 2012;119(5):1123-1129.



CLICK COLUMNS TO REVEAL

1. Jamieson CH. Hematology Am Soc Hematol Educ Program. 2008:436-442. 2. Deininger MWN, et al. Pharmacol Rev. 2003;55(3):401-423. 3. Goldman J. Semin Hematol. 2010;47:302–311. 4. Ben-Neriah Y, et al. Science. 1986;233(4760):212-214. 5. National Cancer Institute website. http://www.cancer.gov/newscenter/newsfromnci/2001/gleevecpressrelease. Accessed May 2014. 6. Drug Information Online. http://www.drugs.com/newdrugs/sprycel-bristol-myers-squibb-chronic-myeloid-leukemia-cml-ph-acute-lymphoblastic-leukemia-ph-all-50.html. Accessed May 2014. 7. Drug Information Online. http://www.drugs.com/newdrugs/novartis-international-ag-ch-fda-approves-tasigna-newly-diagnosed-chronic-myeloid-leukemia-patients-2190.html. Accessed May 2014. 8. Data on file. Novartis Pharma AG. Basel, Switzerland. 9. The NCCN Chronic Myelogenous Leukemia Clinical Practice Guidelines in Oncology (Version 3.2014). © 2014 National Comprehensive Cancer Network, Inc. http://www.nccn.org. Accessed May 2014.



1960 Philadelphia chromosome discovered[1]

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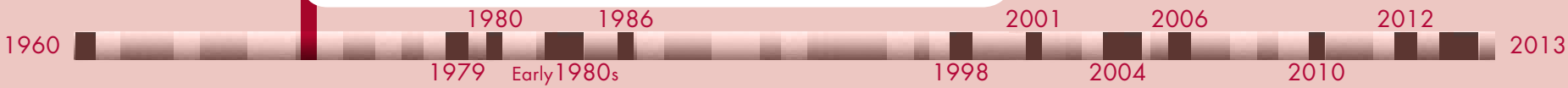
1973 Discovery that the Philadelphia chromosome results from reciprocal translocation of chromosomes 9 and 22, where a portion of the ABL gene from chromosome 9 translocates and fuses with the remaining portion of the BCR gene on chromosome 22[2]



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1979 First successful treatment of CML with bone marrow transplantation[3]

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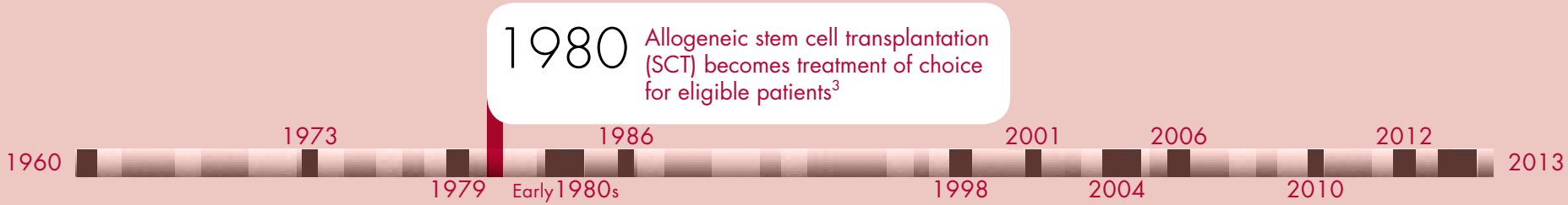

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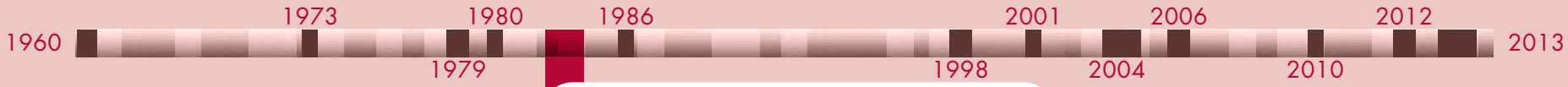

Early
1980s

Interferon introduced for the management of CML in chronic phase and bone marrow transplantation offered[3]

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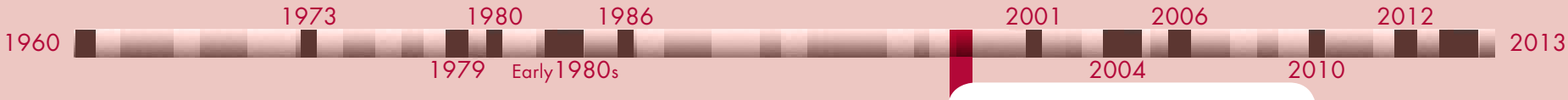
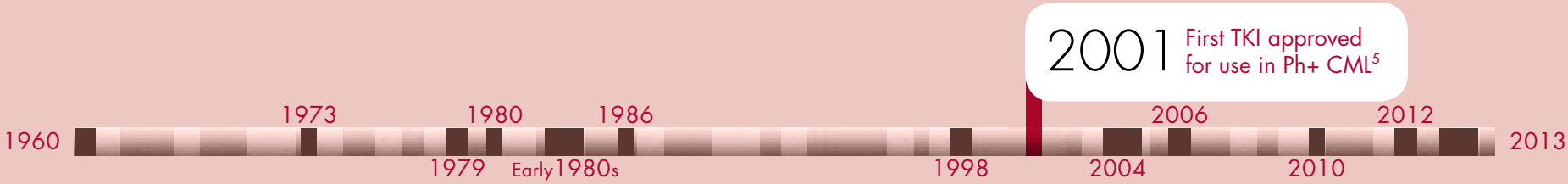


1998 First clinical use of a Bcr-Abl TKI; era of TKIs begins[3]

CLICK COLUMNS TO REVEAL

1. Jamieson CH. Hematology Am Soc Hematol Educ Program. 2008:436-442. 2. Deininger MWN, et al. Pharmacol Rev. 2003;55(3):401-423. 3. Goldman J. Semin Hematol. 2010;47:302–311. 4. Ben-Neriah Y, et al. Science. 1986;233(4760):212-214. 5. National Cancer Institute website. http://www.cancer.gov/newscenter/newsfromnci/2001/gleevecpressrelease. Accessed May 2014. 6. Drug Information Online. http://www.drugs.com/newdrugs/sprycel-bristol-myers-squibb-chronic-myeloid-leukemia-cml-ph-acute-lymphoblastic-leukemia-ph-all-50.html. Accessed May 2014. 7. Drug Information Online. http://www.drugs.com/newdrugs/novartis-international-ag-ch-fda-approves-tasigna-newly-diagnosed-chronic-myeloid-leukemia-patients-2190.html. Accessed May 2014. 8. Data on file. Novartis Pharma AG. Basel, Switzerland. 9. The NCCN Chronic Myelogenous Leukemia Clinical Practice Guidelines in Oncology (Version 3.2014). © 2014 National Comprehensive Cancer Network, Inc. http://www.nccn.org. Accessed May 2014.



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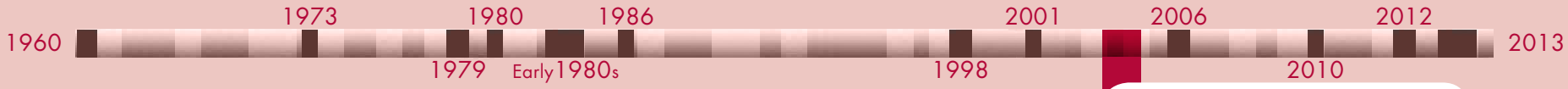
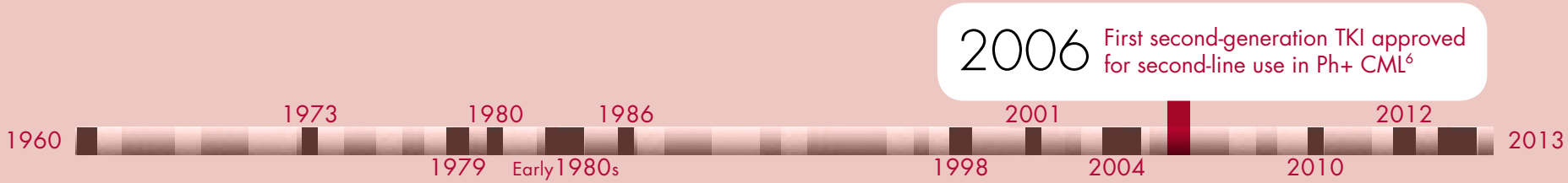
1. Jamieson CH. Hematology Am Soc Hematol Educ Program. 2008:436-442. 2. Deininger MWN, et al. Pharmacol Rev. 2003;55(3):401-423. 3. Goldman J. Semin Hematol. 2010;47:302–311. 4. Ben-Neriah Y, et al. Science. 1986;233(4760):212-214. 5. National Cancer Institute website. http://www.cancer.gov/newscenter/newsfromnci/2001/gleevecpressrelease. Accessed May 2014. 6. Drug Information Online. http://www.drugs.com/newdrugs/sprycel-bristol-myers-squibb-chronic-myeloid-leukemia-cml-ph-acute-lymphoblastic-leukemia-ph-all-50.html. Accessed May 2014. 7. Drug Information Online. http://www.drugs.com/newdrugs/novartis-international-ag-ch-fda-approves-tasigna-newly-diagnosed-chronic-myeloid-leukemia-patients-2190.html. Accessed May 2014. 8. Data on file. Novartis Pharma AG. Basel, Switzerland. 9. The NCCN Chronic Myelogenous Leukemia Clinical Practice Guidelines in Oncology (Version 3.2014). © 2014 National Comprehensive Cancer Network, Inc. http://www.nccn.org. Accessed May 2014.



2004 First clinical use of second-generation TKIs[3]

CLICK COLUMNS TO REVEAL

1. Jamieson CH. Hematology Am Soc Hematol Educ Program. 2008:436-442. 2. Deininger MWN, et al. Pharmacol Rev. 2003;55(3):401-423. 3. Goldman J. Semin Hematol. 2010;47:302–311. 4. Ben-Neriah Y, et al. Science. 1986;233(4760):212-214. 5. National Cancer Institute website. http://www.cancer.gov/newscenter/newsfromnci/2001/gleevecpressrelease. Accessed May 2014. 6. Drug Information Online. http://www.drugs.com/newdrugs/sprycel-bristol-myers-squibb-chronic-myeloid-leukemia-cml-ph-acute-lymphoblastic-leukemia-ph-all-50.html. Accessed May 2014. 7. Drug Information Online. http://www.drugs.com/newdrugs/novartis-international-ag-ch-fda-approves-tasigna-newly-diagnosed-chronic-myeloid-leukemia-patients-2190.html. Accessed May 2014. 8. Data on file. Novartis Pharma AG. Basel, Switzerland. 9. The NCCN Chronic Myelogenous Leukemia Clinical Practice Guidelines in Oncology (Version 3.2014). © 2014 National Comprehensive Cancer Network, Inc. http://www.nccn.org. Accessed May 2014.



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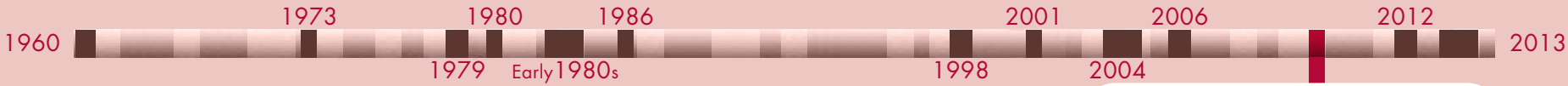
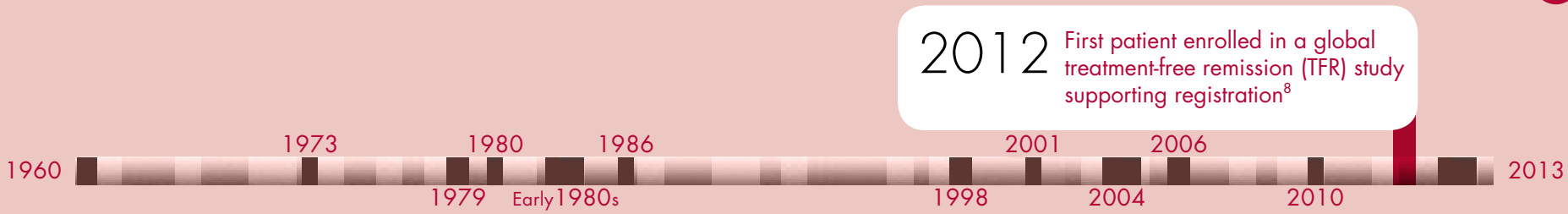
1. Jamieson CH. Hematology Am Soc Hematol Educ Program. 2008:436-442. 2. Deininger MWN, et al. Pharmacol Rev. 2003;55(3):401-423. 3. Goldman J. Semin Hematol. 2010;47:302–311. 4. Ben-Neriah Y, et al. Science. 1986;233(4760):212-214. 5. National Cancer Institute website. http://www.cancer.gov/newscenter/newsfromnci/2001/gleevecpressrelease. Accessed May 2014. 6. Drug Information Online. http://www.drugs.com/newdrugs/sprycel-bristol-myers-squibb-chronic-myeloid-leukemia-cml-ph-acute-lymphoblastic-leukemia-ph-all-50.html. Accessed May 2014. 7. Drug Information Online. http://www.drugs.com/newdrugs/novartis-international-ag-ch-fda-approves-tasigna-newly-diagnosed-chronic-myeloid-leukemia-patients-2190.html. Accessed May 2014. 8. Data on file. Novartis Pharma AG. Basel, Switzerland. 9. The NCCN Chronic Myelogenous Leukemia Clinical Practice Guidelines in Oncology (Version 3.2014). © 2014 National Comprehensive Cancer Network, Inc. http://www.nccn.org. Accessed May 2014.



2010 Second-generation TKI approved for first-line use in Ph+ CML[7]

CLICK COLUMNS TO REVEAL

1. Jamieson CH. Hematology Am Soc Hematol Educ Program. 2008:436-442. 2. Deininger MWN, et al. Pharmacol Rev. 2003;55(3):401-423. 3. Goldman J. Semin Hematol. 2010;47:302–311. 4. Ben-Neriah Y, et al. Science. 1986;233(4760):212-214. 5. National Cancer Institute website. http://www.cancer.gov/newscenter/newsfromnci/2001/gleevecpressrelease. Accessed May 2014. 6. Drug Information Online. http://www.drugs.com/newdrugs/sprycel-bristol-myers-squibb-chronic-myeloid-leukemia-cml-ph-acute-lymphoblastic-leukemia-ph-all-50.html. Accessed May 2014. 7. Drug Information Online. http://www.drugs.com/newdrugs/novartis-international-ag-ch-fda-approves-tasigna-newly-diagnosed-chronic-myeloid-leukemia-patients-2190.html. Accessed May 2014. 8. Data on file. Novartis Pharma AG. Basel, Switzerland. 9. The NCCN Chronic Myelogenous Leukemia Clinical Practice Guidelines in Oncology (Version 3.2014). © 2014 National Comprehensive Cancer Network, Inc. http://www.nccn.org. Accessed May 2014.



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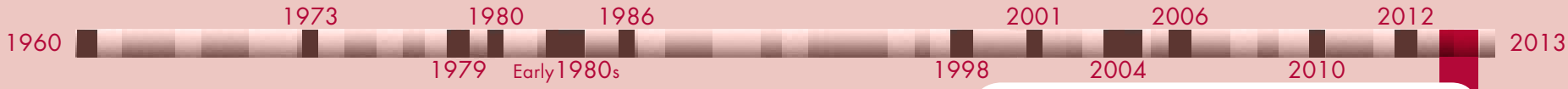

2013 First patient enrolled in a global, registration TFR study[8]

CLICK COLUMNS TO REVEAL

1. Jamieson CH. Hematology Am Soc Hematol Educ Program. 2008:436-442. 2. Deininger MWN, et al. Pharmacol Rev. 2003;55(3):401-423. 3. Goldman J. Semin Hematol. 2010;47:302–311. 4. Ben-Neriah Y, et al. Science. 1986;233(4760):212-214. 5. National Cancer Institute website. http://www.cancer.gov/newscenter/newsfromnci/2001/gleevecpressrelease. Accessed May 2014. 6. Drug Information Online. http://www.drugs.com/newdrugs/sprycel-bristol-myers-squibb-chronic-myeloid-leukemia-cml-ph-acute-lymphoblastic-leukemia-ph-all-50.html. Accessed May 2014. 7. Drug Information Online. http://www.drugs.com/newdrugs/novartis-international-ag-ch-fda-approves-tasigna-newly-diagnosed-chronic-myeloid-leukemia-patients-2190.html. Accessed May 2014. 8. Data on file. Novartis Pharma AG. Basel, Switzerland. 9. The NCCN Chronic Myelogenous Leukemia Clinical Practice Guidelines in Oncology (Version 3.2014). © 2014 National Comprehensive Cancer Network, Inc. http://www.nccn.org. Accessed May 2014.

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The medical community is now conducting several ongoing clinical trials to explore the feasibility of permanent TKI cessation in patients with sustained deep molecular response; some ongoing clinical trials are also exploring the feasibility of cessation in patients with major molecular response.

1. Jamieson CH. Hematology Am Soc Hematol Educ Program. 2008:436-442. 2. Deininger MWN, et al. Pharmacol Rev. 2003;55(3):401-423. 3. Goldman J. Semin Hematol. 2010;47:302–311. 4. Ben-Neriah Y, et al. Science. 1986;233(4760):212-214. 5. National Cancer Institute website. http://www.cancer.gov/newscenter/newsfromnci/2001/gleevecpressrelease. Accessed May 2014. 6. Drug Information Online. http://www.drugs.com/newdrugs/sprycel-bristol-myers-squibb-chronic-myeloid-leukemia-cml-ph-acute-lymphoblastic-leukemia-ph-all-50.html. Accessed May 2014. 7. Drug Information Online. http://www.drugs.com/newdrugs/novartis-international-ag-ch-fda-approves-tasigna-newly-diagnosed-chronic-myeloid-leukemia-patients-2190.html. Accessed May 2014. 8. Data on file. Novartis Pharma AG. Basel, Switzerland. 9. The NCCN Chronic Myelogenous Leukemia Clinical Practice Guidelines in Oncology (Version 3.2014). © 2014 National Comprehensive Cancer Network, Inc. http://www.nccn.org. Accessed May 2014.

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Following the science, Novartis Oncology is looking to redefine what is possible in the treatment of Ph+ CML through a clinical trials program to evaluate the potential for patients with Ph+ CML to live without drug therapy—a concept called treatment-free remission (TFR). The global clinical trials program is part of a Novartis vision called The Path to Cure in CML.

1. Jamieson CH. Hematology Am Soc Hematol Educ Program. 2008:436-442. 2. Deininger MWN, et al. Pharmacol Rev. 2003;55(3):401-423. 3. Goldman J. Semin Hematol. 2010;47:302–311. 4. Ben-Neriah Y, et al. Science. 1986;233(4760):212-214. 5. National Cancer Institute website. http://www.cancer.gov/newscenter/newsfromnci/2001/gleevecpressrelease. Accessed May 2014. 6. Drug Information Online. http://www.drugs.com/newdrugs/sprycel-bristol-myers-squibb-chronic-myeloid-leukemia-cml-ph-acute-lymphoblastic-leukemia-ph-all-50.html. Accessed May 2014. 7. Drug Information Online. http://www.drugs.com/newdrugs/novartis-international-ag-ch-fda-approves-tasigna-newly-diagnosed-chronic-myeloid-leukemia-patients-2190.html. Accessed May 2014. 8. Data on file. Novartis Pharma AG. Basel, Switzerland. 9. The NCCN Chronic Myelogenous Leukemia Clinical Practice Guidelines in Oncology (Version 3.2014). © 2014 National Comprehensive Cancer Network, Inc. http://www.nccn.org. Accessed May 2014.

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The long-term goal of the Novartis Oncology Path to Cure program is to achieve a cure for Ph+ CML for as many patients as possible.

1. Jamieson CH. Hematology Am Soc Hematol Educ Program. 2008:436-442. 2. Deininger MWN, et al. Pharmacol Rev. 2003;55(3):401-423. 3. Goldman J. Semin Hematol. 2010;47:302–311. 4. Ben-Neriah Y, et al. Science. 1986;233(4760):212-214. 5. National Cancer Institute website. http://www.cancer.gov/newscenter/newsfromnci/2001/gleevecpressrelease. Accessed May 2014. 6. Drug Information Online. http://www.drugs.com/newdrugs/sprycel-bristol-myers-squibb-chronic-myeloid-leukemia-cml-ph-acute-lymphoblastic-leukemia-ph-all-50.html. Accessed May 2014. 7. Drug Information Online. http://www.drugs.com/newdrugs/novartis-international-ag-ch-fda-approves-tasigna-newly-diagnosed-chronic-myeloid-leukemia-patients-2190.html. Accessed May 2014. 8. Data on file. Novartis Pharma AG. Basel, Switzerland. 9. The NCCN Chronic Myelogenous Leukemia Clinical Practice Guidelines in Oncology (Version 3.2014). © 2014 National Comprehensive Cancer Network, Inc. http://www.nccn.org. Accessed May 2014.

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Current clinical trial evidence points to sustained deep molecular response as an important milestone to achieve before treatment can be stopped in select patients. Stopping treatment in CML is not a current clinical recommendation and should only be explored in the context of a well-controlled clinical trial.[8]

Once treatment is stopped, accurate and precise assessment of residual molecular disease using the Real Time Quantitative PCR standardized to the International Scale, also called IS RQ-PCR, is essential to identify if a patient's level of disease remains in deep molecular response or if the reintroduction of treatment is needed.

1. Jamieson CH. Hematology Am Soc Hematol Educ Program. 2008:436-442. 2. Deininger MWN, et al. Pharmacol Rev. 2003;55(3):401-423. 3. Goldman J. Semin Hematol. 2010;47:302–311. 4. Ben-Neriah Y, et al. Science. 1986;233(4760):212-214. 5. National Cancer Institute website. http://www.cancer.gov/newscenter/newsfromnci/2001/gleevecpressrelease. Accessed May 2014. 6. Drug Information Online. http://www.drugs.com/newdrugs/sprycel-bristol-myers-squibb-chronic-myeloid-leukemia-cml-ph-acute-lymphoblastic-leukemia-ph-all-50.html. Accessed May 2014. 7. Drug Information Online. http://www.drugs.com/newdrugs/novartis-international-ag-ch-fda-approves-tasigna-newly-diagnosed-chronic-myeloid-leukemia-patients-2190.html. Accessed May 2014. 8. Data on file. Novartis Pharma AG. Basel, Switzerland. 9. The NCCN Chronic Myelogenous Leukemia Clinical Practice Guidelines in Oncology (Version 3.2014). © 2014 National Comprehensive Cancer Network, Inc. http://www.nccn.org. Accessed May 2014.

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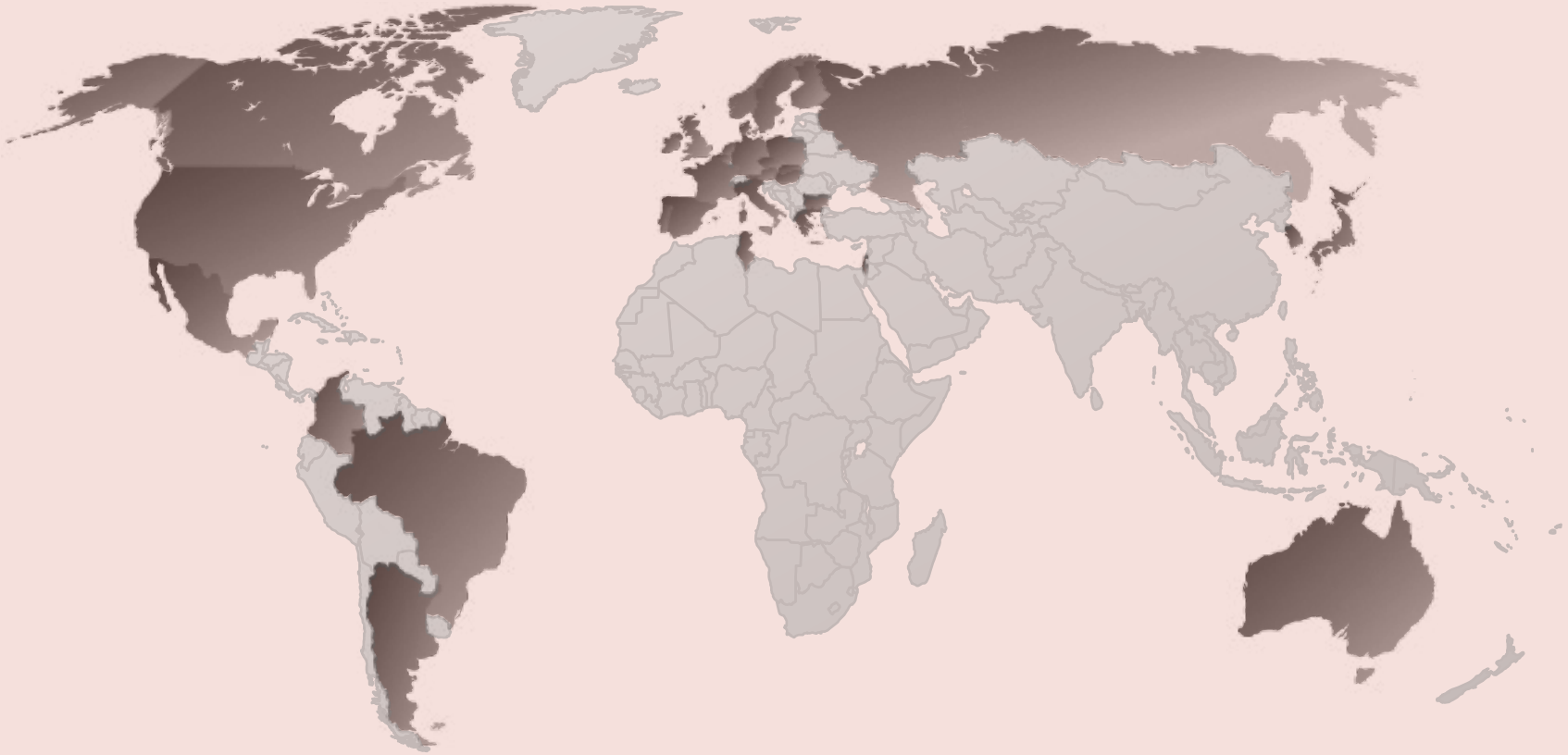
The Novartis Oncology CML clinical trials program includes four treatment-free remission studies that are now underway and actively enrolling Ph+ CML patients in more than 150 study centers across 33 countries. In total, it is planned that more than 1,000 patients will be enrolled in these studies.

1. Jamieson CH. Hematology Am Soc Hematol Educ Program. 2008:436-442. 2. Deininger MWN, et al. Pharmacol Rev. 2003;55(3):401-423. 3. Goldman J. Semin Hematol. 2010;47:302–311. 4. Ben-Neriah Y, et al. Science. 1986;233(4760):212-214. 5. National Cancer Institute website. http://www.cancer.gov/newscenter/newsfromnci/2001/gleevecpressrelease. Accessed May 2014. 6. Drug Information Online. http://www.drugs.com/newdrugs/sprycel-bristol-myers-squibb-chronic-myeloid-leukemia-cml-ph-acute-lymphoblastic-leukemia-ph-all-50.html. Accessed May 2014. 7. Drug Information Online. http://www.drugs.com/newdrugs/novartis-international-ag-ch-fda-approves-tasigna-newly-diagnosed-chronic-myeloid-leukemia-patients-2190.html. Accessed May 2014. 8. Data on file. Novartis Pharma AG. Basel, Switzerland. 9. The NCCN Chronic Myelogenous Leukemia Clinical Practice Guidelines in Oncology (Version 3.2014). © 2014 National Comprehensive Cancer Network, Inc. http://www.nccn.org. Accessed May 2014.

Countries Participating in Treatment-Free Remission Trials



Participating in ≥1 ENEST TFR study

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BCR-ABL: An abnormal gene that is formed when 2 specific chromosomes combine. This gene helps produce a protein called Bcr-Abl, which causes Ph+ CML.[1]

Cytogenetic response: A response to CML treatment in which a blood test or PCR test measures the number of BCR-ABL cells in the blood and bone marrow. This can be in the form of a complete, partial or major cytogenetic response. According to treatment guidelines, a complete cytogenetic response (CCyR or <1% BCR-ABL) ideally occurs within 6 months after starting therapy.[2]

Early molecular response: A response to CML treatment which means BCR-ABL ≤10% at 3 months. Early molecular response is thought to predict future major molecular response and MR4.5 as well as higher progression free and overall survival.[3,4,5]

IS RQ-PCR (International Scale Real-time Quantitative Polymerase Chain Reaction): The most accurate and sensitive method to measure the amount of leukemic cells in the body. Using peripheral blood or bone marrow cells, a PCR test can find a single cell with BCR-ABL gene in 1,000,000 normal cells. Routine PCR testing is important in the management of Ph+ CML, as it helps track progress and is an indication of how a patient is responding to treatment. The international scale is used to standardize and validate a patient's test results.[6]

Molecular response: A deep response to CML treatment in which a PCR test measures the number of BCR-ABL cells in the blood and bone marrow. This can be in the form of a complete or major molecular response. A major molecular response (MMR) ideally occurs within 12 months of starting therapy and is associated with increased probability of progression-free survival and overall survival.[2]

MR4.5: Molecular response of 4.5, also stated as 4.5 log reduction, in which the BCR-ABL gene is reduced to 0.0032%. Clinical evidence to date has shown that after reaching and sustaining this milestone, no patient has had their CML progress to advanced stages of the disease regardless of TKI treatment prescribed, however not all patients may reach this milestone.[7,8,9]

Philadelphia (Ph) chromosome: An abnormal chromosome that is responsible for the uncontrolled production of white blood cells (myeloid cells) that are present in CML.[1]

Treatment-free remission (TFR): A goal in the treatment of Ph+ CML where patients who have achieved and maintained MR4.5 stop drug therapy and are able to maintain an undetectable level of disease. Stopping treatment in CML is not a current clinical recommendation and should only be attempted in the context of a well-conducted clinical study.[3]

Ph+ CML treatment milestones: Responses that indicate how well a patient is responding to treatment. With each subsequent milestone reached on treatment, there are significantly fewer leukemic cells.[2]

Tyrosine kinase inhibitor (TKI): A molecule, delivered in the form of a drug therapy, that targets and blocks the ability of the abnormal BCR-ABL gene to send signals that drive production of the leukemic blood cells. TKIs have become the standard of treatment for CML.[1]

NOVARTIS

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November 2016

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1. American Cancer Society. Detailed Guide: CML. http://www.cancer.org/acs/groups/cid/documents/webcontent/003112-pdf.pdf. 2. The NCCN Chronic Myelogenous Leukemia Clinical Practice Guidelines in Oncology (Version 3.2014). © 2014 National Comprehensive Cancer Network, Inc. http://www.nccn.org. Accessed May 2014. 3. Hehlmann R, et al. ASCO Annual Meeting 2012. Abstract 6510. 4. Baccarani M, et al. Blood. 2013;122(6):872-884. 5. Marin D, et al. J Clin Oncol. 2012;30(3):232-238. 6. Hanfstein B, et al. ASH Annual Meeting 2011. Abstract 783. 7. Radich J. Blood. 2009;114:3376-3381. 8. Hehlmann R, et al. J Clin Oncol. 2014;32(5):415-423. 9. Kantarjian HM, et al. Lancet Oncol. 2011;12(9)841-851. 10. Kantarjian HM, et al. Blood. 2012;119(5):1123-1129.