# What is leukaemia?

## A Guide for Nurses

Leukaemia Care
Nurse Academy

# Introduction

**The 'leukaemias' are a heterogeneous group of neoplastic disorders of blood cells.**

Simplistically, they can be defined as acute or chronic, depending on the degree of haematopoietic cell differentiation on presentation and the speed with which the disease progresses, and myeloid or lymphoid, depending on the cell lineage. This chapter focuses on four main types of leukaemia:

- acute myeloid leukaemia (AML)
- acute lymphoblastic leukaemia (ALL)
- chronic myeloid leukaemia (CML)
- chronic lymphocytic leukaemia (CLL).

If you would like any information on the sources used for this booklet, please email **communications@leukaemiacare.org.uk** for a list of references.

# In this booklet

# About Leukaemia Care

**Leukaemia Care is a national charity dedicated to ensuring that people affected by blood cancer have access to the right information, advice and support.**

## Our services

### Helpline

Our helpline is available 9:00am – 5:00pm Monday - Friday and 7:00pm – 10:00pm on Thursdays and Fridays. If you need someone to talk to, call **08088 010 444**.

Alternatively, you can send a message via WhatsApp on **07500068065** on weekdays 9:00am – 5:00pm.

### Nurse service

We have two trained nurses on hand to answer your questions and offer advice and support, whether it be through emailing **nurse@leukaemiacare.org.uk** or over the phone on **08088 010 444**.

### Patient Information Booklets

We have a number of patient information booklets like this available to anyone who has been affected by a blood cancer. A full list of titles – both disease specific and general information titles – can be found on our website at **www.leukaemiacare.org.uk/support-and-information/help-and-resources/information-booklets/**

### Support Groups

Our nationwide support groups are a chance to meet and talk to other people who are going through a similar experience. For more information about a support group local to your area, go to **www.leukaemiacare.org.uk/support-and-information/support-for-you/find-a-support-group/**

### Buddy Support

We offer one-to-one phone support with volunteers who have had blood cancer themselves or been affected by it in some

way. You can speak to someone who knows what you are going through. For more information on how to get a buddy call **08088 010 444** or email **support@leukaemiacare.org.uk**

## Online Forum

Our online forum, **www.healthunlocked.com/leukaemia-care**, is a place for people to ask questions anonymously or to join in the discussion with other people in a similar situation.

## Webinars

Our webinars provide an opportunity to ask questions and listen to patient speakers and medical professionals who can provide valuable information and support. For information on upcoming webinars, go to **www.leukaemiacare.org.uk/support-and-information/support-for-you/onlinewebinars/**

## Website

You can access up-to-date information on our website, **www.leukaemiacare.org.uk**.

## Campaigning and Advocacy

Leukaemia Care is involved in campaigning for patient well-being, NHS funding and drug and treatment availability. If you would like an update on any of the work we are currently doing or want to know how to get involved, email **advocacy@leukaemiacare.org.uk**

## Patient magazine

Our magazine includes inspirational patient and carer stories as well as informative articles by medical professionals: **www.leukaemiacare.org.uk/communication-preferences/**

# Pathophysiology

Leukaemia predominantly arises from acquired genetic abnormalities in the haematopoietic system. Gene mutations may occur at any stage of cell maturation, preventing normal maturation and/or leading to uncontrolled proliferation to the detriment of normal cell production. Exposure to radiation or carcinogens may cause gene mutations, but the most common reason for the development of leukaemia is the accumulation of mutations associated with ageing.

Box 2.1 sets out some common terminology used when discussing the genetics of leukaemia that, in conjunction with the karyotype shown in Figure 2.1, will help guide you through the following sections.

Identifying mutations in leukaemia. Chromosomal abnormalities, identified by karyotyping of cells in metaphase, include recurrent duplications and deletions. In addition, one type of mutation that is common in leukaemia is a chromosome translocation, resulting in the formation of a novel fusion gene.

The classic fusion gene BCR-ABL1 is a product of a translocation between chromosomes 9 and 22, usually shown as t(9;22). The result of this chromosomal translocation is known as the 'Philadelphia chromosome' (see Figure 2.1).

The Philadelphia chromosome produces the fusion protein BCR-ABL, a constitutively active tyrosine kinase that has been implicated in the pathogenesis of CML, some cases of ALL (Philadelphia-positive [Ph+] ALL) and, rarely, AML. The importance of identifying driver mutations such as this is underlined in CML, in which tyrosine kinase inhibitors (TKIs) such as imatinib have revolutionised the lives of patients with this condition (Figure 2.2). Overexpression of genes may also occur by translocation to actively transcribed regions of the genome. For example, in B-cell ALL, expression of growth receptor gene CRLF2 is dysregulated by its translocation to the immunoglobulin (Ig) heavy chain locus.

Implications of identifying

**BOX 2.1**

## Common terminology in genetics

- Normal cells have 46 chromosomes (22 pairs of autosomes and two sex chromosomes, X or Y).
- The male karyotype (see Figure 2.1) is 46,XY and the female karyotype is 46,XX.
- Each chromosome has a long arm (q) and a short arm (p).
- Trisomy is the gain of an extra chromosome: for example, trisomy of chromosome 8 is denoted as +8.
- Monosomy is a form of genetic loss in which an entire chromosome is lost: for example, monosomy for chromosome 7 is denoted as –7.
- Chromosomal deletion (del) results in the loss of part of a chromosome during DNA replication. A deletion results from two breaks in either the short arm (p) or long arm (q) of the chromosome, with the loss of intervening material (interstitial deletion): for example, deletion of the long arm of chromosome 7 is denoted as del(7q).
- Chromosomal translocation (t) is the process by which a break occurs in the DNA in at least two different chromosomes, with exchange of genetic material between the chromosomes. Reciprocal or balanced translocation refers to an exchange with no obvious overall loss of chromosomal material; an example of a reciprocal translocation is the rearrangement giving rise to the Philadelphia chromosome, t(9;22), seen in CML (see Figure 2.1).
- Chromosomal inversion (inv) requires two breaks in the same chromosome with rotation of the intervening material: for example, inv(16)(p13.1q22).

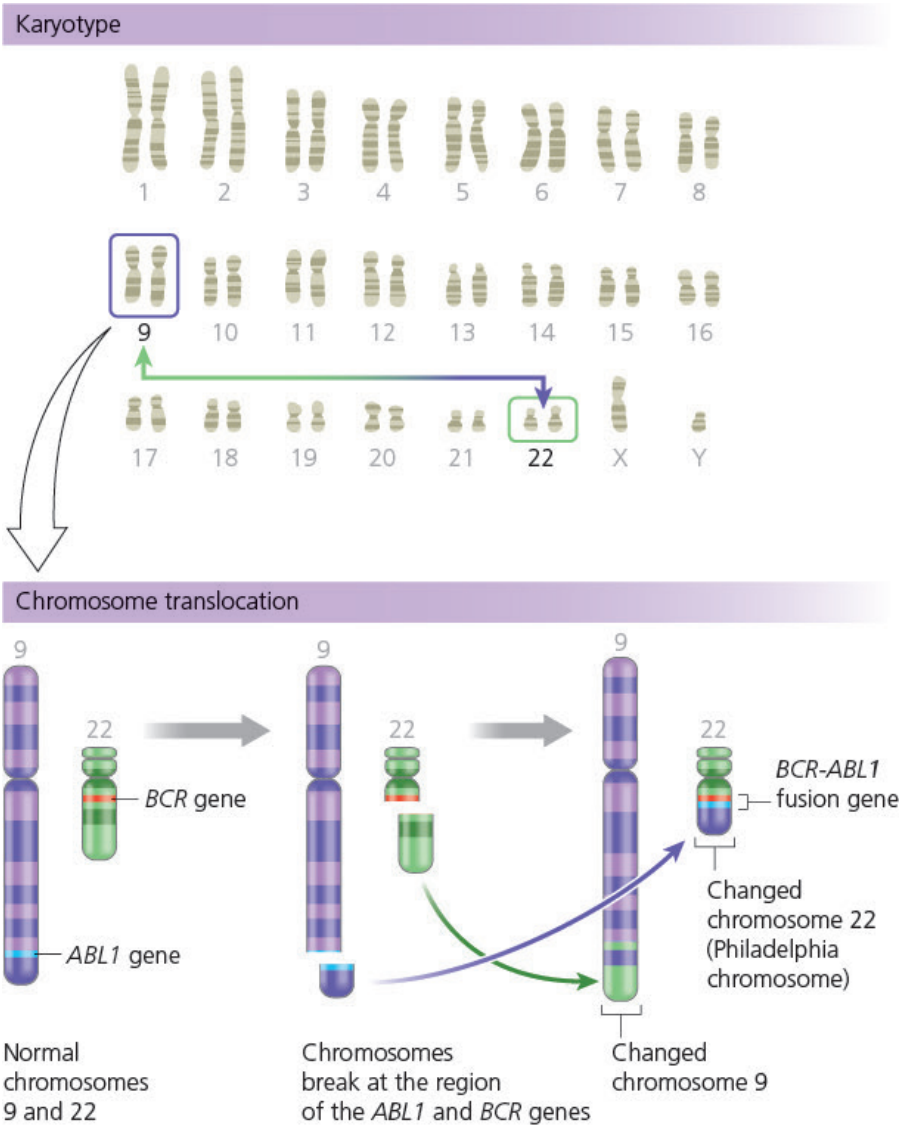


**Figure 2.1 Formation of the BCR-ABL1 fusion gene. Breakage of chromosomes 9 and 22, accompanied by aberrant repair, results in the ABL1 gene from chromosome 9 joining the BCR gene on chromosome 22. The result of this chromosomal translocation is called the Philadelphia chromosome. The BCR-ABL fusion gene is found in most patients with CML, and in some patients with ALL or AML.**

# Pathophysiology (cont.)

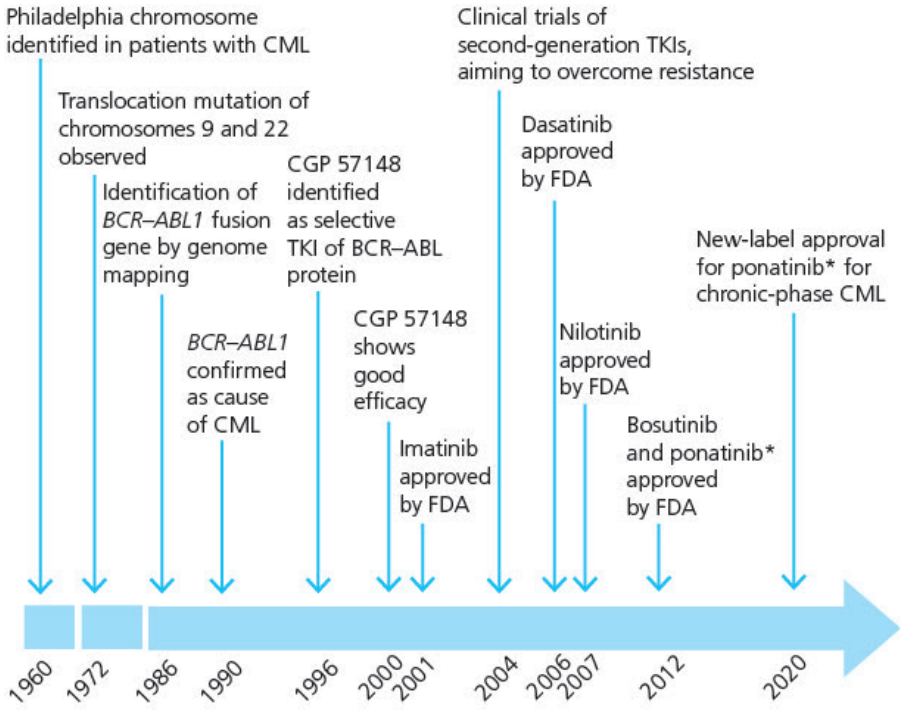


**Figure 2.2 Key events in the development of BCR-ABL TKIs for the treatment of CML, as a result of identifying the chromosomal translocation t(9;22). *Ponatinib is the only BCR-ABL TKI to show efficacy in patients with the T315I mutation. FDA, US Food and Drug Administration. Adapted from Lambert et al. 2013.**

mutations. The identification of chromosomal abnormalities in leukaemias has resulted in appropriate treatment stratification for patients with these conditions. For example, in AML, chromosomal translocation t(8;21) or chromosomal inversion inv(16), which are associated with RUNX1 and its binding partner CBFβ, respectively, are known as core-binding factor leukaemia and are associated with a good response to chemotherapy. In contrast, patients with a complex karyotype (a karyotype with multiple chromosomal mutations) or monosomy 5

# Pathophysiology (cont.)

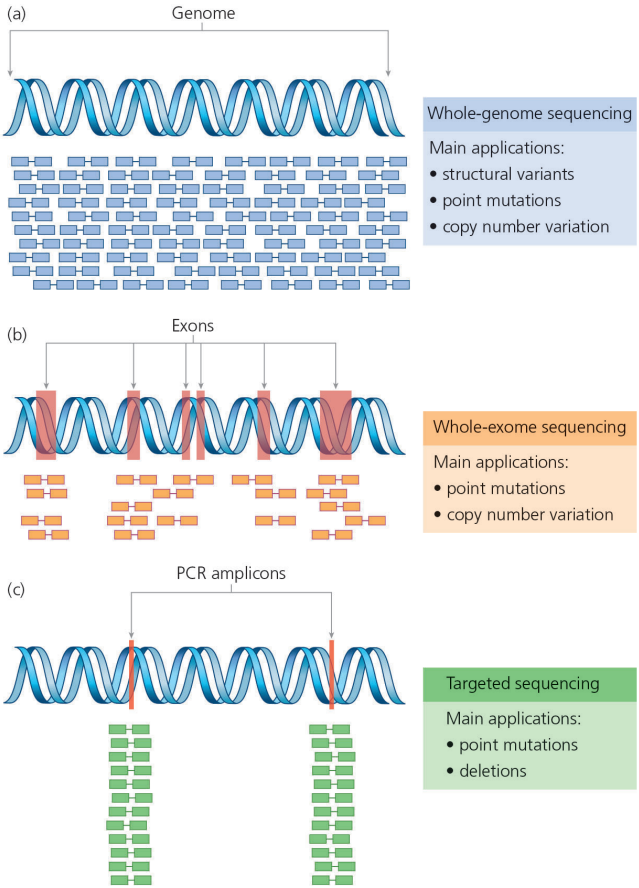


**Figure 2.3 NGS technologies. (a) Whole-genome sequencing includes both gene-coding and non-gene-coding regions of the entire genome. (b) Whole-exome sequencing focuses on the protein-coding regions of the genome, called the exons, which comprise 1–2% of the whole genome. (c) Targeted sequencing uses polymerase chain reaction (PCR) amplification to analyse specific regions of the genome, potentially providing a greater number of reads per genome base. This technology focuses only on the regions of genes where disease-related variants may be localised. Small targeted panels may be cost-effective, but offer limited opportunities to discover new mutations. Adapted from Simon and Roychowdhury 2013.**

or 7 (the presence of only one chromosome 5 or 7, rather than the normal pair) have a poor response to conventional cytotoxic chemotherapy and require further consolidation treatment such as allogeneic haematopoietic stem cell transplantation (HSCT). In CLL, patients with mutations affecting the p arm of chromosome 17 (17p) have a poor treatment response to conventional chemotherapy and are often managed with novel therapies, for example Bruton's tyrosine kinase (BTK) inhibitors such as ibrutinib.

NGS technology has revolutionised the discovery of mutations in leukaemia, allowing the sequencing of patient genomic DNA at continually decreasing costs. Figure 2.3 shows the principles behind the technology and the different utilities of the varied methods by which mutations can be identified. The first whole genome from a patient to be sequenced was that of a patient with AML; subsequently, thousands of AML genomes have been sequenced. Similarly, genomes from patients with ALL, CML and CLL have also been sequenced to provide novel insights into disease biology.

The comprehensive nature of these studies has provided evidence for new paradigms in our understanding of these different diseases. For example, NGS has identified mutations in whole new classes of genes. One such class comprises the genes responsible for the correct splicing of newly transcribed mRNA. These genes, such as U2AF1 and SRSF2, are frequently mutated in both AML and CLL. Knowledge of these genes has potentially important therapeutic implications, as they may become targets for new drugs. Characterisation of the mutations found in large cohorts of patients with AML is providing insights into the molecular cooperativity between different mutations, and further studies may suggest how they interact.

Mechanisms of leukaemogenesis. CLL and CML are both characterised by an increased number of maturing cells. However, in both ALL and AML the leukaemia is characterised by highly proliferative immature cells that are blocked from differentiating. Traditionally, in these acute leukaemias, it has been hypothesised that two mutations in the regulatory mechanisms of haematopoiesis are required (Figure 2.4).

A mutation in a master

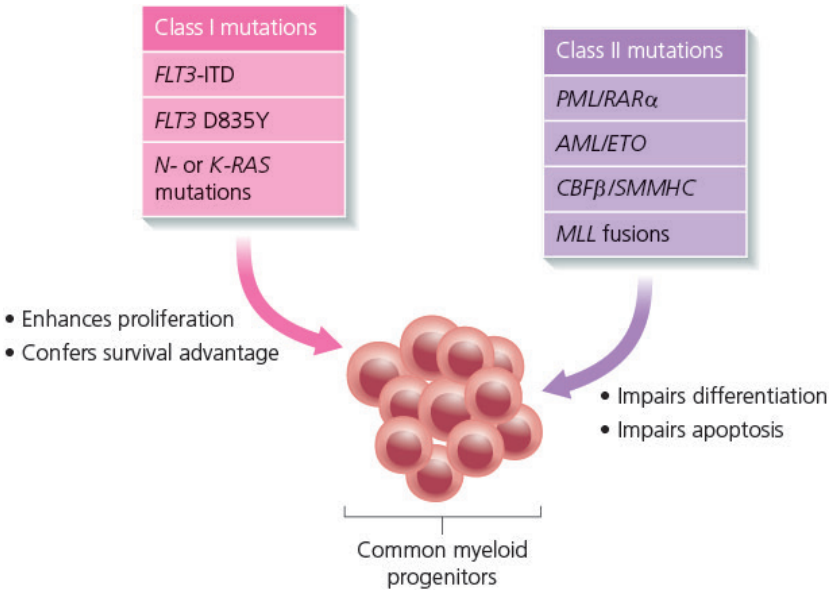


**Figure 2.4 The two-step model of leukaemogenesis in AML suggests that two mutations, each belonging to a different class, are required to produce the leukaemia, when neither is sufficient to do so in isolation. Class I mutations confer a proliferative and/or survival advantage to haematopoietic progenitors, while class II mutations impair differentiation and subsequent apoptosis. Although most of the recently identified mutations do not fit neatly into one of these two classes, they are thought to produce equivalent effects. Adapted from Gilland and Griffin 2002.**

regulatory transcription factor gene, such as RUNX1, results in a block in differentiation and an accumulation of immature progenitors that retain the ability to self-renew indefinitely. Mutations in epigenetic regulators, such as DNMT3A and IDH1 or IDH2, may also produce a similar result. A second mutation, often in a growth receptor pathway, provides an uncontrolled proliferative drive to these mutant cells; examples are mutations in FLT3 or RAS.

Many of these mutations are now targetable. For example, IDH2 inhibition can be achieved with enasidenib, resulting in differentiation of blasts and some clinically significant responses in patients with AML.

Clonal heterogeneity. In B-cell ALL, dynamic changes in the genome at different stages of treatment and relapse have been well characterised. These studies liken

# Pathophysiology (cont.)

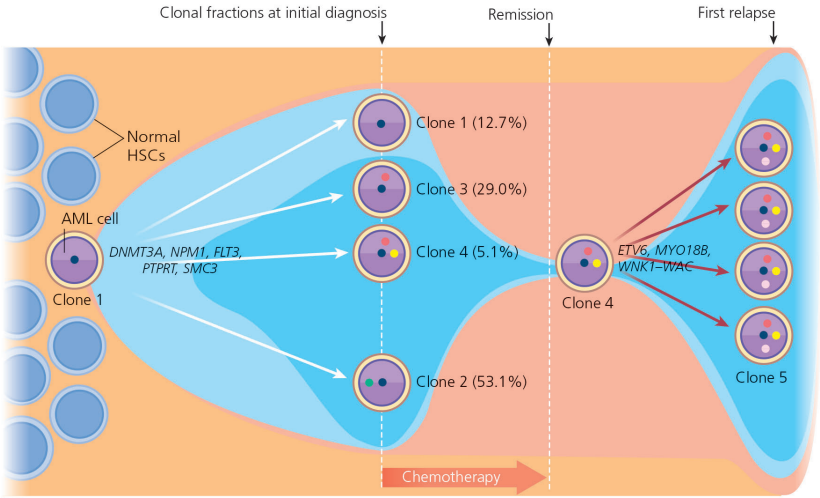


**Figure 2.5 Graphic representation of clonal evolution and heterogeneity in AML. Clone 1 is the founding clone and contains a cluster of somatic mutations (blue dot) in DNMT3A, NPM1, FLT3, PTPRT and SMC3. The coloured dots depicted within each of the clones represent different clusters of mutations. Several clonal subpopulations are identified at diagnosis: clones 2, 3 and 4 have all evolved from clone 1. The additional mutations in clones 2 and 3 may have conferred a proliferative advantage. Although only 5.1% of the tumour cells at diagnosis belonged to clone 4, indicating that this clone may have arisen last, clone 5 has evolved from clone 4, becoming the dominant clone at relapse. Clone 5 has acquired additional mutations in ETV6 and MYO18B, and a WNK1-WAC fusion gene. All of the tumour cells at relapse belong to clone 5, suggesting that one or more of the acquired mutations in clone 5 provided a strong selective advantage that contributed to the relapse. Adapted from Ding et al. 2012.**

# Pathophysiology (cont.)

cancer to an evolutionary process whereby a single malignant clone grows to dominate the leukaemic population. Clonal subpopulations can be defined by the mutations that they share.

In recent years, the use of NGS has enabled unbiased characterisation of these subpopulations through the identification of these mutations as clonal markers (Figure 2.5). Studies of paired samples in patients with AML at diagnosis, remission and relapse have shown that the clonal outgrowth at relapse comprises the same subpopulations that were present at diagnosis but not cleared during remission. This suggests that to achieve cures, these subpopulations must also be eradicated.

Leukaemic stem cells. Significant numbers of patients with leukaemia relapse with the same clone of cells as that identified at diagnosis, suggesting that a subpopulation within the clone has intrinsic treatment resistance. Relapse then occurs when this leukaemic stem cell population reconstitutes the leukaemia. The concept of leukaemic stem cells is well established in AML, where it has been shown that a single cell with a specific immunophenotype can reconstitute AML when transplanted into an immunodeficient mouse model in which the bulk blast cell populations have varying immunophenotypes. Subsequent work has focused on identifying specific cell markers for leukaemic stem cells and the mechanisms for self-renewal that facilitate their persistence. Other workers have sought to identify the related counterpart to leukaemic stem cells in normal haematopoiesis which, in both CML and AML, is likely to be an intermediate committed progenitor with aberrant self-renewal abilities (Figure 2.6).

In B-cell ALL, the model for leukaemic stem cells differs. There has been controversy over the precise immunophenotype of the leukaemic stem cell. Furthermore, there is less evidence of the same hierarchical structure in B-cell ALL as in AML. Nevertheless, leukaemia-initiating potential has been found in a wide range of clonal subpopulations.

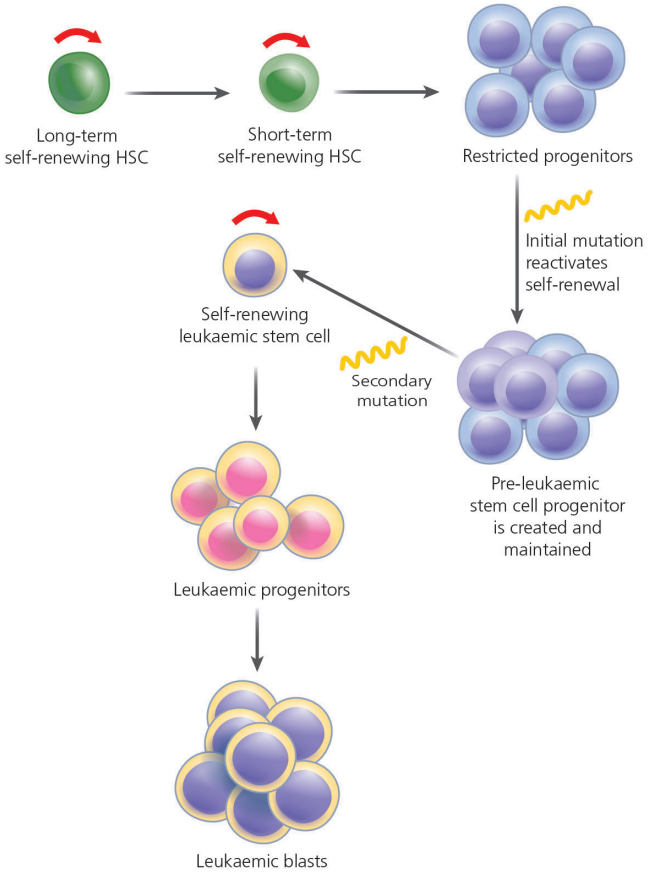


**Figure 2.6 Evidence suggests that leukaemias arise from a malignant counterpart to normal HSCs or progenitor cells that has been transformed by genetic and/or epigenetic aberrations. During normal haematopoiesis, HSCs differentiate into mature blood cells via progenitor populations in a series of lineage restriction steps. However, mutations may occur in the progenitors that have no inherent self-renewal activity. The initial mutation in the progenitor population must therefore confer the capacity to self-renew on these cells for the mutation to be propagated in a self-renewing leukaemic stem cell. The acquired mutations disrupt the normal HSC development, leading to the accumulation of leukaemic blasts.**

# Classification of leukaemias

As discussed at the start of this chapter, there are four main types of leukaemia:

- acute myeloid leukaemia (AML)
- acute lymphoblastic leukaemia (ALL)
- chronic myeloid leukaemia (CML)
- chronic lymphocytic leukaemia (CLL).

Acute myeloid leukaemia. The underlying pathophysiology in AML is the maturational arrest of early-stage myeloid progenitor cells as a result of chromosomal translocations and other genetic and/or epigenetic abnormalities (as discussed earlier in this chapter). The prognosis for a person with AML depends on the subtype of AML.

French–American–British (FAB) classification criteria. The traditional morphological classification of AML was based on the FAB criteria, which assigned patients to one of eight groups, designated M0–M7 based on morphological and cytochemical features (Table 2.1). Although the FAB classification has given way to classifications more reliant on recurrent genetic abnormalities, morphological findings remain a cornerstone of AML classification.

2016 World Health Organization (WHO) criteria. The WHO classification of AML recognises six subgroups:

- AML with recurrent genetic abnormalities (Table 2.2)
- AML with myelodysplasia-related features
- AML with therapy-associated myeloid neoplasms (the result of previous chemotherapy) and myelodysplastic syndrome (MDS)
- AML, not otherwise specified
- myeloid sarcoma
- myeloid proliferations related to Down's syndrome.

Both AML with myelodysplasia-related features and therapy-related AML have particularly poor prognosis and are associated with distinct cytogenetic abnormalities.

Blastic plasmacytoid dendritic cell neoplasm is a rare haematological neoplasm

## TABLE 2.1

## FAB classification of AML

| Subtype | Description |
|---|---|
| **M0** | Undifferentiated acute myeloblastic leukaemia |
| **M1** | Acute myeloblastic leukaemia with minimal maturation |
| **M2** | Acute myeloblastic leukaemia with maturation |
| **M3** | Acute promyelocytic leukaemia |
| **M4** | Acute myelomonocytic leukaemia |
| **M4eos** | Acute myelomonocytic leukaemia with eosinophilia |
| **M5** | Acute monocytic leukaemia |
| **M6** | Acute erythroid leukaemia |
| **M7** | Acute megakaryocytic leukaemia |

Note that this system has been superseded by the 2016 WHO and the 2017 European LeukemiaNet classifications, described in Tables 2.2 and 2.3, respectively, but is still useful for understanding the disease.

## TABLE 2.2

| 2016 WHO classification of AML with recurrent genetic abnormalities* |
|---|
| AML with t(8;21)(q22;q22.1); RUNX1–RUNX1T1 |
| AML with inv(16)(p13.1q22) or t(16;16)(p13.1;q22); CBFB–MYH11 |
| APML with PML–RARA† |
| AML with t(9;11)(p21.3;q23.3); MLLT3–KMT2A |
| AML with t(6;9)(p23;q34.1); DEK-NUP214 |
| AML with inv(3)(q21.3q26.2) or t(3;3)(q21.3;q26.2); GATA2, MECOM |
| AML (megakaryoblastic) with t(1;22)(p13.3;q13.3); RBM15-MKL1 |
| AML with mutated NPM1 |
| AML with biallelic mutations of CEBPA |
| Provisional entities |
| AML with mutated RUNX1 |
| AML with BCR–ABL1 |

*Marrow blast count of ≥ 20% is required, except for AML with the following recurrent genetic abnormalities: t(15;17), t(8;21), inv(16) or t(16;16).

†APML is a specific subtype of AML that results from the t(15;17) chromosome translocation. APML, acute promyelocytic leukaemia.

Adapted from Arber et al. 2016.

# Classification of leukaemias (cont.)

arising from the precursors of plasmacytoid dendritic cells. While it was discussed as a type of myeloid leukaemia in the 2008 WHO classification, insight into the origin and development of the tumour has resulted in its classification as a unique category.

2017 European LeukemiaNet (ELN) risk classification criteria. The ELN criteria further stratify AML subtypes by the level of risk different genetic abnormalities confer (Table 2.3).

Acute lymphoblastic leukaemia. The underlying pathophysiology in ALL is the maturational arrest of early-stage lymphoid progenitor cells due to chromosomal translocations and other genetic and/or epigenetic abnormalities (as discussed earlier in this chapter). The prognosis for a person with ALL depends on the subtype of ALL.

FAB classification criteria. Historically, this morphological classification of ALL assigned patients to one of three groups (Table 2.4). The FAB classification has now given way to the WHO classification.

2016 WHO classification criteria. These subdivide ALL into B- and T-cell ALL. These two entities are morphologically indistinguishable. B-cell ALL, which is more common than T-cell ALL in both adults and children, is further subtyped according to recurrent genetic abnormalities (Table 2.5). As with AML, these different genetic abnormalities convey different prognostic characteristics to the leukaemia.

Problems distinguishing AML from ALL. At times, acute leukaemias may not be readily subdivided into ALL or AML. This may be because of the presence of multiple populations of blasts of differing lineage, or a single population of blasts with the expression of markers associated with different lineages.

Chronic myeloid leukaemia is characterised by increased proliferation of the myeloid cell line without the loss of differentiation. In the natural history of the condition, the disease progresses through three general phases:

- chronic stable phase
- accelerated phase

## TABLE 2.3

## 2017 ELN risk stratification of AML by genetic abnormalities

| Level of risk* | Genetic abnormality |
|---|---|
| **Favourable** | • t(8;21)(q22;q22); RUNX1–RUNX1T1<br>• inv(16)(p13.1q22) or t(16;16)(p13.1;q22); CBFB–MYH11<br>• Mutated NPM1<br>– without FLT3-ITD<br>– with FLT3-ITD (with low allelic ratio < 0.5)<br>• Biallelic mutated CEBPA |
| **Intermediate** | • Mutated NPM1 and FLT3-ITD (with high allelic ratio ≥ 0.5)<br>• Wild-type NPM1<br>– without FLT3-ITD<br>– with FLT3-ITD (with low allelic ratio < 0.5 and without adverse-risk genetic lesions)<br>• t(9;11)(p21.3;q23.3); MLLT3–KMT2A (the presence of t(9;11)(p21.3;q23.3) takes precedence over rare, concurrent adverse-risk gene mutations)<br>• Cytogenetic abnormalities not classified as favourable or adverse |
| **Adverse** | • t(6;9)(p23;q34.1); DEK–NUP214<br>• t(v;11q23.3); KMT2A rearranged<br>• t(9;22)(q34.1;q11.2); BCR–ABL1<br>• inv(3)(q21.3q26.2) or t(3;3)(q21.3;q26.2); GATA2, MECOM(EVI1)<br>• Monosomy –5 or del(5q); monosomy –7; monosomy –17/abn(17p)<br>• Complex karyotype (≥ 3 unrelated chromosome abnormalities in the absence of one of the WHO-criteria recurring translocations or inversions [see Table 2.2])<br>• Monosomal karyotype (1 single monosomy [excluding loss of X or Y] with ≥ 1 additional monosomy or structural chromosome abnormality [excluding core-binding factor AML])<br>• Wild-type NPM1 with FLT3-ITD (with high allelic ratio ≥ 0.5)<br>• Mutated RUNX1 (not to be classified as adverse risk if co-occurs with favourable-risk AML subtypes)<br>• Mutated ASXL1 (not to be classified as adverse risk if co-occurs with favourable-risk AML subtypes)<br>• Mutated TP53 (significantly associated with complex- and monosomal- karyotype AML) |

*Depends on treatment; may change with new therapies.

ITD, internal tandem duplication. Adapted from Döhner et al. 2017.

## TABLE 2.4

## FAB classification of ALL

| Subtype | Description |
|---|---|
| **ALL-L1** | Small cells with homogeneous nuclear chromatin, a regular nuclear shape, small or no nucleoli, scanty cytoplasm and mild-to-moderate basophilia |
| **ALL-L2** | Large heterogeneous cells with variable nuclear chromatin, an irregular nuclear shape, ≥1 nucleoli, variable quantity of cytoplasm and variable basophilia |
| **ALL-L3** | Large homogeneous cells with fine stippled chromatin, a regular nuclear shape, prominent nucleoli and abundant, deeply basophilic cytoplasm; prominent cytoplasmic vacuolation is the most distinguishing feature |

## TABLE 2.5

## 2016 WHO classification of ALL

| **B-cell ALL/lymphoma with recurrent genetic abnormalities** |
|---|
| • t(9;22)(q34;q11.2); BCR-ABL1 |
| • t(v;11q23); MLL rearranged |
| • t(12;21)(p13;q22); TEL-AML1 (ETV6-RUNX1) |
| • Hyperdiploidy |
| • Hypodiploidy |
| • t(5;14)(q31;q32); IL3-IGH |
| • t(1;19)(q23;p13.3); TCF3-PBX1 |
| **Provisional entities** |
| • BCR-ABL1-like |
| • iAMP21 |

| T-cell ALL/lymphoma |
|---|
| Provisional entities |
| • Early T-cell precursor lymphocytic leukaemia |
| • Natural killer-cell lymphocytic leukaemia |

Adapted from Arber et al. 2016.

# Classification of leukaemias (cont.)

- blast phase.

The WHO and ELN suggest different diagnostic criteria for these phases; this includes the response to TKIs in modern practice. Recognition of these different disease phases is important as they relate to greatly differing responses to treatment and may suggest the need for more aggressive treatment, such as stem cell transplantation.

Chronic lymphocytic leukaemia can be subtyped by its staging classification, which is similar to lymphoma staging. There are two main stages:

- asymptomatic early-stage disease
- symptomatic or advanced-stage disease.

CLL and the indolent lymphoma small lymphocytic lymphoma (SLL) are considered by the WHO classification scheme to be the same disease in different clinical phases.

Other forms of leukaemia can also be of lymphoid or myeloid origin; they include:

- hairy cell leukaemia (HCL)
- chronic myelomonocytic leukaemia
- juvenile myelomonocytic leukaemia
- large granular lymphocytic (LGL) leukaemia
- B-cell prolymphocytic leukaemia (B-PLL).

# Key points – what is leukaemia?

- The four main types of leukaemia are AML, ALL, CML and CLL.
- The acute leukaemias are predominantly characterised by the uncontrolled growth of immature poorly differentiated cells blocked from further differentiation.
- The chronic leukaemias are characterised by maturing proliferative later-stage cells.
- The main reason for the development of leukaemia is the accumulation of gene mutations over time. Chromosome translocations are common.
- The identification of chromosomal abnormalities aids treatment stratification in patients. NGS technologies have revolutionised the discovery of mutations in leukaemia.
- Malignant clonal subpopulations identified at diagnosis have also been identified at relapse.
- Prognosis depends on the genetic abnormalities involved and other clinical factors.

Leukaemia Care is a national charity dedicated to providing information, advice and support to anyone affected by a blood cancer.

Around 34,000 new cases of blood cancer are diagnosed in the UK each year. We are here to support you, whether you're a patient, carer or family member.

## Want to talk?


Helpline: **08088 010 444**
(free from landlines and all major mobile networks)

Office Line: **01905 755977**

**www.leukaemiacare.org.uk**

**support@leukaemiacare.org.uk**

Leukaemia Care,
One Birch Court,
Blackpole East,
Worcester,
WR3 8SG



Leukaemia Care is registered as a charity in England and Wales (no.1183890) and Scotland (no. SC049802).
Company number: 11911752 (England and Wales).
Registered office address: One Birch Court, Blackpole East, Worcester, WR3 8SG


Leukaemia Care
YOUR Blood Cancer Charity