Leukaemia Diagnosis Interpretation

All Participants

Participant:  
Trial No:  202105
Date Issued:  22-January-2021  
Closing Date:  11-February-2021

Trial Comments
This exercise was issued to 748 participants. This is the final version of the report.

Your Diagnosis:  
AML with mutated NPM1

Consensus Diagnosis:  
AML with mutated NPM1

Your Diagnosis is classed as being CORRECT DIAGNOSIS (on rare occasions this may not be the consensus diagnosis).

Performance Score:  
0

Running Score:  
50

Consensus

<table>
<thead>
<tr>
<th>Consensus Diagnosis</th>
<th>Your Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lineage</td>
<td>Myeloid Neoplasms</td>
</tr>
<tr>
<td>Subclassification</td>
<td>Acute myeloid leukemia (AML) and related precursor neoplasms</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>AML with mutated NPM1</td>
</tr>
</tbody>
</table>

Breakdown of returned diagnoses

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No Of Returns</th>
<th>Percentage Of Returns</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Returns</td>
<td>353</td>
<td>100.0</td>
</tr>
<tr>
<td>AML with mutated NPM1</td>
<td>289</td>
<td>81.9</td>
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<tr>
<td>Acute monoblastic and monocytic leukaemia</td>
<td>47</td>
<td>13.3</td>
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<tr>
<td>Acute myelomonocytic leukaemia</td>
<td>9</td>
<td>2.5</td>
</tr>
<tr>
<td>Chronic myelomonocytic leukaemia</td>
<td>4</td>
<td>1.1</td>
</tr>
<tr>
<td>AML without maturation</td>
<td>1</td>
<td>0.3</td>
</tr>
<tr>
<td>Myeloproliferative neoplasm, unclassifiable</td>
<td>1</td>
<td>0.3</td>
</tr>
<tr>
<td>Acute promyelocytic leukaemia with PML-RARA</td>
<td>1</td>
<td>0.3</td>
</tr>
<tr>
<td>Mixed phenotype acute leukaemia, NOS, rare types</td>
<td>1</td>
<td>0.3</td>
</tr>
</tbody>
</table>

Conclusion

Issue Date: 06 Apr 2021

Di 202105 1.0.0

Uncontrolled Copy
DO YOU OR ANY OF YOUR COLLEAGUES ASSESS BONE MARROW MORPHOLOGY? DID YOU KNOW THAT WE ALSO RUN A DIGITAL BONE MARROW ASPIRATE PROGRAMME FOR HAEMATO-ONCOLOGY? SEE PAGE 13

Clinical History/Information

A 36-year-old male was rushed to Accident and Emergency after having felt unwell suddenly.

Haematological examination revealed the following: Hb: 100 g/L; WBC: 81.06 x10^9/L; Plt: 31 x10^9/L; RBC: 3.08 x10^12/L; HCT: 0.306 L/L; MCV: 99.4 fL; MCH: 32.5 pg

The bone marrow aspirate was inadequate and particulate due to poor quality of smear spread. Cellularity was haemodiluted; Megakaryopoiesis was decreased but with normal morphology and erythropoiesis was reduced. Granulopoiesis was markedly increased and blasts were increased within 88%. Some cells seen with cytoplasmic inclusion suggestive of haemophagocytosis.

Differential: Erythroid 2%; Myeloid 0%; Lymphoid 0%; Lymphocytes 0%; Plasma Cells 0%; Neutrophils 4%; Metamyelocytes 1%; Myelocytes 5%; Promyelocytes 0%; Blasts 88%; Cells Counted 200. Monocytoid cells seen.

Immunophenotype

The consensus results of the malignant cells had the following phenotype (consensus percentage expression in brackets rounded to the nearest whole percentage).

Positive antigens: CD4 (85%), CD11b (97%), CD11c (98%), CD13 (54%), CD14 (58%), CD33 (94%), CD36 (98%), CD38 (92%), CD45 (100%), CD64 (97%), HLADR (96%).

Negative antigens – CD2 (0%), CD3 (0%), CD5 (0%), CD7 (0%), CD10 (0%), CD15 (24%), CD19 (0%), CD20 (0%), CD34 (0%), CD56 (17%), CD117 (0%), MPO (12%).

Peripheral Blood Morphology - Please note that these comments are based on the clinical history, full blood count results, and digital image alone; no other results have been taken into consideration.

Morphology comments from Professor Wendy Erber
Consultant Haematologist, University of Western Australia, Nedlands, Australia

There is a marked monocytosis. This includes promonocytes and abnormal monocytes with abnormal nuclear morphology and many with azurophilic cytoplasmic granules. No Auer rods seen. Few undifferentiated blast cells are seen.

COMMENT: Blood film appearances are consistent with acute monocytic leukaemia
Example of Digital Images Issued

Figure 1: Peripheral Blood x50 magnification (May Grünwald Giemsa stain)

Figure 2: Peripheral Blood x100 magnification (May Grünwald Giemsa stain)
Cytogenetics and Molecular genetics

PLEASE NOTE: Participants should be aware that, when submitting a diagnosis, if a specific pathology that has not been provided in the clinical details (e.g. molecular or cytogenetics) has been assumed to be present or introduced this will result in their diagnosis being classified as incorrect.

Cytogenetics:
FISH analysis of 100 interphase nuclei has shown no evidence of a MECOM or KMT2A rearrangement (99% chance of detecting a 5% cell population).

Molecular genetics:
NPM1: 4bp Insertion detected; FLT3 ITD: High allelic ratio 42bp ITD detected; FLT3 TKD: No pathogenic variant detected.

Histology:
Histological examination of a single core of bone marrow trephine measuring 12 mm showed increased cellularity (approximately 90%) with occasional and disturbed erythropoiesis. Granulopoiesis was increased greatly without maturation; monocytoid cells were seen. Thrombopoiesis was not identified on H&E; Reticulin staining was normal; MF=0 (WHO 2016; European Consensus 2005).
Exercise Conclusion/Case Discussion

Please note that because this trial requires a diagnosis based on the WHO guidelines, this report is therefore based on the WHO Classification of Tumours of Haematopoietic and Lymphoid Tissues, Revised 4th Edition 2017\(^1\).

Findings from Leukaemia Immunophenotyping - Part One

Robust means and robust standard deviations (SDs) are generated for each antigen for information only and are not performance monitored. The statistics generated for this exercise showed high SDs for some antigens. It is suspected that this could be due to incorrect gate placement. We ask that participants gate on the malignant population, but it is impossible to determine if this has been carried out.

Please note - No data is shown in the Specific Statistics sections for CD38 (manufacturer) and CD4, CD11b, CD38, CD45 and CD56 (fluorochrome). Sufficient returns were received for overall statistics to be generated but returns for specific manufacturer and/or fluorochrome were <20 for each of these antigens therefore statistics were not calculated.

The antigens reported by participants showed good consensus with respect to the most commonly used manufacturers and fluorochromes.

Currently, clone information is not collected by UK NEQAS LI but as part of the Leukaemia programme redesign, this information will be requested once the updated system is implemented.

Based on comments received, we are aware that a very small number of participants found this case challenging in deciding the leukaemic or population of interest as the monocyte population expressed mature immunophenotypic markers. We would encourage participants to make use of the digital image provided with the part 1 exercise; these images are taken from blood films made directly from the patient’s sample (pre-dilution with donated whole blood) and thus will assist in identifying the population of interest. Looking at the image provided would have left participants in no doubt as to the population of interest for this exercise.

For robust means of all antigens based on manufacturer and fluorochromes please see the LI 202105 report.
Leukaemia Diagnostic Interpretation - Part Two

Consensus Diagnosis
AML with mutated NPM1

Correct Diagnoses
AML with mutated NPM1 is one of the most common and relatively specific mutations in AML and is more commonly seen in women. It is present in between 2% and 8% of childhood cases, between 27% and 35% of adult cases and between 45% and 64% of cases in adults with a normal karyotype. Patients present with anaemia and thrombocytopenia and higher white cell and platelet counts than seen in other types of AML. Extramedullary site involvement can occur e.g., the skin, the gingiva and lymph nodes.

The NPM1 mutation is often seen in cases of acute myelomonocytic leukaemia, acute monocytic leukaemia, AML with maturation and AML without maturation. Dysplasia is seen in multiple lineages in up to 25% of de novo AML with mutated NPM1.

Immunophenotypically there is strong expression of CD33 and variable expression of CD13. Expression of CD117, CD123 and CD110 is common but HLADR tends to be negative. Two main subgroups of AML with mutated NPM1 have been reported: one with a monocytic immunophenotypic profile (positive for CD14, CD36 and CD64) and the other with an immature myeloid profile. CD34 is negative in most cases but if CD34 positive, it is often associated with an adverse prognosis.

This genetic mutation is usually associated with a normal karyotype. Chromosomal changes are detected in 5% to 15% of cases including the gain of chromosome 8 and del(9q) which is considered a myelodysplasia-related abnormality in the majority of AMLs but not when a mutation of NPM1 is present. Secondary mutations are common with NPM1 mutations, with mutations of FLT3 and DNMT3A being the most frequently seen.

This diagnosis is classed as correct because the immunophenotype and molecular genetics (NPM1 mutation) match that of this case.

Differential Diagnoses
None for this exercise

Incorrect Diagnoses
Acute monoblastic and monocytic leukaemia accounts for less than 5% of cases of AML. It can occur at any age but is most common in the young.

Morphologically, the monoblasts are large cells with a large amount of cytoplasm which can be moderately to strongly basophilic. There may be vacuoles and fine scattered azurophilic granules. The nuclei are usually round, the chromatin is lacy, and one or more nucleoli may be present. The promonocytes have more irregular nuclei which are slightly convoluted. The cytoplasm is more granulated with the occasional large azurophilic granule and vacuoles are present. Auer rods are rare. Haemophagocytosis may be seen.
Immunophenotypically, there is variable expression of CD13, CD15, CD33, CD65 and generally the expression of 2 monocyte markers such as CD4, CD11b, CD11c, CD14, CD36, CD64, CD68 and lysozyme. There is expression of CD34 in 30% of cases and CD117 is more commonly expressed. The majority of cases are positive for HLADR with Myeloperoxidase (MPO) expressed in acute monocytic leukaemia but less often in acute monoblastic leukaemia.

The t(8;16)(p11.2;p13.3) mutation can be associated with this disease but it is most often associated with haemophagocytosis (most commonly of erythrocytes) by the leukaemic cells and with coagulopathy. Myeloid associated, non-specific cytogenetic abnormalities are seen in the majority of cases.

This diagnosis is classed as incorrect because it fails to take into account the NPM1 mutation which has specific impact on prognostication.

**Acute myelomonocytic leukaemia (AMML)** accounts for between 5% and 10% of cases of AML. It can occur at any age but is more prevalent in older individuals (median age 50). The clinical symptoms include anaemia, thrombocytopenia, fever, and fatigue. The white cell count may also be elevated.

The monocytes and their precursors make up more than 20% of the bone marrow cells and so do neutrophils and their precursors. Morphologically, the monoblasts are large cells with a large amount of cytoplasm which can be moderate to strongly basophilic. There may be vacuoles and fine scattered azurophilic granules. The nuclei are usually round, the chromatin is lacy, and one or more nucleoli may be present. The promonocytes have more irregular nuclei which are slightly convoluted. The cytoplasm is more granulated with the occasional large azurophilic granule and vacuoles are present. The monoblasts and promonocytes cannot always be distinguished from the maturing myeloid cells in bone marrow (BM) films. The peripheral blood (PB) commonly shows a more evident number of maturing monocytes than seen on the BM.

The immunophenotype shows variable expression of CD13, CD15, CD33 and CD65, and one of the blast populations is usually positive for monocyte markers. The expression of CD15, CD36 and strong expression of CD64 shows monocytic differentiation. Frequently there is a population which expresses CD34 and/or CD117. Positivity for HLADR is seen in most cases.

Myeloid associated non-specific cytogenetic abnormalities, e.g., gain of chromosome 8, are seen in the majority of cases.

This diagnosis is classed as incorrect because it fails to take into account the NPM1 mutation which has specific impact on prognostication. In addition, the differential from the BM shows that neutrophils and their precursors make up less than 20% of the bone marrow cells.

**Chronic myelomonocytic leukaemia (CMML)** is a malignancy that exhibits features of myelodysplastic syndrome (MDS) and myeloproliferative neoplasm (MPN). In terms of incidence, a couple of recent studies have shown an incidence of 0.41 cases per 100 000 population with a slight male bias and median age of 65-75 years.

The spleen, liver, skin and lymph nodes often show leukaemic infiltration. Peripheral blood (PB) and bone marrow (BM) are always involved. Increased white cell count (WCC), weight loss, fever, night sweats, fatigue, infection, and bleeding due to thrombocytopenia are all clinical features that may be associated with CMML. Splenomegaly and hepatomegaly may also be observed.
Main feature of CMML is PB monocytosis greater than $1 \times 10^9$/L and these should make up greater than 10% of the total WCC. Although the monocytes may show unusual patterns, they usually tend to be mature and unremarkable morphologically. Blasts and promonocytes may be present but if they make up greater than 20% of the WCC, it is suggestive of acute myeloid leukaemia (AML) instead. In the BM, the most obvious feature tends to be granulocytic proliferation.

CMML is subdivided into three types based on the percentage of blasts and promonocytes in the PB and BM. For CMML-0, there should be less than 2% and 5% of blasts in the PB and BM respectively with no Auer rods seen. For CMML-1, there is between 2% to 4% and 5% to 9% blasts in the PB and BM respectively with no Auer rods seen. For CMML-2, there is between 5% to 19% and 10% to 19% blasts in the PB and BM respectively; Auer rods are seen.

Immunophenotypically, the cells express the usual myelomonocytic antigens such as CD13 and CD33 with variable expression of CD14, CD64 and CD68. Aberrant features are often observed in the monocytes with decreased CD11c, CD13, CD14, CD15, CD16, CD36, CD64 and HLA-DR being the most common. Aberrant expression of CD2 has also been observed.

Genetically, gain of chromosome 8 and loss of chromosome 7 or del(7q) are the frequently observed recurrent abnormalities.

This diagnosis is classed as incorrect because it fails to take into account the $NPM1$ mutation which has specific impact on prognostication. Moreover, the morphology and immunophenotype do not match that of this case.

**AML without maturation** accounts for between 5% and 10% of cases of AML. The majority of patients are adult (median age 46) and often present with anaemia, neutropenia and thrombocytopenia. Leucocytosis with increased blasts is present in some cases.

Morphologically there is a high blast percentage with no significant maturation to neutrophils in the bone marrow and less than 10% of the nucleated bone marrow cells will be maturing granulocytes. Auer rods and/or azurophilic granules are present in myeloblasts but the blast cells may resemble lymphoblasts in other cases; lacking azurophilic granules. A variable proportion of the blasts (always greater than 3%) are positive for MPO or Sudan Black.

Immunophenotypically a population of blasts shows positivity for MPO and one or more myeloid-associated antigens such as CD13, CD33 and CD117. In approximately 70% of cases, CD34 and HLA-DR are expressed. There is generally no expression of CD15 and CD65 (granulocytic maturation), CD14 and CD64 (monocytes) however, CD11b is positive in a few cases. The blasts are negative for cCD3, cCD79a, and cCD22. CD7 is expressed in approximately 30% of cases.

There are no recurrent chromosomal abnormalities associated with this disease.

This diagnosis is classed as incorrect because it fails to take into account the $NPM1$ mutation which has specific impact on prognostication. Moreover, the morphology and immunophenotype do not match that of this case.

**Myeloproliferative neoplasm (MPN), unclassifiable (MPN-U)** is a disease entity used to classify obvious features and presentations of an MPN but does not either come under any of the specific MPN categories or has overlapping features of two or more MPN entities. MPN-U may account for less than 5% of all MPNs.
The spleen and liver may be affected in advanced stages of the disease and these may be characterised by splenomegaly and hepatomegaly. Peripheral blood (PB) and bone marrow (BM) tend to be the main sites affected. Anaemia, increased white cell count, thrombocytosis and cytopenia are all clinical features that may be observed.

The presence of more than 10% blasts in the PB and BM may indicate progression to a more aggressive form of the disease. The accelerated phase is characterised by between 10% to 19% blasts with blasts percentage of more than 20 suggestive of transformation to acute leukaemia.

There is no specific genetic profile for this disease entity. The presence of BCR-ABL1 fusion, rearrangement of PDGFRA, PDGFRB, or FGFR1, PCM1-JAK2 fusion; JAK2, CALR, or MPL mutations will suggest a diagnosis of MPN instead of MPN-U.

This diagnosis is classed as incorrect because it fails to take into account the NPM1 mutation which has specific impact on prognostication. In addition, the presentation, immunophenotype and morphology do not match that of this case. The differential from the BM shows a blast percentage of 88% which is suggestive of an acute myeloid leukaemia.

**Acute promyelocytic leukaemia with PML-RARA** accounts for 5% to 8% of cases of AML in younger patients and is seen most frequently in middle-aged adults. Two types of this disease exist – microgranular (hypogranular) and hypergranular and both are frequently associated with disseminated intravascular coagulation which is associated with a significant number of early deaths in such patients. In the microgranular form, the WCC is extremely high with a short doubling time compared with the hypergranular type.

The nuclei of the promyelocytes in hypergranular APL with PML-RARA are irregular and vary in size. They are often bilobed or kidney-shaped. Densely packed granules in the cytoplasm often obscure the boundary of the nucleus and the cytoplasm. Bundles of auer rods are common. These cells are occasionally seen in the peripheral blood (PB).

In the hypogranular form, the nuclei of the promyelocytes are mostly bilobed and the granules are either low in number or absent. This may sometimes lead to confusion with acute monocytic leukaemia but often a few abnormal promyelocytes with obvious granules and/or bundles of Auer rods are seen.

The immunophenotype differs between the two forms. In the hypergranular form, the expression of CD11a, CD11b, CD18, CD34 and HLA-DR is weak or absent. There is strong expression of CD33, moderate expression of CD13, CD117 is positive in majority of cases. CD15 and CD65 are weak or absent and CD64 is positive. In the hypogranular type, CD34 and CD2 are frequently positive, CD11c can be expressed and CD56 expression is seen in 10% of cases.

In acute promyelocytic leukaemia with PML-RARA, the RARA gene on 17q21.2 fuses with the PML gene on 15q24.1 to produce the PML-RARA fusion gene. There have been very rare cases identified without the classic t(15;17)(q24.1;q21.2) translocation but these cases have either complex variant translocations involving chromosomes 15 and 17 with an additional chromosome or a cryptic insertion of RARA into PML on a submicroscopic level. FLT3 mutations are found in between 30% and 40% of cases. Secondary cytogenetic abnormalities may be found in about 40% of case with gain of chromosome 8 being the most common.

This diagnosis is classed as incorrect because it fails to take into account the NPM1 mutation which has specific impact on prognostication. It also assumes the presence of PML-RARA fusion gene
which is not found in this case. Moreover, the morphology and immunophenotype do not match that of this case.

**Mixed-phenotype acute leukaemia (MPAL) not otherwise specified (NOS), rare types** is a category used to classify certain very rare cases of leukaemia where there is a definite expression of B and T-cell lineage markers in the leukaemic blast population. There have been reports of cases where the leukaemic blasts show B and T-cell as well as myeloid lineage markers (trilineage).

These cases are extremely rare and as such there is not much data with regards to clinical features or genetic profile.

This diagnosis is classified as incorrect because it does not take into consideration the *NPM1* mutation. In addition, the immunophenotype does not match as the leukaemic blasts in this case do not exhibit bi or tri-lineage markers.
# Further Trial Findings

Table 1: Submissions from all participants - laboratories and individuals.

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Total No. of Returns</th>
<th>No. of Laboratories</th>
<th>No. of Individuals</th>
</tr>
</thead>
<tbody>
<tr>
<td>AML with mutated NPM1</td>
<td>289</td>
<td>183</td>
<td>106</td>
</tr>
<tr>
<td>Acute monoblastic and monocytic leukaemia</td>
<td>47</td>
<td>30</td>
<td>17</td>
</tr>
<tr>
<td>Acute myelomonocytic leukaemia</td>
<td>9</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>Chronic myelomonocytic leukaemia</td>
<td>4</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>AML without maturation</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Myeloproliferative neoplasm, unclassifiable</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Acute promyelocytic leukaemia with PML-RARA</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Mixed phenotype acute leukaemia, NOS, rare types</td>
<td>1</td>
<td>0</td>
<td>1</td>
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Table 2: Submissions from laboratory participants only

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<thead>
<tr>
<th>Diagnosis</th>
<th>Laboratory Returns %</th>
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<tbody>
<tr>
<td>AML with mutated NPM1</td>
<td>83.2</td>
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<tr>
<td>Acute monoblastic and monocytic leukaemia</td>
<td>13.6</td>
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<tr>
<td>Acute myelomonocytic leukaemia</td>
<td>1.4</td>
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<tr>
<td>Chronic myelomonocytic leukaemia</td>
<td>0.9</td>
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<tr>
<td>Myeloproliferative neoplasm, unclassifiable</td>
<td>0.5</td>
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<tr>
<td>Acute promyelocytic leukaemia with PML-RARA</td>
<td>0.5</td>
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### Table 3: Submissions from individual participants only

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Individual Returns %</th>
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<tbody>
<tr>
<td>AML with mutated NPM1</td>
<td>79.7</td>
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<tr>
<td>Acute monoblastic and monocytic leukaemia</td>
<td>12.8</td>
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<td>Acute myelomonocytic leukaemia</td>
<td>4.5</td>
</tr>
<tr>
<td>Chronic myelomonocytic leukaemia</td>
<td>1.5</td>
</tr>
<tr>
<td>AML without maturation</td>
<td>0.8</td>
</tr>
<tr>
<td>Mixed phenotype acute leukaemia, NOS, rare types</td>
<td>0.8</td>
</tr>
</tbody>
</table>

Reference:
NOTICE: Do you or any of your colleagues assess bone marrow morphology? Did you know that we also run a digital bone marrow aspirate programme for Haematological Oncology? For further details or to join please visit our website www.ukneqasli.co.uk or email admin@ukneqasli.co.uk. This is low cost and can count towards your Continued Professional Development (CPD) portfolio.

Haematological Malignancy Bone Marrow Aspirate Assessment (Not Accredited)

www.ukneqasli.co.uk
Haematological
Malignancy Bone
Marrow Aspirate
Assessment
(Not Accredited)

This programme is designed to assess
the ability of participants to identify
cell types in a bone marrow aspirate.

Sheffield Teaching Hospitals
NHS Foundation Trust, a UKAS
proficiency testing provider No. 7804,
operating UK NEQAS for Leucocyte
immunophenotyping.

<table>
<thead>
<tr>
<th>Scheme</th>
<th>Examination</th>
<th>Sample format</th>
<th>Distribution per year</th>
<th>Number of samples per distribution</th>
<th>Scoring</th>
</tr>
</thead>
</table>
| Haematological Malignancy Bone Marrow Aspirate
Assessment (Not Accredited)                  | Digital morphology | Web based programme    |                       | Web based digital image           | To be determined |

Should you wish to participate in the new programme or any of the existing ones, please contact *

UK NEQAS LI
Pegasus House
4th Floor Suite
463a Glossop Road
Sheffield, S10 2QD
United Kingdom

Tel: +44 (0) 114 2673600
Fax: +44 (0) 114 2673601
email: admin@ukneqasl.li.co.uk
www.ukneqasl.li.co.uk

* Please note that if your participation with UK NEQAS LI is via a distributor then you should contact your distributor for details of how to register.
4.8.2 a) The proficiency testing provider for this programme is:
UK NEQAS for Leucocyte Immunophenotyping
Pegasus House, 4th Floor Suite
463A Glossop Road
Sheffield, S10 2QD
United Kingdom
Tel: +44 (0) 114 267 3600, Fax: +44 (0) 114 267 3601
e-mail: nicola.rose@ukneqasli.co.uk

4.8.2 b) The coordinators of UK NEQAS LI programmes are Mr Liam Whitby (Director) and Mr Stuart Scott (Centre Manager).

4.8.2 c) Person(s) authorizing this report:
Mr Liam Whitby (Director) or Mr Stuart Scott (Centre Manager) of UK NEQAS LI.

4.8.2 d) No activities in relation to this EQA exercise were subcontracted.

4.8.2 g) The UK NEQAS LI Confidentiality Policy can be found in the Quality Manual which is available by contacting the UK NEQAS LI office. Participant details, their results and their performance data remain confidential unless revealed to the relevant NQAAP when a UK participant is identified as having performance issues.

4.8.2 i) All EQA samples are prepared in accordance with strict Standard Operational Procedures by trained personnel proved to ensure homogeneity and stability. Where appropriate/possible EQA samples are tested prior to issue. Where the sample(s) issued is stabilised blood or platelets, pre- and post-stability testing will have proved sample suitability prior to issue.

4.8.2 l), n), o), r) & s) Please refer to the UK NEQAS LI website at www.ukneqasli.co.uk for detailed information on each programme including the scoring systems applied to assess performance (for BS EN ISO/IEC 17043:2010 accredited programmes only). Where a scoring system refers to the ‘consensus result’ this means the result reported by the majority of participants for that trial issue. Advice on the interpretation of statistical analyses and the criteria on which performance is measured is also given. Please note that where different methods/procedures are used by different groups of participants these may be displayed within your report, but the same scoring system is applied to all participants irrespective of method/procedure used.

4.8.2 m) We do not assign values against reference materials or calibrants.

4.8.2 q) Details of the programme designs as authorized by The Steering Committee and Specialist Advisory Group can be found on our website at www.ukneqasli.co.uk. The proposed trial issue schedule for each programme is also available.

4.8.2 t) If you would like to discuss the outcomes of this trial issue, please contact UK NEQAS LI using the contact details provided. Alternatively, if you are unhappy with your performance classification for this trial, please find the appeals procedure at www.ukneqasli.co.uk/contact-us/appeals-and-complaints/