UK NEQAS
Leucocyte Immunophenotyping

Sheffield Teaching Hospitals NHS
NHS Foundation Trust

BCR::*ABL1* Kinase Domain Variant (Mutation) Status (Accredited)

Distribution – 212203

Participant –

Date Issued – 22 Feb 2022

Closing Date – 01 Apr 2022

Trial Comments

Three vials of lyophilised cell line material (scored samples KDV(M) 144 and 145, plus an optional educational sample KDV(M) Edu A) were distributed to 80 participants for *BCR*::*ABL1* kinase domain variant (mutation) (KDV(M)) analysis. Overall, 75 (93.8%) participants returned results for this trial (scored samples). Five laboratories have not submitted results at the time of trial reporting. Three pre-notified us of their non return and one laboratory was granted an extension to the submission deadline due to the ongoing COVID 19 pandemic.

Sample Comments

In order to best mimic clinical material, samples were formulated from a mixture of cell lines. Samples KDV(M) 144, KDV(M) 145 and KDV(M) Edu A were all composed of a cell line lacking the *BCR*::*ABL1* p210 (major) transcript, a *BCR*::*ABL1* p210 positive cell line expressing 'wildtype' (non-point mutated) *BCR*::*ABL1* fusion transcript and a *BCR*::*ABL1* p210 positive cell line expressing a specific kinase domain variant (point mutation) of the *BCR*::*ABL1* fusion transcript. Sample KDV(M) Edu A was formulated to feature a lower expressed variant allele frequency (VAF).

We would like to acknowledge Dr. Paul La Rosée (University of Jena), who kindly donated the material for this programme. We are also grateful to Novartis who initially supported this pilot EQA scheme through an educational grant and Prof. Johan den Dunnen (Leiden University and Human Genome Variation Society) for previous guidance regarding nomenclature. Thank you to Dr. Aytug Kizilors (King's College Hospital, London) for assistance with the pre-issue testing of the educational sample.

Uncontrolled copy

Sample KDV(M) 144

Your Results

	DNA sequence change(s)	Protein sequence change(s)	*ABL1* Reference sequence	Performance status for this sample
Your result				
Consensus result	**c.730A>G**	**p.Met244Val**	**NM_005157.5***	

Performance status feedback comments (as applicable):

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

Sample KDV(M) 145

Your Results

	DNA sequence change(s)	Protein sequence change(s)	*ABL1* Reference sequence	Performance status for this sample
Your result				
Consensus result	**c.1075T>G**	**p.Phe359Val**	**NM_005157.5***	

Performance status feedback comments (as applicable):

* NM_005157.6 reference sequence is available (https://www.ncbi.nlm.nih.gov/nuccore/NM_005157). The Locus Reference Genomic (LRG) sequence for *ABL1* is now public and can be formally employed (http://ftp.ebi.ac.uk/pub/databases/lrgex/LRG_769.xml).

IMPORTANT: Scoring criteria have been informed by Human Genome Variation Society (HGVS) sequence variant nomenclature recommendations version 19.01 and subsequently version 20.05[1,2]. Please refer to our website for further details regarding the performance scoring system.

Uncontrolled copy

Your Performance

Sample Score(s)		Performance Status Classification (over a six sample period)
Sample KDV(M) 144	**Sample KDV(M) 145**	**Any laboratory assigned unsatisfactory performance status for this trial will be sent an alert email to the default participant contact with details of the classification. Additionally, please refer to the notifications panel in your Participant Hub area of the UK NEQAS LI website for further information.** UK NEQAS LI aim to notify relevant laboratories of persistent unsatisfactory and unsatisfactory performance within 20 working days following the issue of the trial report.

N/A: Not applicable

Sample KDV(M) Edu A – Educational NOT SCORED

Your Results

	DNA sequence change(s)	Protein sequence change(s)	*ABL1* Reference sequence
Your result			
Consensus result	**c.944C>T**	**p.Thr315Ile**	**NM_005157.5***

* NM_005157.6 reference sequence is available (https://www.ncbi.nlm.nih.gov/nuccore/NM_005157). The Locus Reference Genomic (LRG) sequence for *ABL1* is now public and can be formally employed (http://ftp.ebi.ac.uk/pub/databases/lrgex/LRG_769.xml).

Uncontrolled copy

All Participant Results

Detailed Results Breakdown

Nomenclature provided by participants in relation to an *ABL1* NM_005157 based reference sequence unless otherwise stated. Percentage values quoted have been subjected to rounding up/down to 1 decimal place. Descriptions fully compliant with the current Human Genome Variation Society (HGVS) nomenclature recommendations are highlighted green.

Sample KDV(M) 144

Sequence change	Returns	Percentage of returns
DNA sequence change (cDNA)		
c.730A>G [a]	71	94.7
c.730>G [b]	1	1.3
No variant detected	3	4.0
Amino Acid change (protein)		
p.Met244Val[a]	62	82.7
p.M244V [c]	5	6.7
p.(Met244Val) [d]	3	4.0
(p.Met244Val) [d,e]	1	1.3
Met244Val [f]	1	1.3
No variant detected	3	4.0

Sample KDV(M) 145

Sequence change	Returns	Percentage of returns
DNA sequence change (cDNA)		
c.1075T>G [a]	69	92.0
c.944C>T [g,h]	1	1.3
c.1076T>G [g]	1	1.3
No variant detected	4	5.3
Amino Acid change (protein)		
p.Phe359Val [a]	58	77.3
p.F359V [c]	5	6.7
p.(Phe359Val) [d]	2	2.7
(p.Phe359Val) [d,e]	1	1.3
Phe359Val [f]	1	1.3
p.Phe317Leu [i,j]	1	1.3
p.Thr315Ile [g,h]	1	1.3
p.Phe359Ile [i]	1	1.3
p.Phe359Cys [g]	1	1.3
No variant detected	4	5.3

Sample KDV(M) Edu A – Educational NOT SCORED

Sequence change	Returns	Percentage of returns
DNA sequence change (cDNA)		
c.944C>T [a]	64	85.3
c.1075T>G [g,h]	1	1.3
c.764A>T [g]	1	1.3
No variant detected	5	6.7
Not tested	4	5.3
Amino Acid change (protein)		
p.Thr315Ile [a]	55	73.3
p.T315I [c]	6	8.0
p.(Thr315Ile) [d]	1	1.3
(p.Thr315Ile) [d,e]	1	1.3
Thr315Ile [f]	1	1.3
p.Phe359Val [g,h]	1	1.3
p.(Glu255Val) [g]	1	1.3
No variant detected	5	6.7
Not tested	4	5.3

Uncontrolled copy

[a] Nomenclature fully compliant with current HGVS recommendations (RNA/cDNA assay input material).
[b] Erroneous/absent nucleotide at the DNA level.
[c] Three letter amino acid code preferable to single letter.
[d] Parentheses (brackets) used erroneously for this nomenclature description, RNA (or cDNA produced from extracted RNA) was stated as the assay input material.
[e] Symbol syntax error.
[f] Absent prefix symbol.
[g] Out of consensus variant (accompanying protein or DNA description was also out of consensus).
[h] Suspected sample transposition event.
[i] Erroneous amino acid at the protein level (accompanying DNA description was in consensus).
[j] Erroneous amino acid position (accompanying DNA description was in consensus).

Method Breakdown

Please note, figures in the tables below may not tally with the total number of participants returning results due to some participants not returning all data requested or using multiple techniques.

Uncontrolled copy

Input (template) material for the detection/sequencing assay

	Returns
cDNA	73
RNA [a]	2

[a] Next generation sequencing (NGS) user (n=1), plus a suspected typographical entry by a Sanger sequencing user.

PCR method approach

	Returns
Nested PCR	37
Semi-nested PCR	24
Single PCR	13
Single long range PCR	1
Other	2

Target(s) of PCR amplification strategy

	Returns
BCR::*ABL1* fusion (targeting *ABL1* on the translocated chromosome 9 only)	67
Both *BCR*::*ABL1* fusion and *ABL1* on the non-translocated chromosome 9	8

Please note that for *BCR*::*ABL1* positive chronic myeloid leukaemia (CML)/acute lymphoblastic leukaemia (ALL) cases, clinically relevant *ABL1* kinase domain variants (point mutations), potentially conferring tyrosine kinase inhibitor (TKI) resistance, are present on the disease associated *BCR*::*ABL1* fusion allele. Therefore, *ABL1* on the translocated chromosome 9 is conventionally the amplification target for assays involving subsequent sequencing.

Analysis method

	Returns
Sanger Sequencing	59
Illumina MiSeq (NGS)	9
Illumina NextSeq (NGS)	4
Ion Torrent S5 (NGS)	2
Illumina MiniSeq (NGS)	2
Allele Specific PCR	2
Illumina Novaseq (NGS)	1
PACBIO Sequel (NGS)	1

NGS = Next generation sequencing.

Sequencing approach

	Returns
Bidirectional (forward AND reverse)	73
Unidirectional (forward OR reverse)	1
Not applicable	1

Reference sequence

	Returns
NM_005157.5	33
NM_005157.6	31
NM_005157.4	2
NM_005157.3	2
ENST00000318560.6	2
ENST00000318560.5	3
NM_005157 – no version provided	1
LRG_769	1

Uncontrolled copy

Assay journal references (as reported by participants)

	Returns
In house method (no reference)	24
Branford S. and Hughes T. (2006). Myeloid Leukemia Methods and Protocols eISBN 1-59745-017-0, 93-106*	16
Hochhaus A. *et al.* (2002). Leukemia 16:11, 2190-2196	9
Ernst T. *et al.* (2008). Haematologica 93:2, 186-192	6
Alikian M. *et al.* (2012). American Journal of Hematology 87:3, 298-304	6
Soverini S. *et al.* (2013). Blood 122:9, 1634-1648	6
Branford S. *et al.* (2002). Blood 99:9, 3472-3475	5
MODHEM (Network for Molecular Diagnostics of Hematologic Malignancies)	3
Polakova K. *et al.* (2015). J Cancer Res Clin Oncol. 141(5):887-99	2
Polakova K. *et al.* (2008). Leuk Res. 32:8, 1236-1243	2

As stated by >1 participant.

* Branford S. and Hughes T. (2006) Detection of BCR-ABL Mutations and Resistance to Imatinib Mesylate. In: Iland H., Hertzberg M., Marlton P. (eds) Myeloid Leukemia. Methods In Molecular Medicine, vol 125. Humana Press. (ISBN 978-1-58829-485-2).

Uncontrolled copy

Primary assay additional information

	Returns			
	Sanger Sequencing		Next Generation Sequencing (NGS)	
Percentage (%) variant	**Limit of Detection (LoD)**	**Limit of Quantitation (LoQ)**	**Limit of Detection (LoD)**	**Limit of Quantitation (LoQ)**
<1	-	-	3	1
1	1	-	6*	2
3	-	-	2	6
4	-	-	1	-
5	2	1	3	2*
7	1	-	-	-
10	10	3	1	3
15	8	2	-	-
20	36	12	-	-
30	1	-	-	-

Note: 2 of the LoQ values provided by participants have been excluded from the table as they were lower than the stated LoD; suspected typographical errors.

* Includes 1 participant supplementing their NGS approach with Allele Specific PCR.

Quantification – For educational purposes only

Figures in the tables below have been compiled from quantification data submitted by participants with a defined calculation approach. Descriptive statistics are included for those returns stating a PCR strategy (cDNA/RNA template input material) which targets the *BCR*::*ABL1* fusion (*ABL1* on the translocated chromosome 9) prior to analysis with >5 quantification data points available. Values quoted have been subjected to rounding up/down to 1 decimal place. We acknowledge the limitations of this small dataset.

Sample KDV(M) 144 variant (mutation) quantification: % variant allele frequency (VAF)

***ABL1* c.730A>G p.Met244Val**				
Calculation approach	**n**	**Mean**	**Median**	**Range**
(Mut/(Mut+WT)) x 100	21	66.7	72.0	20 - 100
Other	8			

Sample KDV(M) 145 variant (mutation) quantification: % variant allele frequency (VAF)

***ABL1* c.1075T>G p.Phe359Val**				
Calculation approach	**n**	**Mean**	**Median**	**Range**
(Mut/(Mut+WT)) x 100	22	35.6	32.9	20 - 55
Other	8			

Sample KDV(M) Edu A variant (mutation) quantification: % variant allele frequency (VAF)

***ABL1* c.944C>T p.Thr315Ile**				
Calculation approach	**n**	**Mean**	**Median**	**Range**
(Mut/(Mut+WT)) x 100	22	24.8	22.8	15 - 50
Other	9			

Mut = mutation (variant), WT = 'wildtype'. Assay method with *BCR*::*ABL1* enrichment.

Uncontrolled copy

Comments

In line with sample formulation, 72/75 (96.0%) returning participants detected a variant identifiable as p.Met244Val in sample KDV(M) 144.

- Overall 71/72 (98.6%) participants identifying the p.Met244Val variant described it as c.730A>G at the DNA level. One centre submitted nomenclature without the 'wildtype' nucleotide (c.730>G). All laboratories stated the use of an *ABL1* isoform a transcript reference sequence. **We strongly encourage laboratories to always provide a version number with the accession reference to unequivocally identify the reference sequence.**
- There were no gross nomenclature errors at the protein level. However, five of the participants identifying a variant in this sample used the less preferable single letter amino

acid code for protein descriptions. Four laboratories inappropriately included parentheses in the protein nomenclature provided; cDNA/RNA was stated as the assay input material and therefore, the protein level description is not predicted. One participant omitted to include the required p. designation prefix in their protein level description (Met244Val).

- Three laboratories returned a false negative result for sample KDV(M) 144. All utilised bi-directional Sanger sequencing from a semi-nested PCR (cDNA input material) targeting the *BCR*::*ABL1* fusion. The c.730A>G p.Met244Val consensus variant occurred within the stated region of interest of their assays. The median expressed variant load calculated from the trial data for the c.730A>G p.Met244Val variant (VAF=72.0%) was considerably above the assay limit of detection (LoD) quoted by all three participants.
- Four laboratories reported a low-level additional variant c.844G>A p.Glu282Lys (median VAF = 5.3%) in sample KDV(M) 144. This additional variant has previously been noted by UK NEQAS LI as reoccurring in trial samples which incorporate the cell line harbouring c.730A>G p.Met244Val utilised for this EQA programme. The c.844G>A p. Glu282Lys variant is located within a conformational 'switch control' residue[3] but is not currently referred to in available frequently referenced tyrosine kinase inhibitor (TKI) treatment guidelines for chronic myeloid leukaemia (CML)[4-6]. Nevertheless, it is noted as a TKI resistant variant (E282K mutation) by Soverini *et al.* (2020) in their prospective assessment of NGS detectable variants in CML patients (NEXT-in-CML study)[7]. The low-level additional variant c.844G>A p. Glu282Lys was not subject to scoring for this trial.

In line with sample formulation, 69/75 (92.0%) of returning participants detected a variant identifiable as p.Phe359Val in sample KDV(M) 145.

- Overall, 71/75 (94.8%) centres detected a variant in this sample. All participants identifying the consensus variant described it as c.1075T>G the cDNA level. However, two participants provided erroneous accompanying protein nomenclature (p.Phe317Leu, p.Phe359Ile).
- Additionally one laboratory provided out of consensus nomenclature at the DNA level (c.1076T>G) which was accompanied by the protein description (p.Phe359Cys). A further centre is suspected of performing a sample transposition event with educational sample KDV(M) Edu A.
- Similar to the outcomes for sample KDV(M) 144, five participants identifying the consensus variant in sample KDV(M) 145 used the less preferable single letter amino acid code for protein descriptions. Three laboratories inappropriately included parentheses in the protein nomenclature provided; cDNA/RNA was stated as the assay input material and therefore, the protein level description is not predicted. Again, one participant omitted to include the required p. designation prefix in their protein level description (Phe359Val).
- Due to the clinical actionability of particular *BCR*::*ABL1* kinase domain variants, erroneous protein descriptions have the potential to adversely impact patient management. Of relevance to this trial, the second generation TKI treatment recommendations in the clinical context of CML for p.Phe359Val (consensus variant) compared to that of the erroneously reported p.Phe317Leu variant are different in regard to the unsuitability of nilotinib and dasatinib[4-6].
- Four centres failed to detect any variant in sample KDV(M) 145; including one laboratory also submitting a false negative result for sample KDV(M) 144. Overall, three participants utilised bi-directional Sanger sequencing and one an Ion Torrent NGS approach. PCR methodology was variable (cDNA input material) but all stated targeting of the *BCR*::*ABL1*

Uncontrolled copy

fusion. The c.1075T>G p.Phe359Val consensus variant occurred within the stated region of interest of their assays. The median expressed variant load calculated from the trial data for the c.1075T>G p.Phe359Val variant (VAF=32.9%) was above the assay limit of detection (LoD) quoted by all four participants.

Quantification

Overall, 42.7% (32/75) of returning laboratories provided quantitative information for at least one of the samples issued for this trial (KDV(M) 144, 445 and/or Edu A). Please refer to the tables on page 8, which summarises equivalent quantification results submitted by participants.

Educational sample: KDV(M) Edu A

Thank you to the 71/75 (94.7%) of returning participants who tested the optional additional educational sample included in this trial distribution. **Please note, educational sample KDV(M) Edu A is not subject to scoring and performance monitoring.**

- Given the adoption of NGS (affording greater sensitivity to permit the early detection of clinically relevant *BCR*::*ABL1* TKI resistant variants[4-10]) by an increasing number of participants, sample KDV(M) Edu A was formulated to harbour a lower expressed variant load. The aim of the sample was to provide an educational exercise with relevance to NGS users and challenge the LoD for Sanger sequencing based assays.
- Overall, 64/71 (90.1%) of participants opting to analyse the sample detected a variant identifiable as p.Thr315Ile in sample KDV(M) Edu A. Two laboratories returned out of consensus variants for this sample, one of which was due to a suspected sample transposition event with sample KDV(M) 145. Please refer to the table on page 4 for a full breakdown of results for sample KDV(M) Edu A.
- Consistent with pre-issue testing outcomes, the trial median expressed VAF for c.944C>T p.Thr315Ile was 22.8% (interquartile range 9.1%, n=22).
- Five participants failed to detect any variant in sample KDV(M) Edu A. All used a Sanger sequencing approach with a nested (n=3) or semi-nested (n=2) PCR method to target *ABL1* on the translocated chromosome 9 (*BCR*::*ABL1* fusion). Input material was stated as cDNA (one centre submitted 'RNA' but is suspected to have made a typographical error based on the other methodological information provided). Of the laboratories returning a false negative result for sample KDV(M) Edu A, all stated a LoD below that of the median expressed %VAF for the c.944C>T p.Thr315Ile variant (based on information available, LoD range 10-20% VAF, n=4).
- Of note, the c.944C>T p.Thr315Ile median VAF obtained by Sanger sequencing was higher than that derived from NGS approaches; 26.8% (n=10) and 19.0% (n=12), respectively. A single laboratory utilised digital PCR and submitted a VAF of 16.3% for the consensus variant.

Final Remarks

To implement formal sample scoring classification and performance monitoring, a core list of clinically actionable *BCR*::*ABL1* kinase domain variants (point mutations) has been produced (informed by review of the literature and currently available EQA material). The core list of residue (amino acid)

Uncontrolled copy

positions encompasses: p.Met244, p.Gly250, p.Gln252, p.Tyr253, p.Glu255, p.Val299, p.Thr315, p.Phe317, p.Met351 and p.Phe359 (tyrosine-protein kinase ABL1 isoform a, NP_005148.2). During the sample scoring classification process, reference is made to a participant's stated assay scope (as provided at trial results submission). To mitigate the reporting of additional variants (i.e. those of unknown clinical significance and/or cell line artefacts), participants are reminded at trial data entry to return only results with clinical significance.

For the new financial year (2022/23), samples for this programme will continue to be formulated with the aim of suitability for analysis by several commonly employed techniques, including Sanger sequencing (variant(s) representing >20% VAF). UK NEQAS LI will continue to aim to offer periodic additional low-level variant(s) sample (5 - 20% target VAF) as an educational exercise. Variants <5% VAF are beyond the scope of the current scheme.

The cell lines utilised in this programme have been pre-validated by The European Treatment Outcome Study (EUTOS) group. However, it is important to note they are cell lines stably transfected with cDNA constructs and therefore do not exactly reflect the genetic context of clinical samples. At this time no alternative suitable material exists for the purposes of *BCR*::*ABL1* kinase domain variant (mutation) testing EQA.

Repeat samples are available for all programmes. In the event that your local quality control criteria are not met please contact us. Please do not submit results based on a suboptimal nucleic acid extraction.

PLEASE NOTE: Following the recent publication of the HGNC consensus statement regarding fusion gene nomenclature (Bruford *et al.* 2021)[11], UK NEQAS LI are working to implement use of the double colon (::) for the description of fusion genes. Historical nomenclature may persist in some areas of our website and trial documentation for an interim period due to IT constraints.

Uncontrolled copy

Reference(s)

1. Den Dunnen, J. T. *et al.* HGVS Recommendations for the description of sequence variants: Update *Hum. Mutat.* 37, 564–569 (2016).
2. http://varnomen.hgvs.org/ (Version 20.05).
3. Chan W. *et al.* Conformational control inhibition of the BCR-ABL1 tyrosine kinase, including the gatekeeper T315I mutant, by the switch-control inhibitor DCC-2036. *Cancer Cell* 19(4):556-68 (2011).
4. Hochhaus, A. *et al.* (2020) European LeukemiaNet 2020 recommendations for treating chronic myeloid leukemia. *Leukemia* 34(4):966-984.
5. Smith, G. *et al.* A British Society for Haematology Guideline on the diagnosis and management of chronic myeloid leukaemia. *Br J Haematol.* (2020) 191(2):171-193 5.
6. NCCN Chronic Myeloid Leukemia (Version 3.2022).
7. Soverini, S. *et al.* Prospective assessment of NGS-detectable mutations in CML patients with nonoptimal response: the NEXT-in-CML study. *Blood* 135(8):534-541 (2020).
8. Soverini, S. *et al*. (2020) Molecular testing in CML between old and new methods: Are we at a turning point? *J. Clin. Med.* 27;9(12):3865.
9. Soverini, S. *et al.* (2019) Next-generation sequencing for BCR-ABL1 kinase domain mutation testing in patients with chronic myeloid leukemia: a position paper. *J. Hematol. Oncol.* 12(1):131.
10. Kizilors, A. *et al*. (2019) Effect of low-level BCR-ABL1 kinase domain mutations identified by next-generation sequencing in patients with chronic myeloid leukaemia: a population-based study. *Lancet Haematol*. 6:276-284.
11. Bruford, E, *et al.* (2021) HUGO Gene Nomenclature Committee (HGNC) recommendations for the designation of gene fusions. *Leukemia* 35(11):3040-3043.

Information with respect to compliance with standards BS EN ISO/IEC 17043:2010

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
e-mail: amanda.newbould@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) Pre issue testing of samples for this programme is subcontracted, although the final decision about sample suitability lies with the EQA provider; no other activities in relation to this EQA exercise were subcontracted. Where subcontracting occurs, it is placed with a competent subcontractor and the EQA provider is responsible for this work.

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 proven 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/

4.8.4) The UK NEQAS LI Policy for the Use of Reports by Individuals and Organisations states that all EQA reports are subject to copyright, and, as such, permission must be sought from UK NEQAS LI for the use of any data and/or reports in any media prior to use. See associated policy on the UK NEQAS LI website: http://www.ukneqasli.co.uk/eqa-pt-programmes/new-participant-information/

Uncontrolled copy