UK NEQAS
Leucocyte Immunophenotyping

Sheffield Teaching Hospitals NHS
NHS Foundation Trust

Pilot Lymphoid Gene Panels (Not Accredited)

Distribution - Lymphoid GP 232401 Participant

Date Issued – 09 November 2023 Closing Date – 05 January 2024

Trial comments

This trial was issued to 46 participants, of which 39 (84.8%) returned results. Two participants informed us of their intended non return of results.

We encourage laboratories to test all samples issued as part of the Lymphoid Gene Panels programme, even if the referral reason is suggestive of a lymphoid neoplasm that would not routinely be tested within the laboratory repertoire. Whilst a referral reason may provide information on the potential lymphoid neoplasm, testing of all EQA sample distributions enables assessment of laboratory Next Generation Sequencing (NGS) panels. There are likely to be samples issued where variants in genes overlap with multiple lymphoid neoplasms, providing insight into the performance of laboratory NGS panels. Furthermore, this programme remains in pilot phase and is still developing and as such, is not currently performance monitored.

Sample comments

One lyophilised sample (Lymphoid GP 107) was prepared and distributed by UK NEQAS LI. Sample Lymphoid GP 107 was manufactured using cell line material. A clinical scenario accompanied this sample with genetic testing requested for a query lymphoid/precursor lymphoid neoplasm.

Sample Lymphoid GP 107

Did you detect a reportable DNA sequence change in Sample Lymphoid GP 107:

Your variant results

Gene	Your DNA sequence variant detected	Your protein variant	Your variant classification

Please note, all submitted variant(s) of unknown clinical significance may not be reflected in the above table for individual participants due to formatting and space constraints.

All participant results

Please note, in the interests of clarity we will only summarise variants reported by >10 participants.

Gene	n#	Variant classification^			Variant detected (consensus)*		Median VAF (%) (IQR)+
		Strong clinical significance	Potential clinical significance	Unknown clinical significance	DNA sequence description	Protein level description	
TP53	39/39	34	3	2	c.586C>T	p.(Arg196*)	48.0 (28.8)
FBXW7	27/29	19	7	1	c.1513C>T	p.(Arg505Cys)	60.0 (6.6)
TP53	24/39	17	6	0	c.375G>A	p.(Thr125=)	48.0 (49.5)
CREBBP	21/24	8	12	1	c.4074del	p.(Phe1358Leufs*18)	48.0 (49.8)
ARID1A	15/21	5	9	1	c.4555del	p.(Gln1519Argfs*8)	24.0 (15.0)
PTEN	13/20	11	3	0	c.699_700delinsGGCCCATGG	p.(Arg234Alafs*11)	45.2 (40.1)
PTEN	13/20	9	4	0	c.737_738insCTGAAGTTCATGTACTTTGAGTTCCCTCAGCCCTGGGTT	p.(Leu247*)	53.0 (21.0)
IKZF1	13/16	3	9	1	c.484C>T	p.(Arg162Trp)	24.8 (2.0)
KMT2D	12/16	7	4	1	c.9265del	p.(Val3089Trpfs*30)	49.0 (21.5)
KMT2D	11/16	6	5	0	c.5549del	p.(Gly1850Alafs*2)	21.1 (4.0)

#Total number of participants reporting this variant/number of participants stating the inclusion of the relevant gene on their panel.
^ Based on Li *et al* (2017) Joint consensus recommendations from the Association for Molecular Pathology, American Society of Clinical Oncology and College of American Pathologists.[1] Variant classification by participants utilising alternative systems may have been aligned (where possible) to the equivalent Li *et al* category (if available/applicable). Variant classification breakdowns are not equal to the sum of the total number of participants reporting the variant in any given gene as some participants provided information that could not be aligned to the equivalent Li *et al*. categories and some did not provide variant classification information.
* Results returned by participants, at both the DNA and protein level, may have been harmonised to the equivalent Human Genome Variation Society (HGVS) approved nomenclature (http://varnomen.hgvs.org/) during the compilation of 'All Participants' results table. Protein nomenclature includes parenthesis as it represents a prediction from analysis at the DNA level. Please contact UKNEQAS LI for reference sequence information.
+ Descriptive statistics calculated for any variant with >10 quantification data points. Percentage values quoted have been subjected to rounding up/down to 1 dp. IQR = Interquartile range.

Your performance

Performance	Performance Status for this sample	Performance Status Classification Over 12 Month Period	
		Satisfactory	Critical

Please note: this programme is not currently performance monitored. We will work towards a performance monitoring system as the programme develops.

Methods

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.

NGS platform used

	Returns
Illumina MiSeq	13
Illumina NextSeq	9
Illumina NovaSeq (no further information provided)	6
ThermoFisher Scientific (Life Tech) Ion S5	4
Illumina MiniSeq	2
Illumina NovaSeq 6000	2
Thermo Fisher Scientific (Life Tech) Genexus	2
ThermoFisher Scientific (Life Tech) Ion S5 XL	1

NGS panel description

	Returns
Custom commercially developed	22
Qiagen QIAseq Human Myeloid Neoplasms Panel	2
Illumina Trusight Myeloid Sequencing Panel	2
Illumina AmpliSeq™ Myeloid Panel	1
Roche RMH HaemOnc Panel	1
ThermoFisher Scientific Lymphoma Core DNA Panel	1
SOPHiA DDM™ Community CLL Clonality Solution (Chronic Lymphocytic Leukemia v3) Panel	1
In-house Panel	1
Other	5

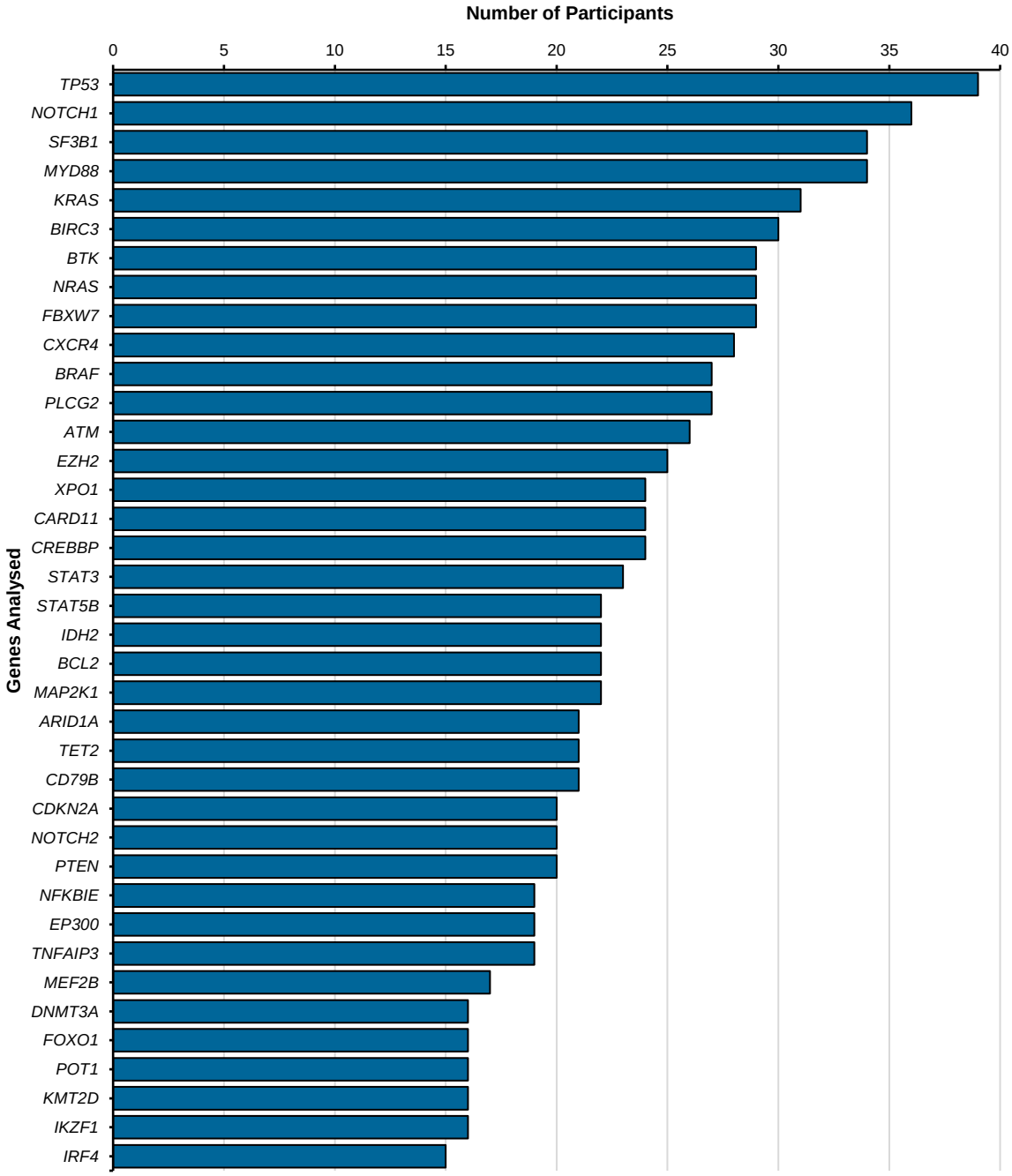

Figure 1: Histogram depicting genes routinely analysed by participants. Only genes routinely analysed by ≥15 participants are recorded in the histogram.

Annotation database resources

	Returns
COSMIC (Catalogue Of Somatic Mutations In Cancer)	39
ClinVar (NCBI)	36
The TP53 Database hosted by NCI (previously IARC TP53 database)	30
dbSNP (Short Genetic Variations, NCBI)	21
The Genome Aggregation Database (gnomAD)	19
VarSome	18
OncoKB	17
The Clinical Knowledgebase (CKB) Jackson Laboratory	11
Seshat TP53 database	11
My Cancer Genome (Vanderbilt-Ingram Cancer Center)	8
Franklin by Genoox	6
The Cancer Genome Atlas (TCGA)	5
HGMD (The Human Gene Mutation Database)	5
cBioPortal	5
OMIM (NCBI)	4
GENIE	2

As stated by ≥2 participants.

Large-scale sequencing project dataset(s) routinely consulted during variant interpretation

	Returns
The Genome Aggregation Database (gnomAD)	32
The Exome Aggregation Consortium (ExAC)	13
1000 Genomes	12
NHLBI-GO Exome Sequencing Project (ESP)	6

As stated by ≥2 participants.

Published guideline(s) and/or recommendation(s) referenced to inform classification of somatic variant clinical significance/pathogenicity (in a Haemato-Oncology context)

	Returns
Li, M.M. *et al.* Standards and Guidelines for the Interpretation and Reporting of Sequence Variants in Cancer. *J Mol Diagn.* 19(1):4-23 (2017).	29
Froyen, G. *et al.* Standardization of Somatic Variant Classifications in Solid and Haematological Tumours by a Two-Level Approach of Biological and Clinical Classes: An Initiative of the Belgian ComPerMed Expert Panel. *Cancers (Basel)*. 11(12): 2030 (2019).	9
Horak, P. *et al*. Standards for classification of pathogenicity of somatic variants in cancer (oncogenicity): Joint recommendations of Clinical Genome Resource (ClinGen), Cancer Genomics Consortium (CGC), and Variant Interpretation for Cancer Consortium (VICC). *Genet Med*. 24(5):986-998 (2022).	9
Sukari, M.A. *et al.* A classification system for clinical relevance of somatic variants identified in molecular profiling of cancer. *Genet Med.* 18(2):128–136 (2016).	3
Koeppel, F. *et al*. Standardisation of pathogenicity classification for somatic alterations in solid tumours and haematological malignancies. *Eur J Cancer*. 159:1-15 (2021).	3

As stated by ≥2 participants.

Genome Assembly

	Returns
GRCh37/hg19	32
GRCh38	7

Minimum variant allele frequency (VAF) for reporting the identification of a single nucleotide variant

	Returns
5%	23
4%	1
3%	4
1-2%	11

Minimum variant allele frequency (VAF) for reporting the identification of an indel (deletion/duplication/insertion) variant

	Returns
5%	25
4%	1
3%	5
1-2%	8

Trial Comments

Methodology

- The vast majority of participants employed bridge amplified reversible dye terminator-based platforms from Illumina (n=32 data returns, 82.1% of participants).
- Five participants utilised a myeloid based panel in this trial distribution.
- Of the 39 laboratories providing information regarding genome assembly, 32 participants referenced GRCh37/hg19, with seven participants referenced the GRCh38/hg38 genome-based assembly. At the time of reporting, GRCh38.p14 (equivalent to the UCSC hg38) is the latest human genome release (7th October 2023) from NCBI Genome Data Viewer (https://www.ncbi.nlm.nih.gov/genome/gdv/).
- The minimum Variant Allele Frequency (VAF) quoted for reporting both single nucleotide and indel (insertion/duplication and deletion variants ranged from 1% - 5%, with a median of 5%.
- The median minimum acceptable coverage (read depth) for reporting a single nucleotide variant was 250x (range 10-1000x); for reporting an indel (insertion/duplication/deletion) variant the minimum acceptable coverage was 300x (range 10-1000x).
- All participants (n=39) provided information relating to the number of genes analysed on the NGS panel. A total of 205 different genes were present on participant NGS panels. The median number of genes tested on a given panel by laboratories for sample Lymphoid GP 107 was 38 (range 1-80).
- The most commonly analysed genes for this trial distribution based on the referral information were: *TP53* (39 participants, 100% returns), *NOTCH1* (36 participants, 92.3% participant returns), *SF3B1* (34 participants, 87.2% returns), *MYD88* (34 participants, 87.2% returns), *KRAS* (31 participants, 79.5% returns), and *BIRC3* (30 participants, 76.9% returns).

Sample Lymphoid GP 107

All participants (n=39) returning results for this trial indicated the detection of at least one DNA sequence variant in sample Lymphoid GP 107. Among all participant data returns, variants were reported in 23 genes. A summary of the most frequently reported variants (10 variants across seven genes) has been summarised in the 'All Participant results' table on page 2. From the set of 10 variants, in depth discussion will primarily focus on variants most frequently reported (>20 participants).

Classification of variants in this trial was largely in line with somatic variant classifications outlined in Li *et al.,* (2017) guidelines. One participant reported the clinical significance of the a TP53 variant as 'low' despite indicating the use of the Horak *et al*., (2022) guidelines, which classify variants into oncogenic, likely oncogenic, variant of uncertain significance, likely benign or benign[2]. For clarity, variant classifications in this dataset have been aligned to Li *et al.,* (2017) joint consensus recommendations from the Association for Molecular Pathology, American Society of Clinical Oncology and College of American Pathologists[1] (where possible). This classification system utilises a tier system from I-IV, ranging from variants of strong, potential, or unknown clinical significance and includes benign/likely benign variants. **Please note for the purposes of this EQA programme, we only require the reporting of variants of strong, potential, or unknown clinical significance. Variants considered benign or likely benign do not need to be reported.**

In total, 39/39 (100%) participants that analysed *TP53* reported the NM_000546.6(*TP53*):c.586C>T p.(Arg196*) variant in exon 6. Of the 39 participants reporting the variant, 34 (87.2%) participants classified the variant as having strong clinical significance and three participants (7.7%) classified the variant as of potential clinical significance. Two participants did not provide a variant classification.

- The median VAF reported for this variant was 48% with an interquartile range of 28.8% and a median read depth of 2,000x coverage.
- The variant has been reported in dbSNP (rs397516435)[3], however, this has only been reported three times in gnomAD in a global exome analysis[4]. These were all reported in a European (non-Finnish) population.
- This variant has been documented in the COSMIC database[5] (COSV52663748) on 530 occasions, linked to various cancer types, including 27 entries associated with lymphoid malignancies. Furthermore, the variant is present in ClinVar[6] (VCV000043589.45) in association with two different cancer types and hereditary cancer predisposition syndromes.
- This variant is listed in the TP53 Database[7] (previously IARC) (240 entries, 15 in association with lymphoid malignancies), Seshat[8] (869 entries) and is also present in the UMD database[9].
- For the predicted protein change associated with the *TP53* variant there were a small number of minor non compliances in relation to HGVS nomenclature descriptions[10]. Thirty-six (92.4%) participants reported the predicted protein change as either p.(Arg196Ter) or p.(Arg196*), in full compliance with HGVS recommendations. Two participants (5.1%) reported the predicted change as p.Arg196Ter, which is mostly compliant with recommendations, however, it should be noted that if DNA has been utilised as input material, parentheses are required as any protein change is only predicted based on the DNA variant detected[10]. One (2.6%) participant reported the predicted change as p.R196*, which is largely compliant, three letter amino acid code is preferred when describing protein changes and as above, parentheses are required when analysing DNA.

Twenty-four (61.5%) out of 39 participants analysing *TP53* reported the NM_000546.6(TP53):c.375G>A p.(Thr125=) variant in exon 4. An additional participant reported detection of a NM_000546.6(TP53):c.375G>A p.(Tyr125=) variant, providing out of consensus protein nomenclature. Of the 24 participants reporting the variant, 16 (66.7%) participants classified the variant as having strong clinical significance and six participants (25%) classified the variant as of potential clinical significance. One participant (4.2%) reported the clinical significance of the variant as 'low' despite indicating the use of the Horak *et al*., (2022) guidelines, which classify variants into oncogenic, likely oncogenic, variant of uncertain significance, likely benign or benign[2]. A further participant did not provide variant classification information. Of the fourteen participants that failed to detect (or did not report) the variant, 12 provided information indicating that the NGS panel utilised sequenced across this variant region, with two providing no information. Ten participants indicated that full coverage was achieved across this gene, with no internal quality control (QC) issues reported. A further four participants provided no information relating to internal QC.

- The median VAF reported for this variant was 48% with an interquartile range of 49.5% and a median read depth of 2,199x coverage.
- The variant has been reported in dbSNP (rs55863639)[3], however, this has been reported three times on gnomAD, (twice in global exome and once in global genome analysis)[4].
- This variant has been listed 49 times in the COSMIC database[5] (COSV52718605) in association with various cancer types, including seven listings in association with lymphoid neoplasms.
- This variant is listed in the TP53 Database[7] (previously IARC) (16 entries, two in association with lymphoid malignancies), Seshat[8] (77 entries) and is also present in the UMD database[9].
- This synonymous variant has been shown to result in aberrant splicing, with a shorter form of the TP53 protein observed *in vitro*[11]. The TP53 Network of the European Research Initiative on Chronic Lymphocytic Leukemia (ERIC) group indicate that if a synonymous variant is detected it is important to check the predicted effect on splicing. Synonymous variants affecting codon 125 (c.375G>A and c.375G>T) are classed as pathogenic[12].
- For the predicted protein change associated with the *TP53* variant there were a number of non-compliances in relation to HGVS nomenclature descriptions[10]. Fourteen (56%) participants reported the predicted protein change as either p.(Thr125=), in full compliance with HGVS recommendations. One participant (4%) reported the predicted change as p.Thr125=, which is mostly compliant with recommendations, however, it should be noted that if DNA has been utilised as input material, parentheses are required as any protein change is only predicted based on the DNA variant detected[10]. One participant (4%) reported a p.(Thr125=)), which uses the placement of parentheses incorrectly. One participant (4%) reported a p-(Thr125=), which uses an incorrect character with the protein letter prefix (p.). One participant reported the amino acid change as p.(Thr125Thr) and one p.(T125T). In these instances, the equals (=) symbol should be utilised to indicate that a sequence was tested but found to be unchanged, rather than using the same amino acid at the end of the description.
- One participant (4%) reported the protein nomenclature as p.= and two (8%) as p.(=), which is non complaint with HGVS recommendations. The use of the p.= description indicates that the entire protein coding region was analysed, and no variant was

identified that changes the protein sequence[10]. Both participants reported use of DNA as input material and not RNA. A further participant reported a p.? predicted amino acid change, which is out of consensus with the responses returned by all participants.

- It should be noted that description of the variant at the DNA level is required when providing predicted protein nomenclature, since, depending on the amino acid, there can be up to five different nucleotide substitutions that can leave an amino acid unchanged[10].

In total, 27/29 (93.1%) participants that analysed *FBXW7* reported the NM_001349798.2(FBXW7):c.1513C>T p.(Arg505Cys) variant in exon 12. Of the 27 participants reporting the variant, 19 (70.4%) participants classified the variant as having strong clinical significance, seven participants (25.9%) classified the variant as of potential clinical significance and one (3.7%) as of unknown clinical significance. The participants that failed to detect (or did not report) the variant sequenced across this variant region and did not report any internal quality control issues.

- The median VAF reported for this variant was 60% with an interquartile range of 6.6% and a median read depth of 2,000x coverage.
- This variant has been listed 206 times in the COSMIC database[5] (COSV55891274) in association with various cancer types, including fifty-seven listings in association with lymphoid neoplasms.
- The variant has been reported in dbSNP (rs149680468)[3], however, this has been reported once in a global exome analysis on gnomAD[4].
- Furthermore, the variant is present in ClinVar[6] (VCV000069961.2) in association with multiple cancer types.
- Twenty-four participants (88.9%) reporting the variant described the predicted amino acid change as p.(Arg505Cys), in compliance with HGVS recommendations. A further two participants (7.4%) reported the variant as p.Arg505Cys. Parentheses are required in this context as DNA has been analysed thus any protein change is only predicted based on the DNA variant detected[10]. The final participant reported the variant as p.Arg505Cys), with inconsistent utilisation of parentheses to describe a predicted amino acid change from DNA variant information.

In total, 21/24 (87.5%) participants routinely analysing *CREBBP* in the context of lymphoid neoplasms reported the NM_004380.3(CREBBP):c.4074del p.(Phe1358Leufs*18) variant in exon 24. Of the 21 participants reporting the variant, eight (38.1%) participants classified the variant as having strong clinical significance, 12 participants (57.1%) classified the variant as of potential clinical significance and one (4.8%) did not provide a variant classification. Two of the three participants that failed to detect (or did not report) the variant sequenced across this variant region, with one providing no sequence coverage information. The three participants did not provide information on internal QC or coverage across this variant region.

- The median VAF reported for this variant was 48% with an interquartile range of 49.8% and a median read depth of 1,910x coverage.
- This variant has been listed three times in the COSMIC database[5] (COSV52119051) in both cell lines and a lymphoid neoplasm.
- The variant is not present in been reported in dbSNP[3], gnomAD[4] or ClinVar[6].
- HGVS nomenclature for this *CREBBP* variant was largely in accordance with the recommendations for protein descriptions. Eleven out of 21 (52.4%) participants described the predicted amino acid change as p.(Phe1358LeufsTer18); with a further eight (38.1%) reporting p.(Phe1358Leufs*18). Both variant descriptions are fully compliant with HGVS recommendations. Two participants reported the amino acid change as p.Phe1358LeufsTer18. It should be noted that if DNA has been utilised as input material, parentheses are required as any protein change is only predicted based on the DNA variant detected[10].

For the remaining variants reported by more than 10 participants, there was a general observation in relation to the reporting of HGVS nomenclature, in particular, protein descriptions. When reporting predicted protein changes, HGVS recommendations indicate that when DNA is utilised as input material, parentheses are required as any protein change is only predicted based on the DNA variant detected.

There were three variants identified across three genes (*CREBBP*, *ARID1A* and *KMT2D*) that resulted in a nucleotide deletion event. When describing deletions, HGVS recommendations indicate that the deleted nucleotide sequence (whether single or multiple nucleotides) should not be included in the HGVS DNA description, since the deleted nucleotides can be deduced from the positional numbering and therefore including this sequence produces a longer description with redundant information[10].

In addition, when providing the reference sequence utilised during analysis, it is important to ensure that a sequence identifier must only identify one reference sequence[10]. HGVS recommendations state that version numbers are required to distinguish between sequences. Only reference sequences with version numbers are suitable for defining and describing a sequence variant within a given gene. Furthermore, to better standardise variant description and facilitate clinical reporting, the HGVS advocate use of the transcript reference sequence(s) specified by the MANE Select collaboration project[13].

Poorly curated variant nomenclature and use of incomplete or alternative reference sequence information impedes the ability of a laboratory to effectively search the relevant published data sets and literature during the variant classification process and thus, has the potential to impact a patient's diagnosis, prognostication and/or treatment. **We strongly encourage laboratories to verify the nomenclature generated by automated software systems/pipelines, as it may not fully comply with the current HGVS recommendations.**

We would like to thank participants for their continued engagement with the Lymphoid Gene Panels programme, particularly when considering the complexity of the data returns. We are looking to introduce changes to improve the ease of data returns by developing a laboratory record, reflecting a laboratory's current practice in relation to testing lymphoid samples. We ask participants to ensure that there is accurate curation of submitted data for trial Lymphoid GP 232402, as this will form the basis for the laboratory record for each participant.

Please note: The information provided herein is for participant information only. Clinical decision making with regards to variant interpretation, pathogenicity, actionability and predicted disease outcomes should not be based solely on comments provided by UK NEQAS LI in this EQA trial report. It is beyond the scope of this programme to comment conclusively on the clinical significance of the variants reported by participants. We acknowledge the limitations of this EQA exercise.

References

1. Li, M. M., Datto, M., Duncavage, E. J. *et al.* Standards and Guidelines for the Interpretation and Reporting of Sequence Variants in Cancer: A Joint Consensus Recommendation of the Association for Molecular Pathology, American Society of Clinical Oncology, and College of American Pathologists. *J. Mol. Diagn*. 2017;**19**(1): 4-23.

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6. Landrum, M. J., Lee, J. M., Benson, M. *et al.* ClinVar: improving access to variant interpretations and supporting evidence. *Nucleic Acids Res.* 2018;**46**(D1): D1062–D1067.

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9. Leroy, B., Anderson, M. & Soussi, T. TP53 Mutations in Human Cancer: Database Reassessment and Prospects for the Next Decade. *Hum. Mutat.* 2014;**35**(6): 672–688. https://p53.fr/tp53-database

10. Human Genome Variation Society (HGVS). Available at: https://hgvs-nomenclature.org/stable/ . (Accessed: 11 March 2024).

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12. Malcikova, J., Tausch, E., Rossi, D. *et al*. ERIC recommendations for TP53 mutation analysis in chronic lymphocytic leukemia—update on methodological approaches and results interpretation. *Leukemia*. 2018; 32: 1070–1080.

13. Morales, J., Pujar, S., Loveland, J. E. *et al*. A joint NCBI and EMBL-EBI transcript set for clinical genomics and research. *Nature*. 2022;604: 310–315.

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:
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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/