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Diagnostic Proficiency Testing

Survey 2014

Final Report

prepared by
Brian Fowler

1. Geographical distribution of participants

In 2014, 19 laboratories from 10 countries subscribed to the scheme. For both surveys 19 laboratories submitted results.

Country	Number of participants
Austria	1
Canada	3
China	1
Czech Republic	1
Estonia	1
Germany	3
Norway	1
Sweden	2
Switzerland	1
USA	5

2. Samples and Shipment

The samples contain a small amount of thiomersal and have been heat-treated. They were pre-analysed in our institute after 3 days incubation at ambient temperature (to mimic possible changes that might arise during transport). In all six samples the typical metabolic profiles were preserved after this process. The urine samples were distributed to participants on April 7th at ambient temperature by CSCQ using the courier DHL. Delivery times of samples reported by the courier ranged from 1 to 3 days. There were discrepancies with times reported by participants suggesting possible internal delays.

3. Tests

Analyses of amino acids, organic acids, mucopolysaccharides, oligosaccharides and purines/ pyrimidineswere required in 2014.

4. Schedule of the scheme in 2014

Sample distribution	April 07, 2014
Start of analysis of Survey 2014/1	April 28, 2014
Survey 2014/1 - Results submission	May 19, 2014
Survey 2014/1 - Reports	July 08, 2014
Start of analysis of Survey 2014/2	June 09, 2014
Survey 2014/2 – Results submission	June 30, 2014
Survey 2014/2 - Reports	August 11, 2014
Annual meeting of participants	SSIEM, Innsbruck, September 2, 2014
Annual Report 2014	April 2015

This year we were able to use the evaluation programme to generate individual lab reports and these were distributed in good time on July 8th and August 11th. Feedback on the content and style of these reports is invited.

5. Receipt of samples and results

Receipt of samples (sent on April 07, 2014)

Receipt (days after shipment)	Delivery reported by participants	Delivery reported by DHL
1	5	10
2	3	8
3	3	1
4	2	
21-51	5	
15th March!!	1	

Date of reporting of results

19 of 19 labs returned results for both surveys, mainly by the deadline.

6. Scoring system

Two criteria are evaluated: analytical performance, interpretative proficiency including recommendations for further investigations. Due to the large variability in reporting results in various countries, recommendations pertaining to treatment are not evaluated in proficiency testing. However, they are still reported and summarised by the scheme organisers.

A	Analytical performance	Correct results of the appropriate tests	2	max 2
		Partially correct or non-standard methods	1	
		Unsatisfactory or misleading	0	
I	Interpretative proficiency&Recommendations	Good (diagnosis was established)	2	max 2
		Helpful but incomplete	1	
		Misleading or wrong diagnosis	0	

The **total score** is calculated as a sum of these two criteria. The maximum to be achieved is 4 points per sample. The scores were calculated only for laboratories submitting results.

7. Results of samples and evaluation of reporting

Sample A: **Hypoxanthine-Guanine Phosphoribosyltransferase Deficiency (Lesch-Nyhan Disease) OMIM No. 300332**

Patient details provided
12 months old boy with muscular hypotonia, dystonia and slight global retardation; the sample was taken at 5 years while under treatment.

Further information
The patient was found to have elevated xanthine, hypoxanthine and uric acid in urine and plasma uric acid at 12 months of age. HPRT activity in lymphocytes was deficient (Amsterdam) confirming Lesch-Nyhan syndrome. The diagnosis was confirmed by DNA analysis. Treatment has been undertaken with allopurinol.
The sample came from Zürich Children's Hospital.

Analytical performance: Labs performing purine and pyrimidine analysis were able to detect increased hypoxanthine, xanthine and when investigated uric acid (12 of 19) whilst one lab reported upper normal values of hypoxanthine and xanthine but elevated uric acid.

Interpretative proficiency
All but two labs correctly interpreted the abnormal metabolites as HPRT deficiency (11 of 19).

Overall impression:
Diagnosis required purine and pyrimidine analysis allowing correct diagnosis (11 labs) with overall efficiency of 59%.

Analytical Details

Creatinine
n=17, no value n=2
median= 2.65
mean= 2.62
SD= 0.02
min, max= [2.25, 2.85]

pH
n=10
median= 8.00
mean= 7.95
SD= 0.24
min, max= [7.00, 9.00]

Spot tests

Nitrites
0: 2
+: 6

Ketones
0: ..... 5 Trace: 1
+: 5

Purine and Pyrimidine analysis

	n	points
Xanthine/Hypoxanthine elevated	13	1
Uric acid	8	1

Hypoxanthine
n=10
median= 458.50
mean= 453.00
SD= 90.23
min, max= [292.00, 595.00]

Xanthine
n=8
median= 500.00
mean= 490.25
SD= 101.85
min, max= [335.00, 684.00]

Uric Acid
n=5
median= 1991.00
mean= 2378.00
SD= 523.89
min, max= [1918.00, 3019.00]

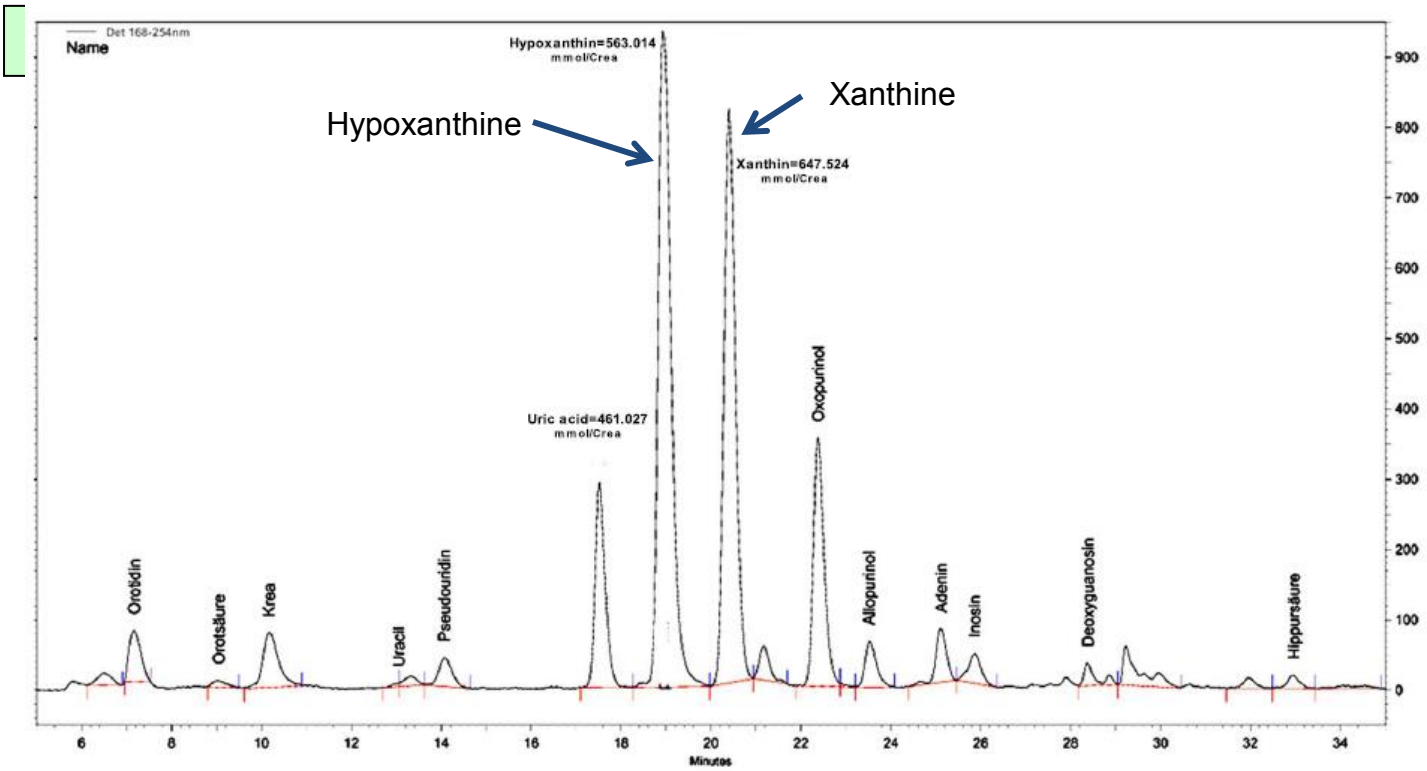
Interpretation

	n	Points
HPRT deficiency (Lesch-Nyhan)	11	2
Recommendation to perform PuPy analysis	1	1
Xanthinuria / Mb cofactor deficiency	2	0
No diagnosis/Normal	5	0

Critical error
This sample was not considered as eligible

P & P chromatogram

P & P HPLC (courtesy O. Sass, Zürich Children's Hospital)



Sample B: Purine nucleoside phosphorylase deficiency, OMIM No. 613179.

Patient details provided:
Mental retardation noted at 9 months of age. At 2 yrs there were frequent infections and stomatitis + persisting mental retardation Urine collected at 2y. 10m. on treatment.

Further information
This patient excreted massively increased purine metabolites (deoxyinosine,guanosine and inosine) and low urate pointing to purine nucleoside phosphorylase deficiency. The patient was picked up initially by urinary organic acid analysis and the diagnosis has been confirmed by DNA analysis. At 2 yrs 10 mo the boy was hospitalised in preparation for transplantation.
The sample was kindly provided by Prof. Elisabeth Holme, Gothenburg.

Analytical performance*:*
Purine and pyrimidine analysis was key to making the diagnosis with detection of abnormal metabolites (13 labs) whilst one lab found the abnormalities on organic acid analysis.

Interpretative proficiency:
14 of 19 labs correctly interpreted the abnormal findings as purine nucleoside phosphorylase deficiency.

Overall impression
14 of 19 labs were able to make the correct diagnosis, with overall proficiency of 75%.

Analytical Details

Creatinine
n=17
median= 1.15
mean= 1.15
SD= 0
min, max= [1.00, 1.26]

pH
n=10
median= 7.75
mean= 7.70
SD= 0.39
min, max= [7.00, 9.00]

Spot tests
All negative

Purine and Pyrimidine analysis (n=19)

	n	points
Increased (deoxy-)guanosine, (deoxy-)inosine	13	2
Low uric acid	9	
Normal uric acid	2	

Guanosine
n=9
median= 642.00
mean= 661.44
SD= 305.46
min, max= [256.00, 1086.00]

Inosine
n=9
median= 1415.00
mean= 1097.22
SD= 569.19min, max= [140.00, 1829.00]

Deoxyguanosine
n=8
median= 435.00
mean= 421.75
SD= 123.30
min, max= [202.00, 630.00]

Deoxyinosine
n=8
median= 546.50
mean= 495.62
SD= 199.35
min, max= [169.00, 775.00]

Uric acid
n=6
median= 45.25
mean= 44.09
SD= 1581.43
min, max= [0.03, 100.00]]

Organic acid analysis

	n	points
Increased (deoxy-)guanosine, (deoxy-)inosine	1	2

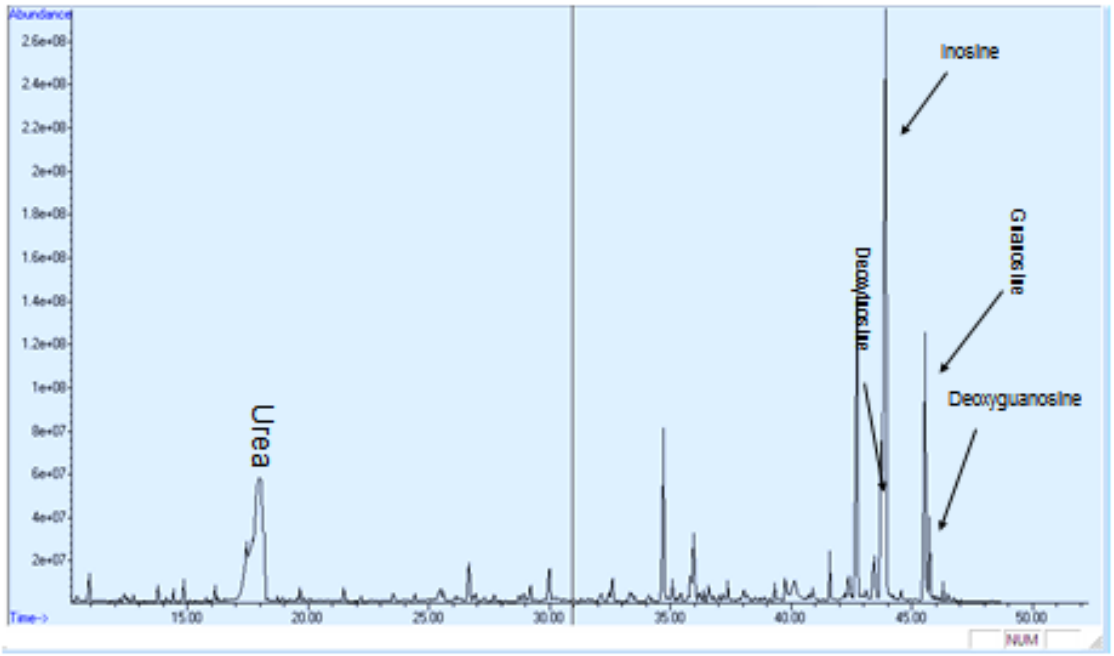
Interpretation

	n	Points
Purine nucleoside phosphorylase deficiency	14	2
combined methylmalonic and malonic acidemia	1	0
Normal pattern / no disorder	4	0

Critical error
This sample was not considered as eligible

GC-MS of metabolites

Purine nucleoside phosphorylase deficiency



Sample C: MPS IVA: Galactosamine-6-Sulfatase deficiency OMIM No. 253000.

Patient details provided:
2 ½ year old girl with growth retardation, stiff joints and abnormal dentition. Psychomotor development was normal.The urine was taken at the age of 15 years on symptomatic treatment.

Further information
The diagnosis of Morquio type A was made by the age of 3y by demonstration of elevated keratan sulphate and deficiency of galactosamine 6-sulphatase in leukocytes and fibroblasts. So far only one heterozygous mutation of the gene has been found (c-853delTTC). The patient has been treated for the last 2 years with enzyme replacement (Vimizin).
The sample came from Zürich Children's Hospital.

Analytical performance:
16 of 19 labs found elevated GAGs and 11 further identified keratan sulphate.

Interpretative proficiency:
13 labs correctly diagnosed morquio disease, one did so but had found normal keratan sulphate.
One lab found an unspecified MPS disorder (increased GAGs) whilst two labs that did not perform GAG analysis mentioned such a disorder in comments

Overall impression
This was a somewhat difficult sample due to the relatively low, but still clearly elevated level of keratin sulphate. Overall efficiency was 74%.

Analytical Details

Creatinine
n=17
median= 2.30
mean= 2.30
SD= 0.01
min, max= [2.04, 2.49]

pH
n=10
median= 6.00
mean= 6.35
SD= 0.39
min, max= [6.00, 8.00]

Spot tests
Ketones
0: ..... 7 Trace: 0
+: ..... 0 ++: 0
+++: 1

GAG screening n=3
0: 7
Trace: 0
+: 0
++: 0
+++: 1

GAG quantitative n=16

	n	Points
Elevated	15	1
Normal	1	0

Glycosaminoglycans quantitative
n=15
median= 15.50
mean= 15.78
SD= 32.56
min, max= [7.50, 30.80]

Glycosaminoglycans fractionation

Keratan sulphate	n	Points
increased	11	1
Normal	1	0

Interpretation

	n	Points
MPS type IV	14	2
MPS type IV but Keratan sulphate normal	1	1
MPS disorder as differential diagnosis	3	1
No diagnosis (test not available)	1	0

Critical error
This sample was considered as eligible if reported as completely normal with no recommendation for appropriate further testing.

GAG Electrophoresis (patient 1 = sample C

Sample D: Beta-ketothiolase deficiency, ACAT1 gene), OMIM 203750

Patient detail provided:
This female child was hospitalised at 2 years of age because of a complex viral infection associated with metabolic acidosis. Recovered well and subsequently remained healthy. Urine collected at 8 years of age whilst on specific treatment.

Further information
The sample was obtained from a 3 year old girl with keto-thiolase deficiency/2-oxothiolase deficiency who was receiving no treatment. She was hospitalised because of metabolic decompensation due to double infection with adenovirus and calicivirus when high levels of 2-methylacetoacetate, 2-methyl-3-hydroxybutyric acid and tiglylglycine were found. The diagnosis was confirmed by enzyme assay. This sample was contributed by Dr. M. Engval, Huddinge/Stockholm, Sweden. The sample had also been distributed in 2009.

Analytical performance: 19 labs performed organic acid analysis and correctly identified one or both of the key metabolites, tiglylglycine (1 point) and 2-methyl-3-hydroxy butyric acid (1 point). Another key metabolite, 2-methylacetoacetate was reported to be increased by 3 labs and excluded by 6 labs.

Interpretative proficiency:
The correct diagnosis of beta-ketothiolase deficiency as first or alternative diagnosis was made by 18 labs (scored with 2 points) whilst one lab specified only 2-methyl-3-hydroxybutyric aciduria (one point).Five labs gave 2-methyl-3-hydroxybutyryl-CoA dehydrogenase deficiency as most likely based on the lack of increased 2-methylacetoacetate which however was found by some labs. Also the presence of two isomers of 2-methyl-3-hydroxybutyric acid, indicative of ketothiolase deficiency, was reported by two labs. See chromatogram below.

Overall impression:
Good overall performance (90% efficiency) of a straight forward sample although some labs incorrectly gave 2-methyl-3-hydroxybutyryl-CoA dehydrogenase deficiency as most likely.

Analytical Details

Creatinine
n=18 (one value 0.159)
median= 1.68
mean= 1.66
SD= 0
min, max= [1.50, 1.80]

pH
n=10
median= 8.00
mean= 7.90
SD= 0.32
min, max= [7.00, 9.00]

Spot tests

All negative

Organic acid analysis (n= 19)

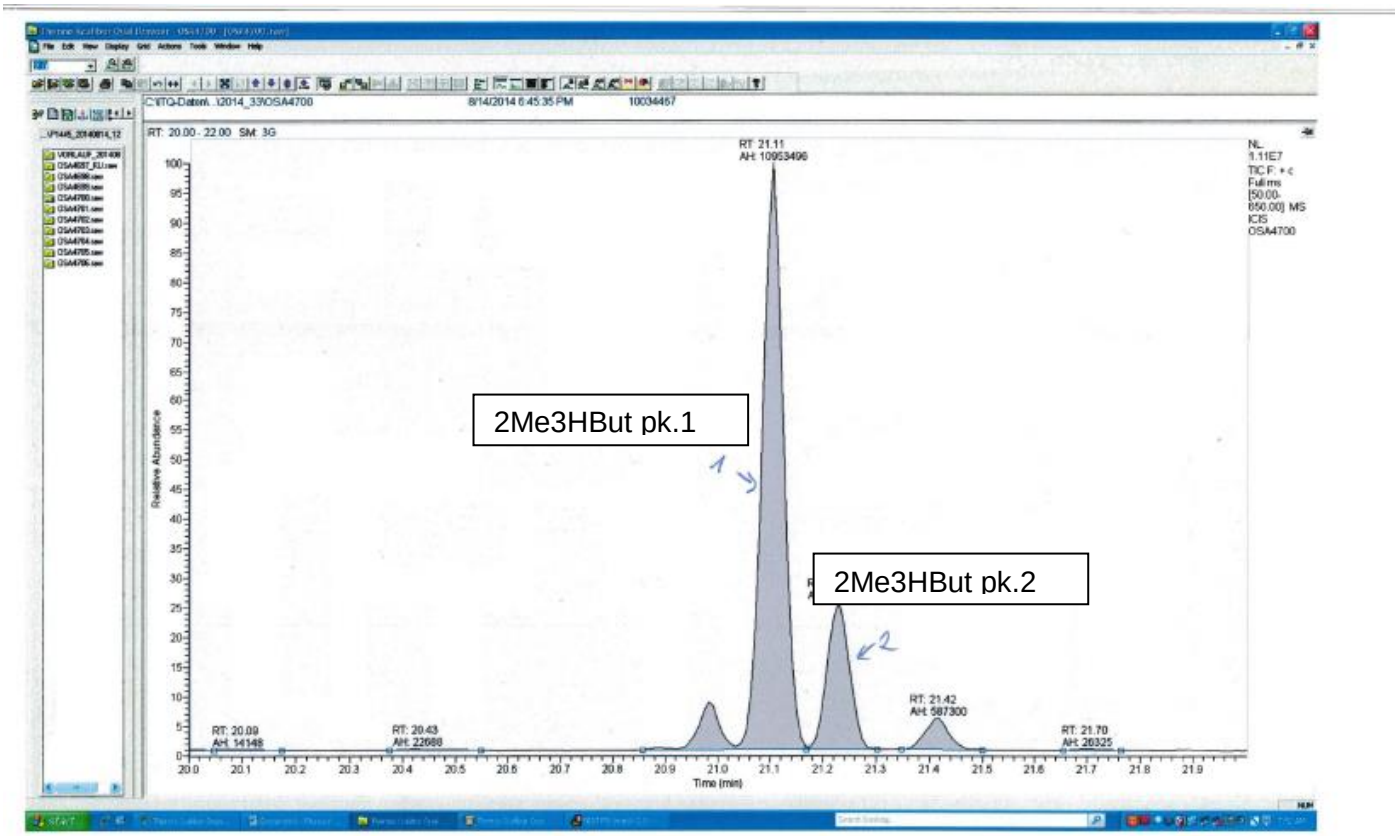
	n	points
2-methyl-3-hydroxy butyric acid elevated	19	1
Tiglylglycine elevated	18	1
Methylacetoacetic acid trace-elevated	3	-
Methylacetoacetic acid absent - normal	6	-

Organic acids quantitative **tiglylglycine** n=6 median= 52.50 mean= 59.61 SD= 22.77 min, max= [30.00, 99.70]	Organic acids quantitative **2-methyl-3-hydroxybutyrate]** n=7 median= 490.00 mean= 598.14 SD= 435.67 min, max= [213.00, 1604.00]

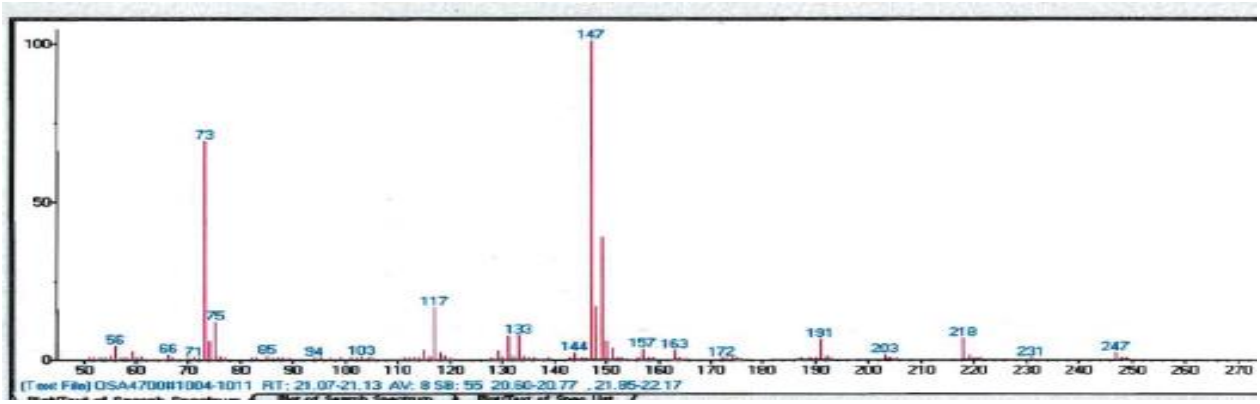
Interpretation

	n	Points
beta-ketothiolase deficiency	13	2
2-methyl-3-hydroxybutyryl-CoA dehydrogenase deficiency with ketothiolase possible	5	1
Only 2-methyl-3-hydroxybutyryl-CoA dehydrogenase deficiency	1	0

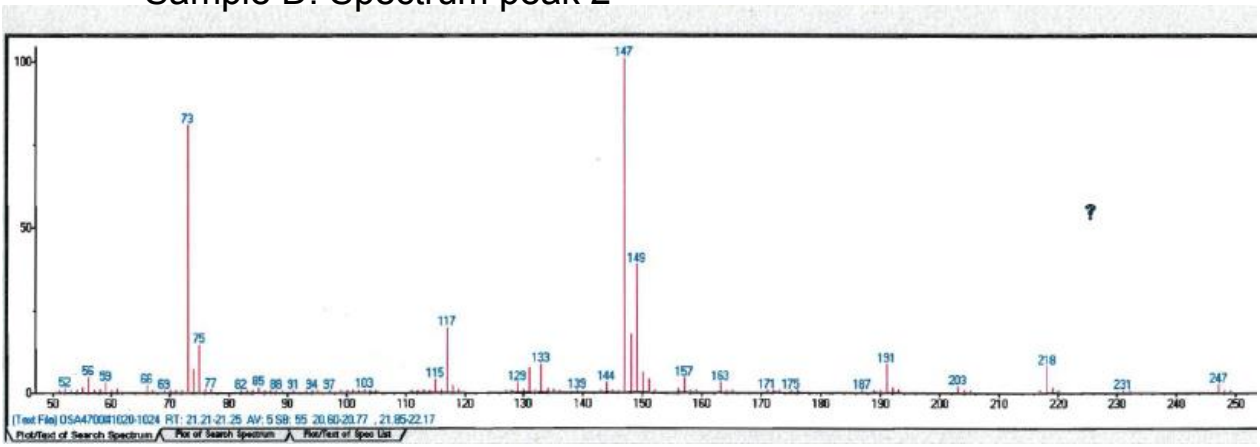
GC-MS of organic acids (O.Sass Zürich) showing two forms of 2Me3HB



Sample D: Spectrum peak 1



Sample D: Spectrum peak 2



Sample E: Hypermethioninaemia due to methionine adenosyltransferase deficiency, OMIM 250850 (MAT1A gene).

Patient details provided:
4 years old girl on a mainly vegetarian diet. No clinical symptoms.

Further information
4 years old girl detected on newborn screening which showed high methionine, she has never had clinical symptoms, is on a mostly vegetarian „diet". Mutation analysis showed homozygosity for IVS2+1G>A (c.169+1G>A in intron 2). This sample was kindly provided by Dr. Sabine Scholl, Innsbruck.

Analytical performance:18 labs correctly reported high methionine (2 points). Other abnormalities were also reported: figlu(3 labs);homocitrulline(1); pipecolic acid (1); saccharopine (1); arginine (2); sarcosine (1).

Interpretative proficiency:
Although all but one lab found high methionine only 5 labs correctly diagnosed MATI/III deficiency.

Overall impression:
Good analytical proficiency but poor interpretation of this finding.

Analytical Details

Creatinine
n=18 (one value 0.455)
median= 4.90
mean= 4.84
SD= 0.03
min, max= [4.45, 5.10]

pH
n=10
median= 7.00
mean= 7.00
SD= 0.00
min, max= [7.00, 7.00]

Spot tests
All negative

Amino acid analysis

	n	points
Methionine elevated	18	2
figlu	3	
arginine	2	
homocitrulline	1	
pipecolic acid	1	
saccharopine	1	
sarcosin	1	

Amino acids quantitative
methionine
n=17
median= 60.50
mean= 108.89
SD= 140.46
min, max= [31.00, 498.00]

GAG fractionation

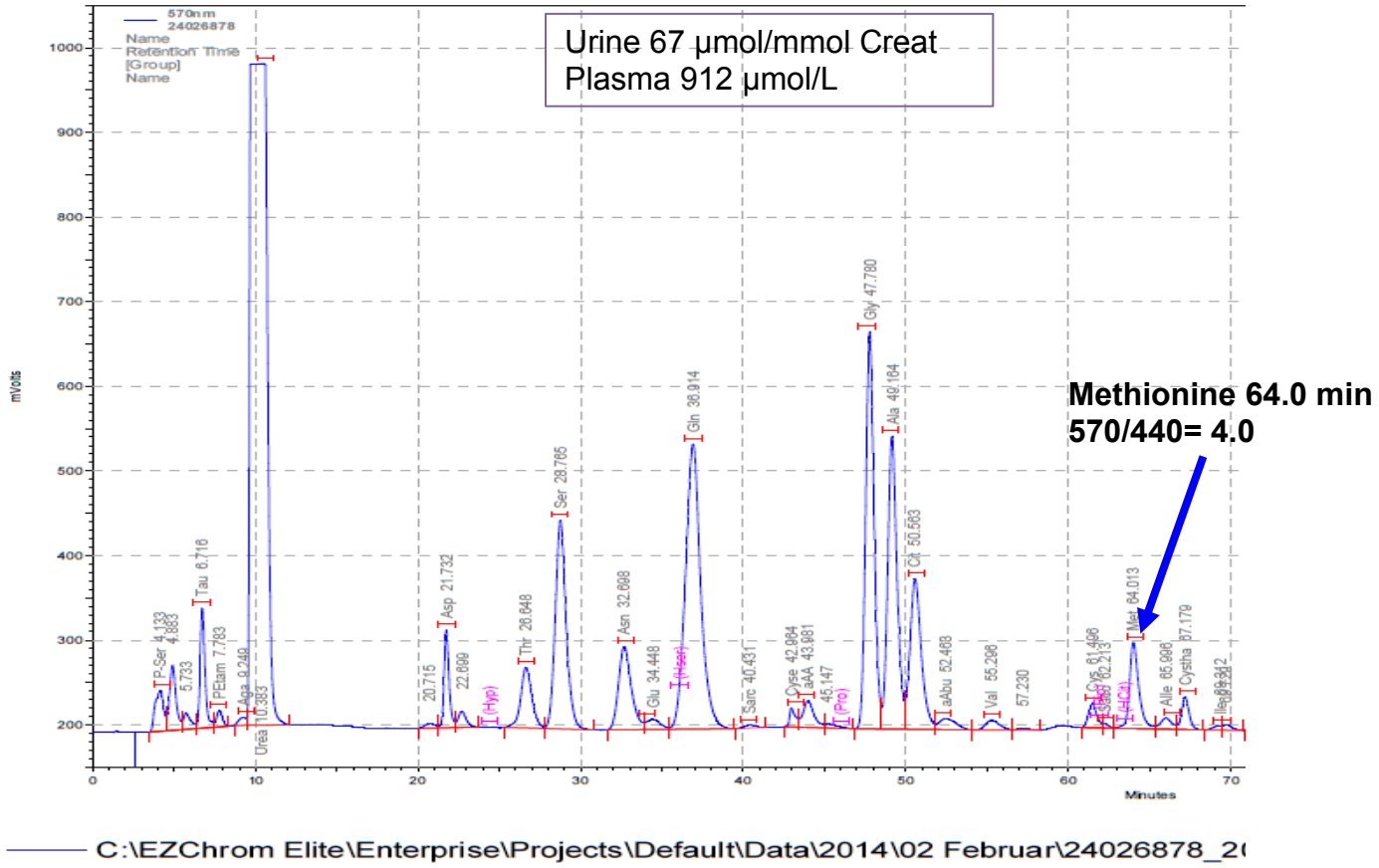
Dermatan Sulphate / Heparan Sulphate	12
Dermatan Sulphate	4
Not Done	3

Interpretation

	n	Points
Methionine S-adenosyl transferase deficiency	5	2
Exclude MAT	1	1
CBs deficiency	1	0
Figlu-uria	6	0
Folic acid deficiency	1	0
Peroxisomal disorder	1	0
Sacharopinuria	1	0
No disorder	3	0

IE Chromatogram

IE Chromat (courtesy O. Sass)



Sample F: Hyperornithinemia-hyperammonemia-homocitrullinuria (HHH) syndrome (treated with citrulline), mutation of the SLC25A15 gene, OMIM 238970.

This was the common sample and full details of the performance within all schemes can be found on the ERNDIM website
(http://www.erndim.org/store/docs/BFv2.DPT2014commonsample-VUHAABAA354192-01-10-2014.pdf)

Patient details provided:
Following uneventful pregnancy and birth this male child showed mild hypotonia at 6 months of age. A few months later, developmental delay and failure to thrive with elevated transaminases was observed. The urine was collected at the age of 8.75 years whilst receiving specific treatment.

Further information
The child showed the first symptoms at 6 months, then at 14 months liver dysfunction when increased Ammonia (200) increased ornithine, orotic acid and mild increase of homocitrulline was found leading to diagnosis of HHH . Mutation in exon 2 of SLC25A15 found - c.208_209delGCinsTT. The sample came from Zürich Children's Hospital.

Analytical performance*:*
Only 6/19 labs reported elevated homocitrulline (one point) but all labs found elevated orotic acid and / or uracil (one point).

Interpretative proficiency:
All labs reported an abnormality of the urea cycle of various types (one point) and 9 labs reported HHH syndrome (two points) based on finding increased homocitrulline or specified this as a possible disorder.

Overall impression:
All labs reported an abnormality of the urea cycle of various types (one point) and 9 labs reported HHH syndrome (two points) based on finding increased homocitrulline or specified this as a possible disorder.

Analytical Details

Creatinine
n=18 (one value 0.88)
median= 9.47
mean= 9.40
SD= 0.16
min, max= [8.60, 10.10]

pH
n=10
median= 7.00
mean= 7.00
SD= 0.00
min, max= [7.00, 7.00]

Spot tests
All negative

Amino acid analysis

	n	points
Homocitrulline increased	6	1
Citrulline increased	19	0
Methionine elevated	2	0
Ornithine elevated	15	0
Arginine elevated	17	0

Homocitrulline]
n=6
median= 53.50
mean= 85.26
SD= 64.94
min, max= [28.00, 199.00]

Citrulline
n=19
median= 60.00
mean= 106.87
SD= 136.79
min, max= [51.70, 596.00]

Ornithine
n=15
median= 39.00
mean= 94.78
SD= 116.27
min, max= [24.00, 366.40]

Arginine
n=17
median= 38.70
mean= 81.54
SD= 124.43
min, max= [14.62, 496.00]

Organic acid analysis

	n	points
Orotic acid elevated	14	1

Organic acids column chromatography/**orotic acid**
n=5
median= 37.90
mean= 42.73
SD= 20.47
min, max= [17.00, 68.79]

Purine/Pyrimidine analysis

	n	points
Orotic acid / uracil elevated	14	1

Orotic acid
n=3
median= 14.00
mean= 17.00
SD= 4.24
min, max= [14.00, 23.00]

Uracil
n=6
median= 90.50
mean= 92.93
SD= 39.33
min, max= [51.00, 143.60]

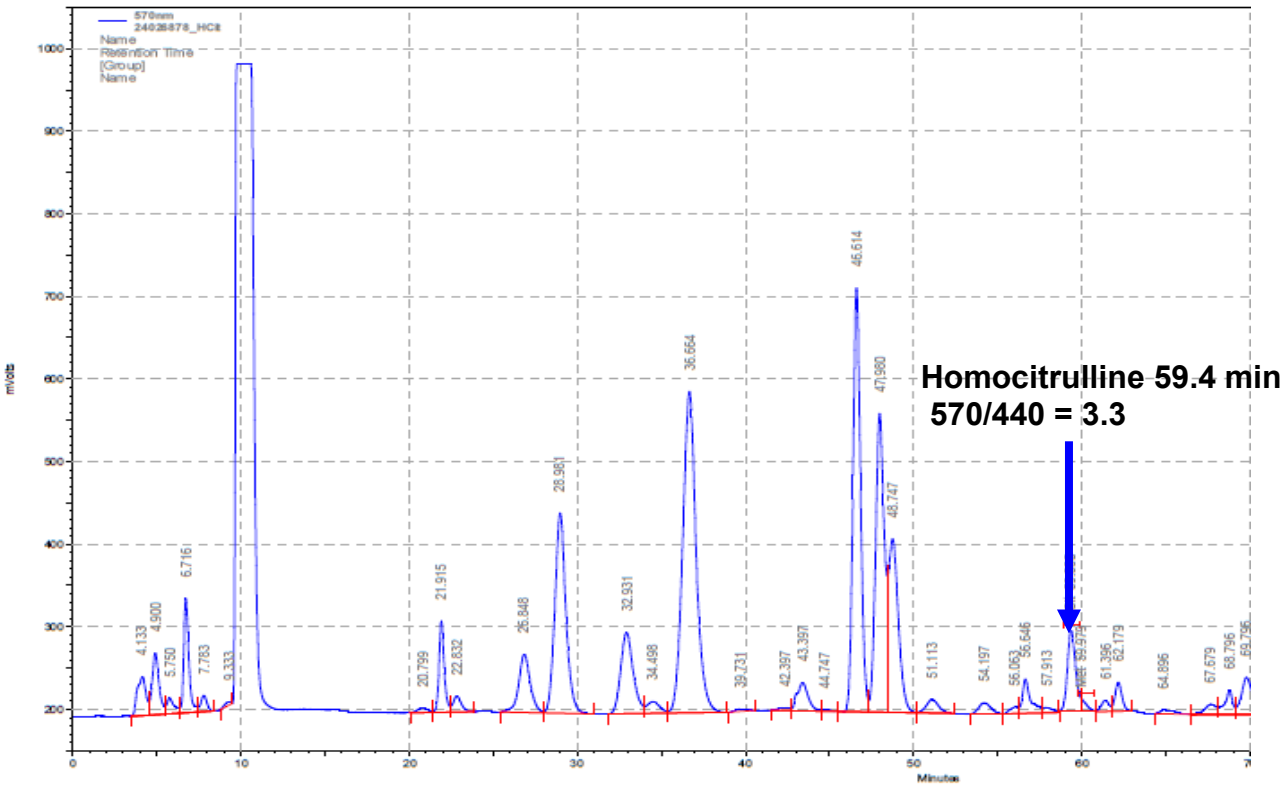
Interpretation

	n	Points
Hyperornithinemia-hyperammonemia-homocitrullinuria (HHH) syndrome	7	2
HHH specified as differential	2	2
OTC deficiency	6	1
Citrullinaemia	3	1
Arginase deficiency	1	1

Critical error

Failure to detect elevated orotic acid was considered to be a critical error

IE Chromatogram



8. Scores

Overall proficiency

Sample	Diagnosis	A (%)	I (%)	total (%)
A	Hypoxanthine-Guanine Phosphoribosyltransferase Deficiency (Lesch-NyhanDisease) OMIM No. 300332	58	61	59
B	Purine nucleoside phosphorylase deficiency, OMIM No. 613179.	74	76	75
C	MPS IVA: Galactosamine-6-Sulfatase deficiency OMIM No. 253000.	71	76	74
D	Beta-ketothiolase deficiency, ACAT1 gene), OMIM 203750	97	82	90
E	Hypermethioninaemia due to methionine adenosyltransferase deficiency, OMIM 250850 (MAT1A gene).	95	31	63
F	Hyperornithinemia-hyperammonemia-homocitrullinuria (HHH) syndrome (treated with citrulline), mutation of the SLC25A15 gene, OMIM 238970.	66	74	70

Total scores

	Survey 1			Survey 2			
Lab No	A	B	C	D	E	F	Total
1	0	4	4	4	2	4	18
2	1	0	2	2	2	2	9
3	3	4	4	4	4	2	21
4	4	4	4	4	4	4	24
5	4	4	2	3	0	4	17
6	4	4	2	3	2	2	17
7	4	4	4	4	2	2	20
8	1	0	0	3	4	4	12
9	3	4	4	3	4	3	21
10	4	4	4	4	3	2	21
11	3	4	2	4	2	2	17
12	4	4	2	3	4	2	19
13	1	4	4	4	2	2	17
14	1	4	1	4	2	4	16
15	4	4	4	4	3	3	22
16	0	0	1	4	2	3	10
17	0	1	4	4	2	4	15
18	0	0	4	3	2	2	11
19	4	4	4	4	2	2	20

The scores proposed by us were evaluated by a **second advisor** and confirmed at the Scientific Advisory Board meeting in November. At this meeting the cut off point for satisfactory performance was set at **at least 16 points**. Labs failing to reach this mark will receive a performance advice letter.

Detailed Scores: A,B,C

Lab no	Sample A HPRT def.			Sample B PNP def.			Sample C MPS IVA			
	A	I	Total	A	I	Total	A	I	Total	Total
1	0	0	0	2	2	4	2	2	4	**8**
2	1	0	1	0	0	0	1	1	2	**3**
3	1	2	3	2	2	4	2	2	4	**11**
4	2	2	4	2	2	4	2	2	4	**12**
5	2	2	4	2	2	4	1	1	2	**10**
6	2	2	4	2	2	4	1	1	2	**10**
7	2	2	4	2	2	4	2	2	4	**12**
8	0	1	1	0	0	0	0	0	0	**1**
9	1	2	3	2	2	4	2	2	4	**11**
10	2	2	4	2	2	4	2	2	4	**12**
11	1	2	3	2	2	4	1	1	2	**9**
12	2	2	4	2	2	4	1	1	2	**10**
13	1	0	1	2	2	4	2	2	4	**9**
14	1	0	1	2	2	4	0	1	1	**6**
15	2	2	4	2	2	4	2	2	4	**12**
16	0	0	0	0	0	0	0	1	1	**1**
17	0	0	0	0	1	1	2	2	4	**5**
18	0	0	0	0	0	0	2	2	4	**4**
19	2	2	4	2	2	4	2	2	4	**12**
ratio	22/38	23/38	**45/76**	28/38	29/38	**57/76**	27/38	29/38	**56/76**	
%	58	61	59	74	76	75	71	76	74	

Detailed Scores: D,E,F

Lab no	Sample D Ketothiolase def.			Sample E MAT def.			Sample F HHH syndrome			
	A	I	Total	A	I	Total	A	I	Total	Total
1	2	2	4	2	0	2	2	2	4	**10**
2	2	0	2	2	0	2	1	1	2	**6**
3	2	2	4	2	2	4	1	1	2	**10**
4	2	2	4	2	2	4	2	2	4	**12**
5	1	2	3	0	0	0	2	2	4	**7**
6	2	1	3	2	0	2	1	1	2	**7**
7	2	2	4	2	0	2	1	1	2	**8**
8	2	1	3	2	2	4	2	2	4	**11**
9	2	1	3	2	2	4	1	2	3	**10**
10	2	2	4	2	1	3	1	1	2	**9**
11	2	2	4	2	0	2	1	1	2	**8**
12	2	1	3	2	2	4	1	1	2	**9**
13	2	2	4	2	0	2	1	1	2	**8**
14	2	2	4	2	0	2	2	2	4	**10**
15	2	2	4	2	1	3	1	2	3	**10**
16	2	2	4	2	0	2	1	2	3	**9**
17	2	2	4	2	0	2	2	2	4	**10**
18	2	1	3	2	0	2	1	1	2	**7**
19	2	2	4	2	0	2	1	1	2	**8**
ratio	37/38	31/38	**68/76**	36/38	12/38	**48/76**	25/38	28/38	**53/76**	
%	97	82	90	95	31	63	66	74	70	

9. Assessment of performance

Steps have been taken within the Scientific Advisory Board of ERNDIM to set the level of good performance within a proficiency scheme. Letters of support to those laboratories with clear poor performance will be issued. The level for satisfactory performance for this year will be set at the SAB meeting in November. A special meeting of scientific advisors took place in November 2012 to consider how to harmonise scoring within all our qualitative schemes and the question of introducing critical errors in our schemes. Here it was decided to incorporate recommendations into interpretation giving a 2 plus 2 scoring system. Also the concept of **critical error** was accepted to be introduced in **2014.** Thus labs failing to make a correct diagnosis of a sample considered as eligible for this category will be deemed not to have reached a satisfactory performance even if their total points for the year exceed the limit set at the SAB.

10. Annual meeting

The annual meeting of participants of the 5 DPT centres took place during the SSIEM symposium in Innsbruck on Tuesday, September 2nd, 09.00.

11. Changes planned for 2015

No changes are envisaged

12. Tentative schedule and fee in 2015

Sample distribution	April 07, 2015
Start of analysis of Survey 2015/1	April 27, 2015
Survey 2015/1 - Results submission	May 18, 2015
Survey 2015/1 - Reports	July 10, 2015
Start of analysis of Survey 2015/2	June 08, 2015
Survey 2015/2 – Results submission	June 29, 2015
Survey 2015/2 - Reports	August 15, 2014
Annual meeting of participants	SSIEM, Lyon, September 1, 2015
Annual Report 2015	April 2016

Fee will follow.

13. ERNDIM certificate of participation

A combined certificate of participation covering all EQA schemes will be provided to all participants who take part in any ERNDIM scheme. For the DPT scheme this certificate will indicate if results were submitted and whether satisfactory performance was achieved in the scheme.

Basel / Zürich, August 2014 Revised April 2015.

Brian Fowler
Scientific advisor