ERNDIM DPT Centre

University Children's Hospital Basel
Metabolic Unit

Postfach, CH-4031Basel, Switzerland

Diagnostic Proficiency Testing- Basel

Survey 2011

Annual Report

prepared by
Brian Fowler

1. Geographical distribution of participants

In 2011, 21 laboratories from 10 countries participated in the scheme and all participants bar one submitted results in at least one of the two surveys.

Country	Number of participants
Austria	1
Canada	3
China	1
Estonia	1
Germany	6
Norway	1
Sweden	2
Switzerland	2
UK	1
USA	3

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 May 5th at ambient temperature by CSCQ using the courier DHL.
Delivery times of samples reported by the courier ranged from 1 to 5 days.

3. Tests

Analyses of amino acids, organic acids, mucopolysaccharides, oligosaccharides and purines/ pyrimidines were required in 2011.

4. Schedule of the scheme in 2010

Sample distribution	May 05, 2011, Tuesday
Start of analysis of Survey 2011/1	May 09, 2011, Monday
Survey 2011/1 - Results submission	May 30, 2011, Monday
Survey 201/1 - Reports	June 13, 2011, Monday
Start of analysis of Survey 2011/2	June 20, 2011, Monday
Survey 2011/2 – Results submission	July 11, 2011, Monday
Survey 2011/2 - Reports	August 05, 2011, Friday
Annual meeting of participants	August 30, 2011, in Geneva at SSIEM
Annual Report 2011	December

5. Receipt of samples and results

Receipt of samples (sent on May 10, 2010)

Receipt (days after shipment)	Delivery reported by DHL
1 day	14
3 days	1
4 days	3
5 days	3

Date of reporting of results

Due to the introduction of the new website entry for results we extended the originally planned deadlines.
A,B,C: deadline 30.05.2011: delay : before deadline n=12, 1 day - n= 4, 2 days - n=1, 3 days - n=2, 12 days. One participant failed to return results and was excluded from the evaluation of results.
D,E,F: deadline 11.07.2011 nineteen labs returned results by the deadline. Two laboratories failed to return results and were was excluded from the evaluation of results

6. Scoring system

Three criteria are evaluated: analytical performance, interpretative proficiency and 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	Good (diagnosis was established)	2	max 2
		Helpful but incomplete	1	
		Misleading or wrong diagnosis	0	
R	Recommendations	Helpful	1	max 1
		Unsatisfactory or misleading	0	

The **total score** is calculated as a sum of these three criteria. The maximum to be achieved is 5 points per sample. The scores were calculated only for laboratories submitting results.

7. Results of samples and evaluation of reporting

Sample A: (Very) long chain acylCoA dehydrogenase deficiency

Patient details
The patient presented on the second day of life with a cardiac arrest and was resuscitated. There was lactic acidaemia, a massive increase of plasma CPK and cardiomyopathy. The urine was obtained at the age of one year whilst on treatment.
This patient has long chain acylCoA dehydrogenase deficiency, confirmed by enzyme assay in fibroblasts and by mutation analysis.

Analytical performance: Organic acid analysis was critical.
As well as increased lactate the finding of small but significant amounts of unsaturated dicarboxylic acids was critical in pointing to the correct diagnosis. 19 labs reported on organic acid analysis but only 8 labs reported the key metabolites. Overall analytical performance was 57.5 %.
Interpretative proficiency: The correct diagnosis was considered to be a long chain fatty acid disorder which was scored two points. Mention of another or unspecified mitochondrial fatty acid disorder scored one point. Overall interpretative proficiency was 42.5.%.
Recommendations: Measurement of acylcarnitines and / or relevant enzyme /mutation analysis was considered helpful.

Overall impression: This was a difficult case reflecting the organic acid profile in the non-crisis situation in this patient.

Analytical Details

Creatinine (n=20) 0.37 – 0.86, median 0.73

pH (n=11) 5 – 8, median 7.5

Spot tests

Glucose	Protein	Blood
+ n=4 0 n=8	+ n=9 0 n=1	++ - +++ n=7 0 n=1

Organic acid analysis (n=19)

	n	points
Unsaturated dicarboxylic acids	8	2
Dicarboxylic aciduria	13	1

Special assays – acyl carnitines (n=5)

	n	points
Free elevated	4	
C6, C8, C10 elevated	1	
C4 – C8 elevated	1	
"others"	1	

Interpretation

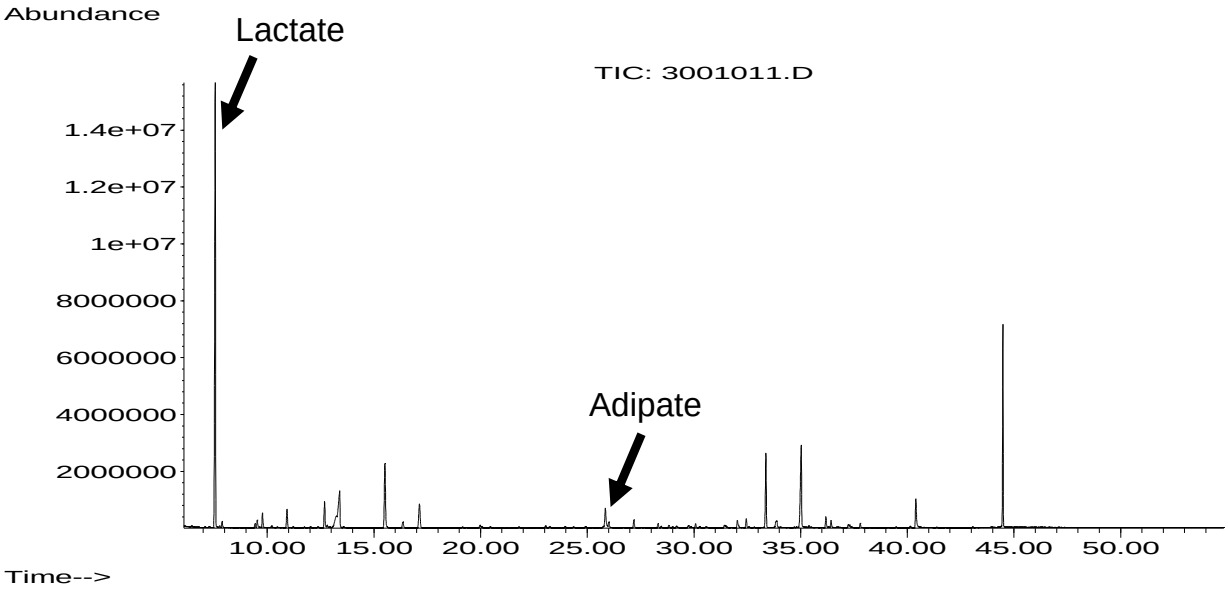
	n	Points
Long-chain fatty acid oxidation defects (very-long-chain Acyl-CoA dehydrogenase Deficiency)	5	2
Carnitine palmitoyltransferase II (CPT2) deficiency. /Carnitine transport defect	4	1
Long chain fatty acid disorder	2	1
LCHAD deficiency / Mitochondrial trifunctional protein deficiency	3	0
Lactic acidosis of any cause / Respiratory chain defect	6	0

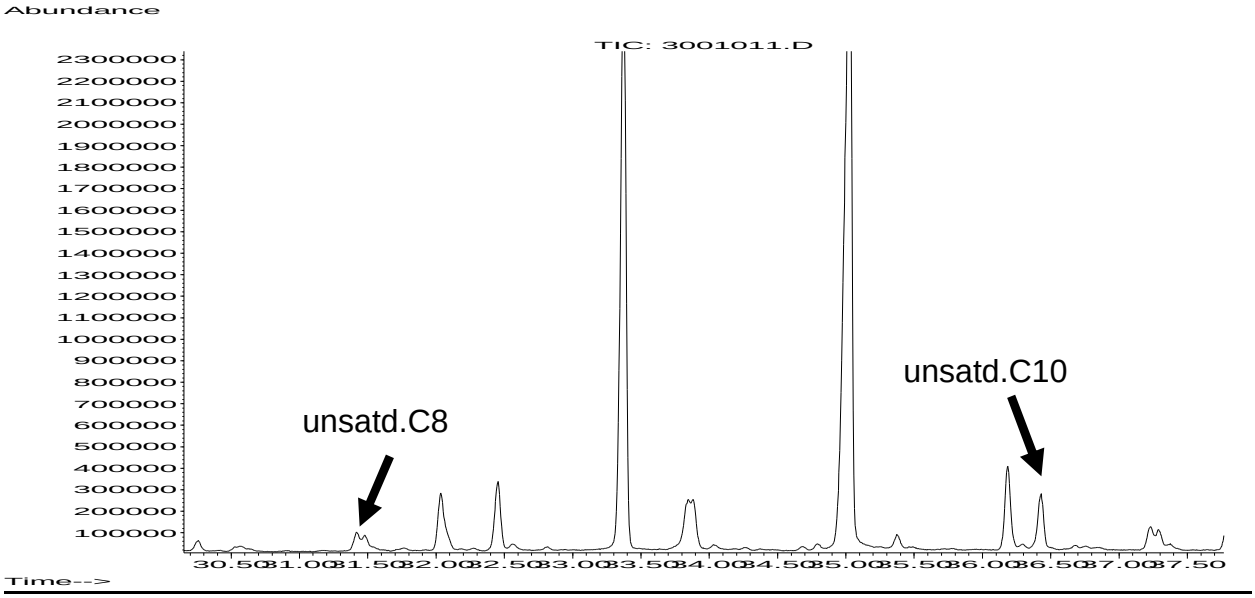
Recommendations for further tests

	n	points
acylcarnitines in blood	11	1
Relevant enzyme / mutation analysis	1	1
Not relevant enzyme / mutation analysis	3	0
Cardiomyopathy investigations	1	0
Mitochondrial disease investigations	2	0
Free carnitine in blood	1	0
Further clinical information	1	0

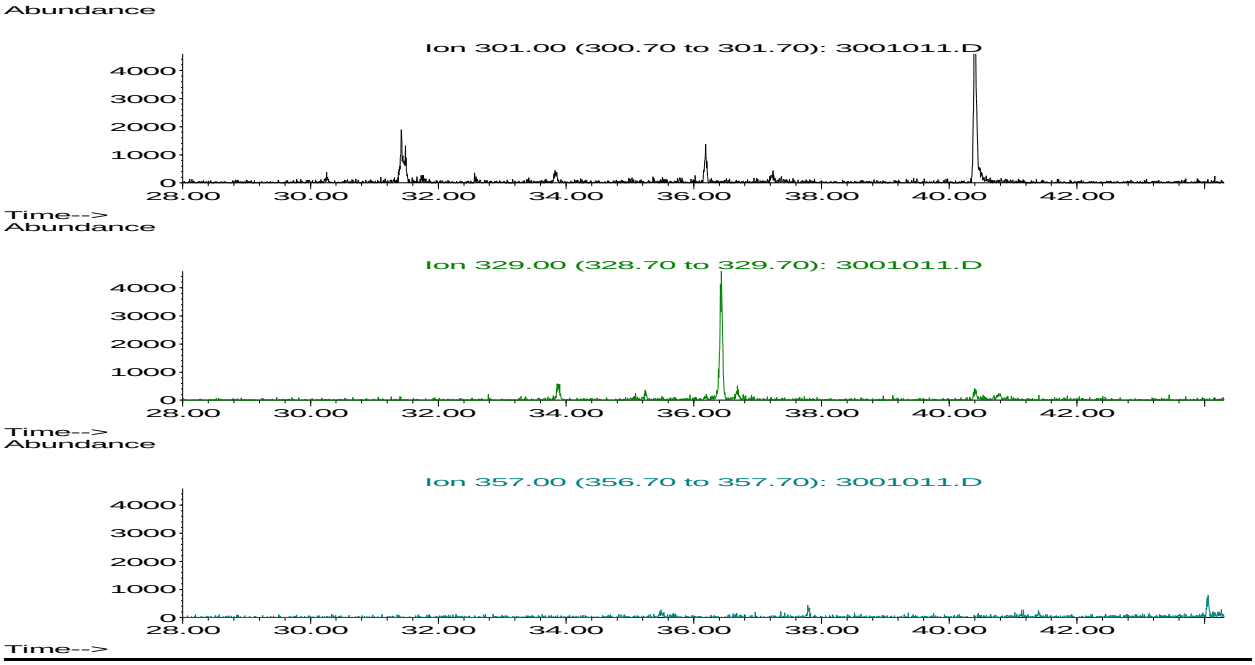
Chromatograms etc.

GC/MS total ion chromatogram

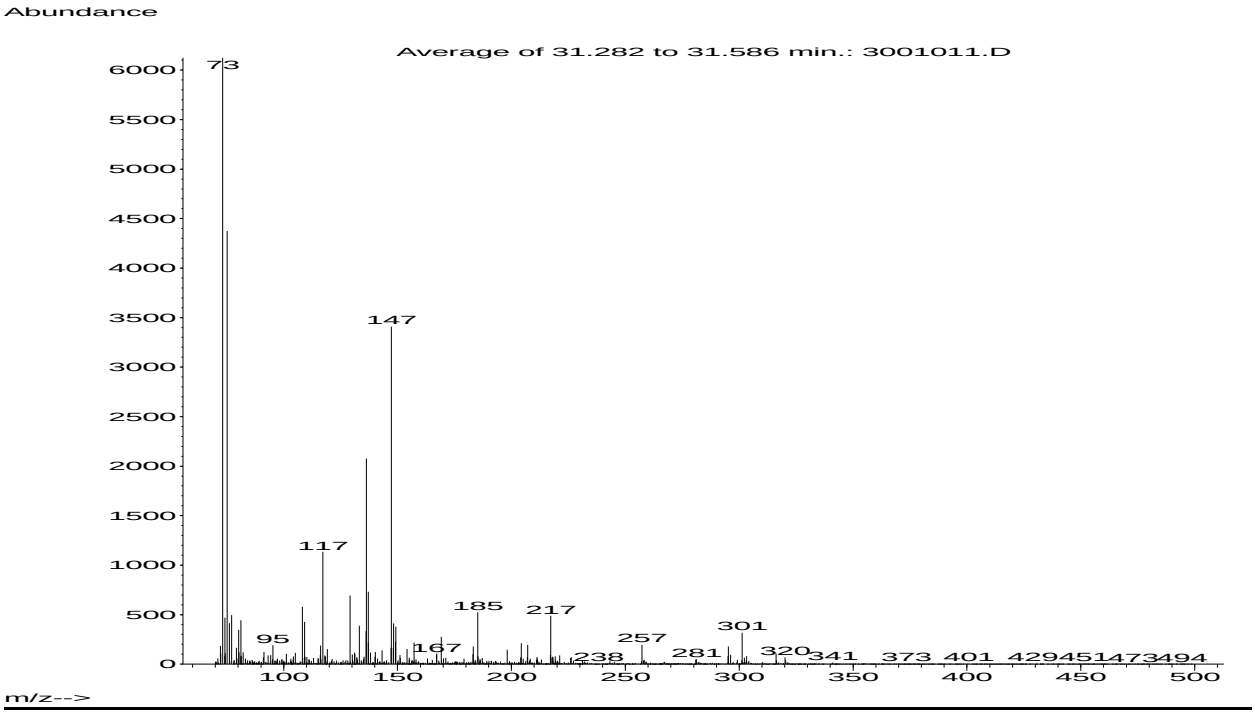




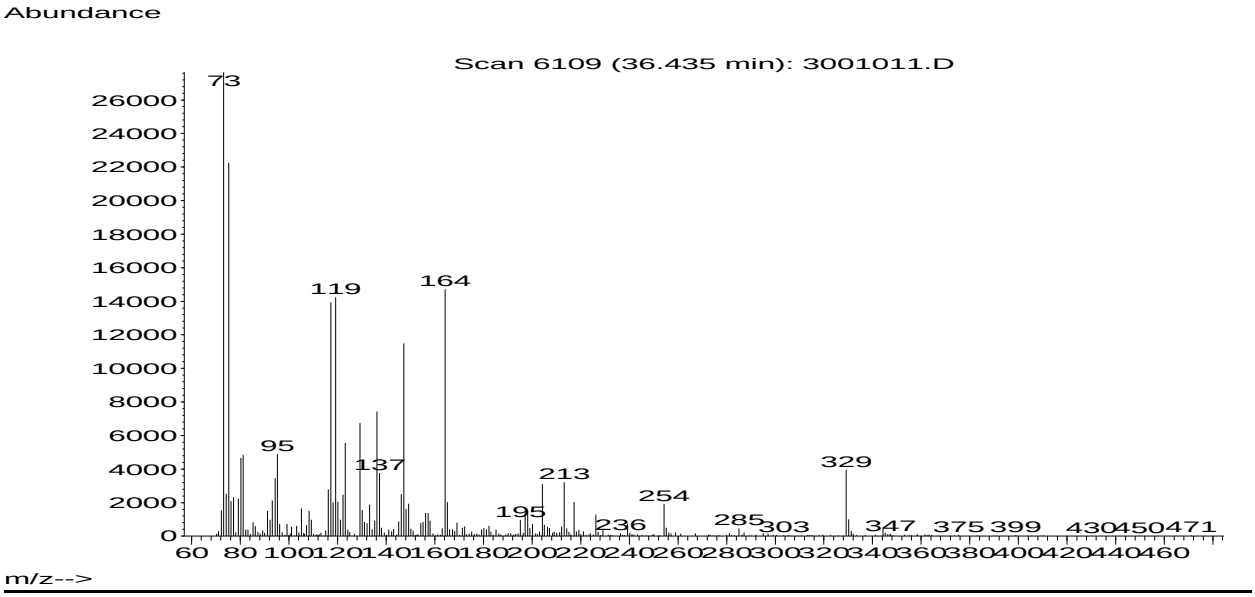
Extracted ions: 301 (Unsatd C8); 329 (unsatd C10); 357 (unsatd C12)



unsatd.C8 Mass Spectrum



unsatd.C10 Mass Spectrum



Sample B: Non-Ketotic Hyperglycinaemia

Patient:
The patient was admitted to hospital on the second day of life because of a severe apnoeic attack and convulsions. He was neurologically abnormal with hypertonicity, hyperexcitability and abnormal EEG. The urine was collected at 3 years of age whilst on treatment.
The patient was diagnosed with non-ketotic hyperglycinaemia due to high CSF and plasma Glycine, subsequently confirmed by enzyme assay

Analytical performance*:* Amino acid analysis was considered essential for this sample, performed by 20 labs. All but one lab found greatly elevated glycine. Organic acid analysis was also widely performed and metabolites related to treatment were found. Overall analytical performance was 95 %.

Interpretative proficiency: a diagnosis of non-ketotic hyperglycinaemia (glycine encephalopathy) was considered correct. Overall performance in interpretation was 85 %.

Recommendations*:* Analysis of glycine in both plasma and CSF was considered essential and enzyme and mutation analysis as helpful.

Overall impression:
This was considered to be a straightforward sample but incorrect analysis in one case and incorrect interpretation in two others led to the lower than expected overall level of performance.

Analytical Details

Creatinine (n=20) 0.99 – 1.68, median 1.48

pH (n=12) 5-7, median 6.5

Spot tests

Amino acid analysis (n=20)

	n	points
Elevated Glycine	19	2
Normal amino acids	1	0

	n	Range	Median
Glycine	19	1515 - 42000	3276

Organic acid analysis (n= 19)

	n	points
Benzoate elevated	16	0
Hippurate elevated	17	0
Normal	1	0

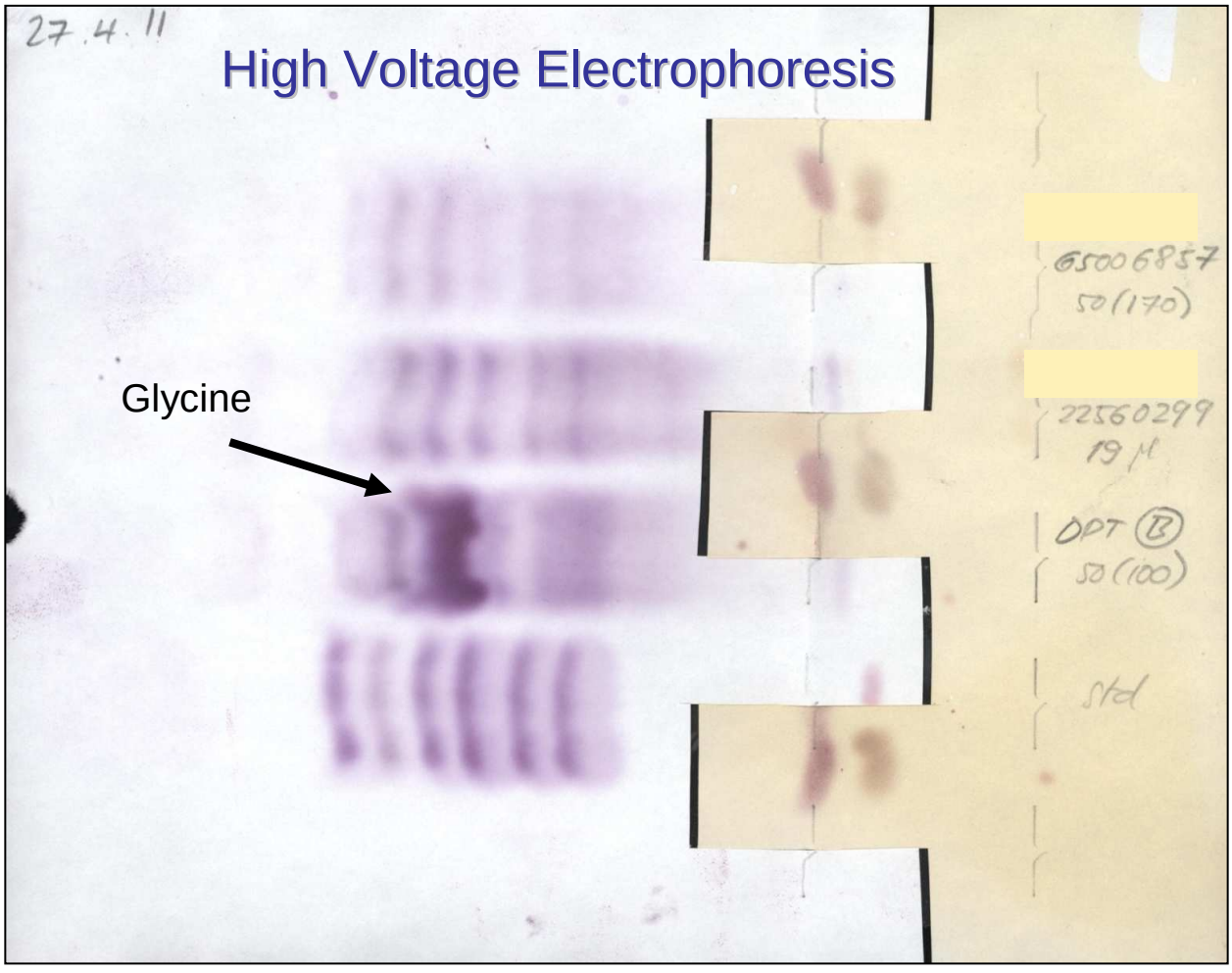
Interpretation

	n	Points
Glycine encephalopathy / nonketotic hyperglycinemia (NKH; MIM #605899)	16	2
Non-ketotic hyperglycinemia on benzoate treatment	1	2
Hyperammonemia under treatment due to NAGS or CPS deficiency	2	0
No diagnosis by urine organic acid analysis or urine amino acid analysis.	1	0

Recommendations for further tests

	n	points
Plasma **and** CSF glycine	15	1
Plasma glycine	2	0
Relevant enzyme / mutation analysis	15	1
Not relevant enzyme / mutation analysis	2	0
No recommendation	1	0

Chromatograms etc.



Sample C: MPS I

Patient:
A female child presented in early childhood with coarse facies and developed abnormalities of joint functions and moderately delayed mental development. Urine was collected at the age of 30y.
This patient has MPS I confirmed by enzyme assay.

Analytical performance*:* mucopolysaccharide analysis was considered essential. The finding of increased GAG and dermatan sulphate with or without heparan sulphate was considered correct. 19 laboratories performed mucopolysaccharide analysis and all found increased GAG which received 1 point. 1 additional point was given for GAG differentiation with identification of dermatan sulphate. One laboratory does not perform GAG analysis and recommended that this should be done. The overall analytical performance of this sample was 82.5% although only one lab failed to make a diagnosis of an MPS disorder.

Interpretative proficiency: a diagnosis of MPS in general received 1 point, and 1 point was given for mention of MPS type I. The interpretative proficiency for this sample was 87.5%.

Recommendations*:* confirmation of diagnosis by enzyme assay (α-iduronidase), mutation analysis (*IDUA* gene), GAG quantitation and differentiation in labs not performing those tests were considered helpful.

Overall impression:
This was a straightforward sample that we would expect to be identified as an MPS disorder.

Analytical Details

Creatinine (n=20) 2.61 – 4.03, median 3.55

pH (n=13) 5 -6, median 6

Spot tests

GAG screening n=4		
+ - +++		

GAG analysis (n=19)

GAG quantitative	n	points
elevated	17	1

	n	Range	Median
GAG g/mol Creat	16	0.04 - 76	34.7

GAG differentiation	n	points
Dermatan sulphate	6	1
Dermatan + heparan Sulphate	4	1

Interpretation

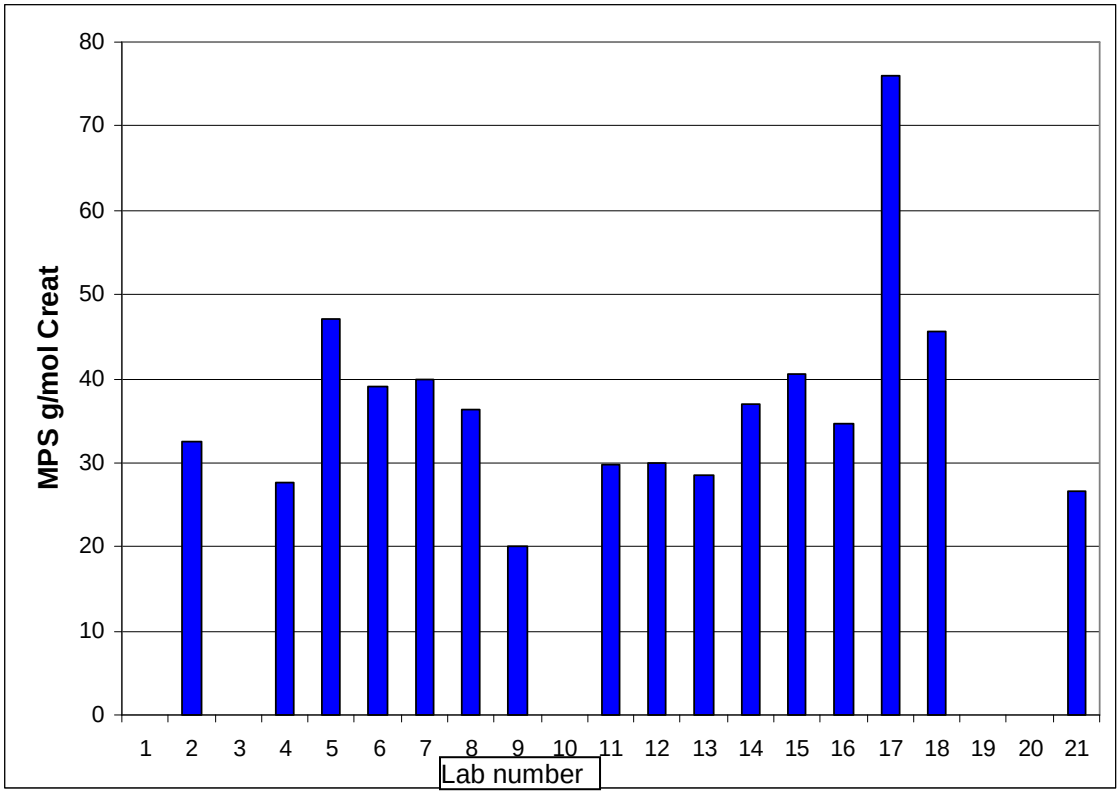
	n	Points
Mucopolysaccharidosis type I (Hurler/Scheie).	10	2
MPS I, II or VI.	2	2
MPS 1 or MPS 2	1	2
MPS VI (MPS I)	2	2
Highly suspicious for MPS	4	1
No diagnosis by urine amino or urine organic acid analysis. Clinical description suggests mucopolysaccharidosis	1	0

Recommendations for further tests

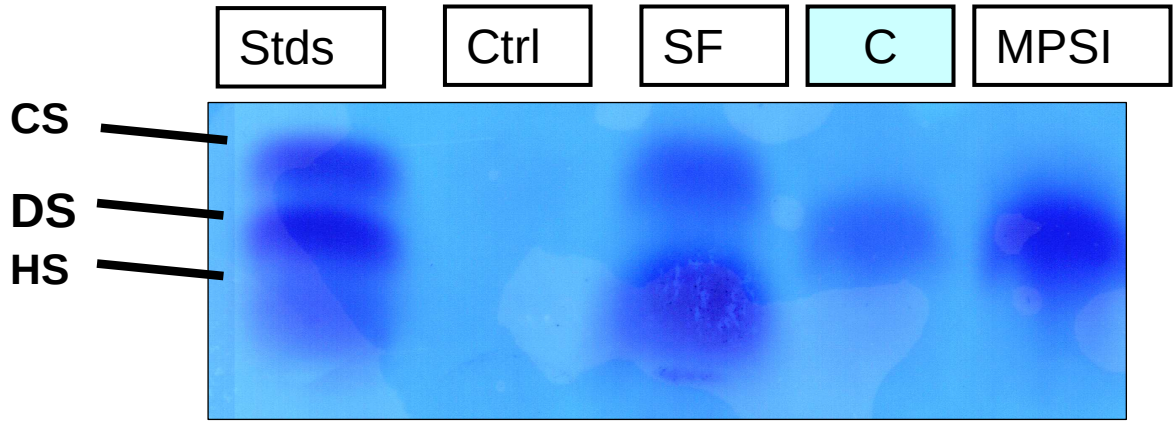
	n	points
Alpha-Iduronidase assay in leucocytes or fibroblasts	14	1
Relevant enzyme / mutation analysis	2	1
GAG differentiation if not done in own lab	5	1

Chromatograms etc.

MPS quantitation



MPS Electrophoresis (courtesy P. Burda, Children's Hospital, Zürich)



Sample D: Glyceroluria associated with Xp21 contiguous gene deletion

Patient:
The patient is one of male twins, both affected with congenital *adrenal hypoplasia, hypogonadotropic hypogonadism, duchenne muscular dystrophy and mental retardation. Urine was obtained at the age of 17 months during admission to hospital due to an infection.
The patient was diagnosed with Xp21 contiguous gene deletion.
The sample was provided by Dr. Sabine Scholl-Bürgi from Innsbruck, Austria.

*Note the mistake of "renal" instead of "adrenal" in the details distributed with the sample.

Analytical performance: Organic acid analysis or a specific method for glycerol was essential and was done by all labs. Overall performance was 100%.

Interpretative proficiency: The correct diagnosis was considered to be glyceroluria associated with Xp21 contiguous gene deletion. Proficiency for interpretation for this sample was also good at 100%.

Recommendations: Genetic and / or enzyme analysis is considered helpful.

Overall impression: This was a straightforward case correctly diagnosed by all but one lab with overall proficiency of 96%.

Analytical Details

Creatinine (n=19) 1.34 – 1.78, median 1.50

pH (n=12) 8-9, median 9

Spot tests

Nitrites n=10	Protein n= 10	
+ n=5 0 n=5	+ n=5 0 n=5	

Organic acid analysis (n= 18)

	n	points
Glycerol (Grossly) elevated	19	2

	n	Range	Median
Glycerol	9	307 – 130619	22500

Interpretation

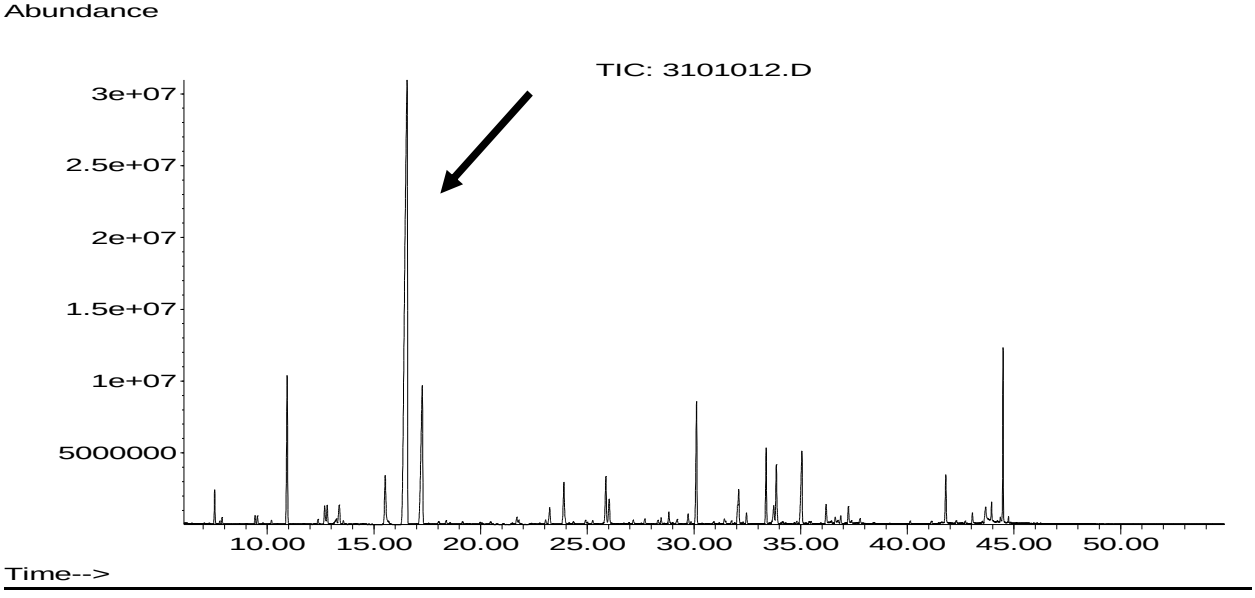
	n	Points
Glycerol kinase deficiency as a component of a Xp21 contiguous gene syndrome	15	2
Hyperglyceroluria due to glycerol kinase deficiency	3	2
confusing pattern of deviating diagnostic glycerol, not at a level characteristic for glycerol kinase deficiency	1	0

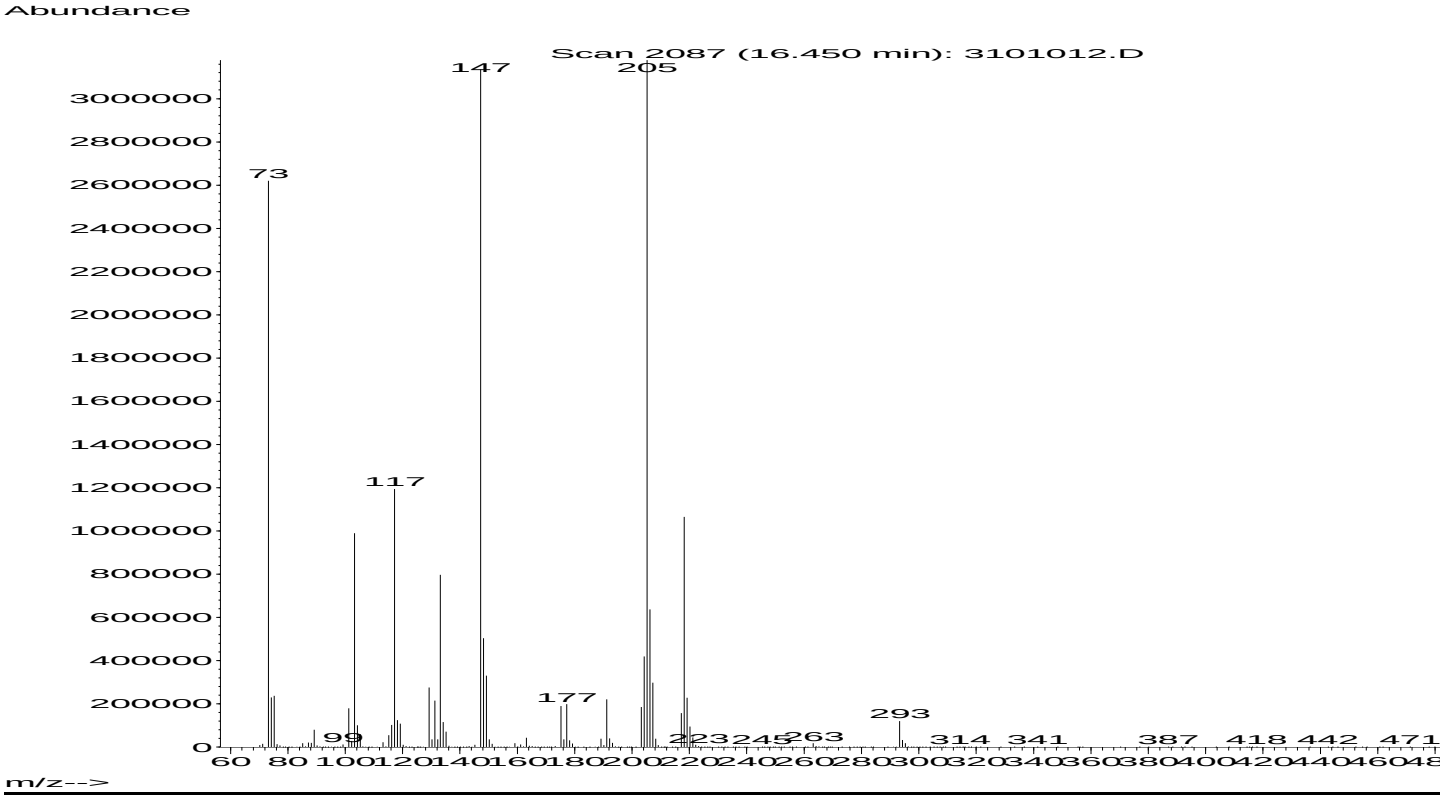
Recommendations for further tests

	n	points
Repeat urine for glycerol	2	
Plasma glycerol	6	1
Triglycerides	1	
GKD enzyme assay	7	1
Genetic analysis	16	1
Cortisol / endocrinologic investigatons	6	

Chromatograms etc.

GC/MS total ion chromatogram




Abundance
Scan 2087 (16.450 min): 3101012.D
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Sample E: Homocystinuria /Methylmalonic aciduria due to the cblC defect

Patient: The patient was admitted at 26 days of age to the emergency department following a history of sleepiness and feeding difficulties. He presented with severe breathing difficulties, recurrent cyanosis followed by respiratory arrest. He recovered after intubation but showed liver and kidney dysfunction, convulsions, muscular hypotonia, lethargy and pancytopenia. He responded rapidly to treatment. The urine was collected at one year of age whilst on treatment. The patient has the cblC defect confirmed by fibroblast studies of propionate fixation, methionine synthesis and cobalamin coenzyme uptake in fibroblasts and mutation analysis. The sample was provided by Prof. Matthias Baumgartner, Zürich, Switzerland.

Analytical performance*:* Organic acid analysis and amino acid analysis specifically for homocysteine were considered necessary. All labs reported elevated methylmalonic acid (one point) but only 5 reported homocysteine or homocysteine to be elevated (one point). Cystathionine which is a pointer to the correct diagnosis was reported by 6 labs. The analytical performance of this sample was 79.%.

Interpretative proficiency: a diagnosis of combined methylmalonic aciduria and homocystinuria due to either the cblC / cblD / cblF defect was considered correct. The interpretative proficiency for this sample was 74%.

Recommendations: plasma measurements of key metabolites and B12 or folate as well as appropriate fibroblast studies or mutation analysis were considered helpful.

Overall impression: This was a difficult sample with only low levels of homocysteine that were nevertheless detectable using specific methods. Also cystathionine is a helpful marker in remethylation disorders.

Analytical Details

Creatinine (n=19) 0.598 – 0.85, median 0.707

pH (n=12) 5-8, median 7

Spot tests

Sulphite		
Trace n=1 O n=2		

Amino acid analysis (n=19)

	n	mmol/mol Creat	points
Cystathionine elevated	6	59, 68, 71	1
Glycine elevated	5	477, 606, 637, 725, 784	
Homocysteine elevated	3	4.8, 21.2, 82.6*	1
Homocystine elevated	2	2, 8	1
Homocystine normal	2	0, 0	
Hydroxyproline elevated	1	100	
Methionine normal	1	13.4	
Proline elevated	1	115	
Sarcosine elevated	1	131	
Taurine elevated	4	646, 694, 715, 790	
		* IEC method!!	

Organic acid analysis (n= 19)

	n	mmol/mol Creat	points
Methylmalonic acid elevated	19	n=13 70 – 208 median 98	1
Methyl citrate elevated	8	7, 19, 20, 24, 34	
Ethylmalonic acid elevated	6	45.2, 45.2, 62, 92	

Interpretation

	n	Points
Methylmalonic and homocystinuria (cbl C or cblD or cblF)	10	2
Methylmalonic aciduria, probably B12-responsive. CblA and / or cblB	4	1
Mild MMA/B12 deficiency, mutase defect	2	1
Fazio-Londe disease	2	0
Non ketotic hyperglycinaemia / MMA	1	0

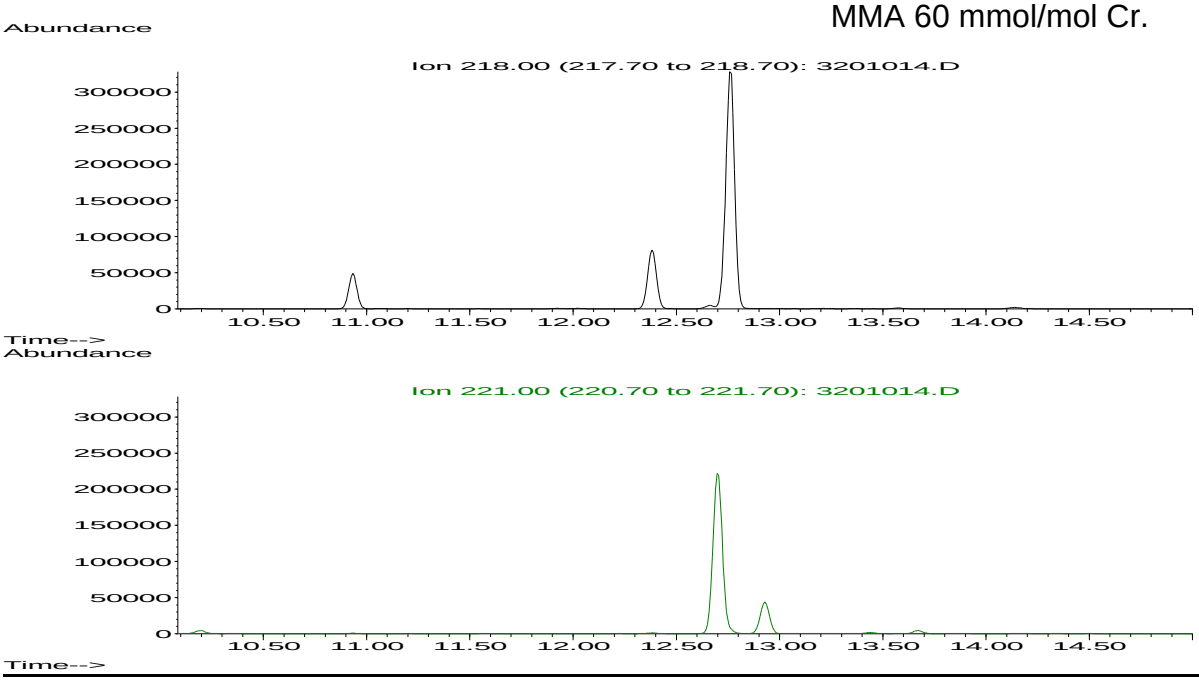
Recommendations for further tests

	n	points
Plasma MMA / HC / Methionine	15	1
Fibroblast / Complementation / mutations for MMA/HC defects	13	1
Mutation analysis cblA/B	1	1
Cbl and or folate levels	4	1
Acyl carnitines	3	
Repeat urine organic acids	1	
Exclude B12 deficiency	1	
Blood cell count	1	
B12 supplementation	1	

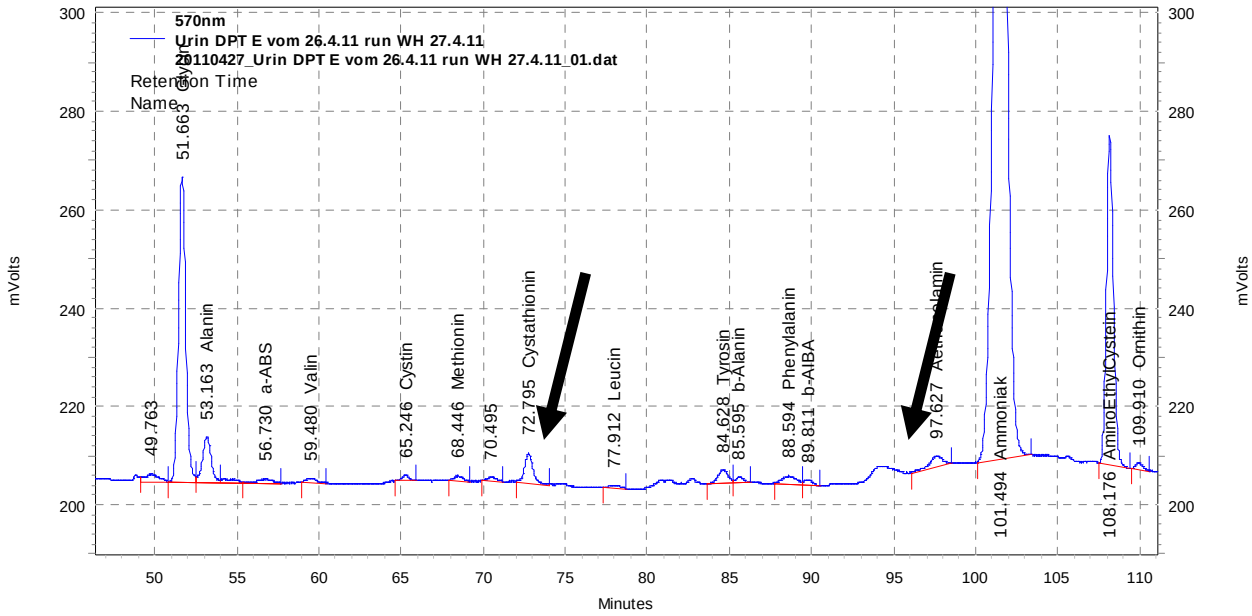
Chromatograms etc.

MMA stable isotope dilution, SIM

MMA 60 mmol/mol Cr.



Ion-exchange Chromatography



Total Homocysteine 8 mmol/mol Cr ref. <3)

Sample F: GAMT deficiency

Patient:
The patient is a 7-year old girl with developmental retardation, in particular speech delay. She has epilepsy, for which she is treated with valproic acid.

The patient is a 7-year old girl with developmental retardation, in particular speech delay. She has epilepsy, for which she is treated with valproic acid.
This sample was provided by Erasmus MC in Rotterdam.
Initial metabolic screening in urine revealed elevated guanidinoacetic acid: 640 mmol/mol (ref. values <129), and relatively low creatine: 15 mmol/mol (<754). Guanidino-acetic acid in plasma was 27.2 umol/L (ref values 0.5-3.6), which was strongly elevated. These results indicated guanidino-acetic acid methyltransferase (GAMT) deficiency. Enzyme and mutation analysis is pending. The patient is currently on treatment (creatine, ornithine, benzoate, low protein).

Analytical performance: Measurement of guanidine acetic acid and creatine was critical for this sample. Those labs that performed these assays found the relevant abnormality with overall analytical proficiency of 58%.

Interpretative proficiency: the correct diagnosis is guanidino-acetate methyltransferase deficiency. Interpretation is straightforward and was correct in all cases in which the relevant analysis was performed.

Recommendations: Measurements of GAA and creatine in plasma backed up by GAMT enzyme and / or mutation analysis was considered helpful.

Full details of this sample were presented at the joint ERNDIM workshop on August 30th at the annual SSIEM symposium in Geneva and are mounted on the ERNDIM website under Meetings & Reports / ERNDIM Workshop, SSIEM Geneva, 30.08.2011.

Analytical Details

Creatinine (n=19) 2-9 – 3-95, median 3-33

pH (n=12) 6 - 7, median 7

Spot tests

Amino acid analysis (n=20)

	n	mmol/mol Creat	points
Glycine elevated	15	600 – 1291 median 895	
Alanine	6	129 – 152 median 141	

Special assays (n= 19)

	n	mmol/mol Creat	points
Guanidino acetic acid elevated	10	n=8 220, 252, 333, 492, 500, 502, 600, 640	2
creatine low normal / normal	9	N=7 0.02, 14, 19, 22.5, 23, 23, 61	

Interpretation

	n	Points
Guanidino-acetate methyltransferase deficiency (GAMT)	11	2
Glycinuria secondary to valproate medication.	2	0
No diagnosis obtained	5	0
biotinidase deficiency	1	0

Recommendations for further tests

	n	points
GAA, Creatinine in plasma and / or CSF	5	1
GAMT enzyme assay	6	1
GAMT mutation analysis	9	1
Repeat urine GAA, Creatine	1	1
Determine GAA	1	1
Plasma (CSF) amino acids	5	0
No follow up	1	0
Biotinidase	1	0

8. Scores

Overall proficiency

Sample	Diagnosis	A (%)	I (%)	R (%)	total (%)
A	(Very) long chain acylCoA dehydrogenase deficiency	**57.5**	**42.5**	**80**	**56**
B	Non-Ketotic Hyperglycinaemia	**95**	**85**	**75**	**87**
C	Mucopolysaccharidosis type I	**82.5**	**87.5**	**100**	**87**
D	Glyceroluria associated with Xp21 contiguous gene deletion	**100**	**95**	**89**	**96**
E	Homocystinuria /Methylmalonic aciduria due to the cblC defect	**79**	**74**	**84**	**78**
F	GAMT deficiency	**58**	**58**	**63**	**59**

Total scores

	Survey 1			Survey 2			
Lab No	**A**	**B**	**C**	**D**	**E**	**F**	**Total excluding sample F***
1	**4**	**5**	**3**	**5**	**3**	**0**	20
2	**5**	**5**	**5**	**5**	**5**	**0**	25
3	**4**	**5**	**5**	**5**	**1**	**5**	20
4	**4**	**5**	**5**	**5**	**4**	**5**	23
5	**5**	**5**	**5**	**5**	**3**	**5**	23
6	**2**	**5**	**5**	**3**	**5**	**5**	20
7	**1**	**5**	**5**	**5**	**3**	**0**	19
8	**3**	**4**	**5**	**5**	**5**	**5**	22
9	**3**	**5**	**4**	**5**	**4**	**5**	21
10	**1**	**5**	**5**	**5**	**5**	**5**	21
11	**2**	**5**	**3**	**5**	**4**	**1**	19
12	**0**	**0**	**5**	**4**	**2**	**5**	11
13	**4**	**5**	**5**	**5**	**1**	**5**	20
14	**3**	**5**	**5**	**5**	**5**	**0**	23
15	**5**	**2**	**4**	**4**	**5**	**0**	20
16	**1**	**5**	**5**	**5**	**5**	**5**	21
17	**1**	**5**	**3**	**-**	**-**	**-**	9
18	**0**	**4**	**5**	**5**	**5**	**0**	19
19	**5**	**2**	**1**	**5**	**5**	**0**	18
20	**-**	**-**	**-**	**-**	**-**	**-**	0
21	**3**	**5**	**5**	**5**	**4**	**5**	22

*This year the scores proposed by the scheme organiser 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 **18/30**. However sample F was not included in scores for performance since the measurement of guanidine-acetate is not one of the required methods for participation. Therefore the level for satisfactory performance was adjusted to **15/25**. Labs failing to reach this mark will receive a performance advice letter.
Appeals against allocated scores should be sent to the scientific advisor within two weeks of receipt of this report.

Detailed Scores: A,B,C

Lab no	Sample A (Very) long chain acylCoA dehydrogenase deficiency				Sample B Non-Ketotic Hyperglycinaemia				Sample C Mucopolysaccharidosis type I				
	A	I	R	Total	A*	I	R	Total	A	I	R	Total	Total
1	1	2	1	4	2	2	1	5	1	1	1	3	12
2	2	2	1	5	2	2	1	5	2	2	1	5	15
3	2	1	1	4	2	2	1	5	2	2	1	5	14
4	2	1	1	4	2	2	1	5	2	2	1	5	14
5	2	2	1	5	2	2	1	5	2	2	1	5	15
6	1	0	1	2	2	2	1	5	2	2	1	5	12
7	0	0	1	1	2	2	1	5	2	2	1	5	11
8	1	1	1	3	2	2	0	4	2	2	1	5	12
9	1	1	1	3	2	2	1	5	1	2	1	4	12
10	0	0	1	1	2	2	1	5	2	2	1	5	11
11	1	0	1	2	2	2	1	5	1	1	1	3	10
12	0	0	0	0	0	0	0	0	2	2	1	5	5
13	2	1	1	4	2	2	1	5	2	2	1	5	13
14	2	1	0	3	2	2	1	5	2	2	1	5	13
15	2	2	1	5	2	0	0	2	1	2	1	4	11
16	0	0	1	1	2	2	1	5	2	2	1	5	11
17	1	0	0	1	2	2	1	5	1	1	1	3	9
18	0	0	0	0	2	2	0	4	2	2	1	5	9
19	2	2	1	5	2	0	0	2	0	0	1	1	8
20	-	-	-	-	-	-	-	-	-	-	-	-	-
21	1	1	1	3	2	2	1	5	2	2	1	5	13
ratio	23/40	17/40	16/20	56/100	38/40	34/40	15/20	87/100	33/40	35/40	20/20	87/100	
%	57.5	42.5	80	56	95	85	75	87	82.5	87.5	100	87	

Detailed Scores: D,E,F

Lab no	Sample D Glyceroluria associated with Xp21 contiguous gene deletion				Sample E Homocystinuria /Methylmalonic aciduria due to the cblC defect				Sample F GAMT deficiency				
	A	I	R	Total	A*	I	R	Total	A	I	R	Total	Total
1	2	2	1	5	1	1	1	3	0	0	0	0	8
2	2	2	1	5	2	2	1	5	0	0	0	0	10
3	2	2	1	5	1	0	0	1	2	2	1	5	11
4	2	2	1	5	1	2	1	4	2	2	1	5	14
5	2	2	1	5	1	1	1	3	2	2	1	5	13
6	2	0	1	3	2	2	1	5	2	2	1	5	13
7	2	2	1	5	1	1	1	3	0	0	0	0	8
8	2	2	1	5	2	2	1	5	2	2	1	5	15
9	2	2	1	5	1	2	1	4	2	2	1	5	14
10	2	2	1	5	2	2	1	5	2	2	1	5	15
11	2	2	1	5	2	1	1	4	0	0	1	1	10
12	2	2	0	4	1	1	0	2	2	2	1	5	11
13	2	2	1	5	1	0	0	1	2	2	1	5	11
14	2	2	1	5	2*	2	1	5	0	0	0	0	10
15	2	2	0	4	2	2	1	5	0	0	0	0	9
16	2	2	1	5	2	2	1	5	2	2	1	5	15
17	-	-	-	-	-	-	-	-	-	-	-	-	0
18	2	2	1	5	2	2	1	5	0	0	0	0	10
19	2	2	1	5	2	2	1	5	0	0	0	0	10
20	-	-	-	-	-	-	-	-	-	-	-	-	0
21	2	2	1	5	2	1	1	4	2	2	1	5	14
ratio	38/38	36/38	17/19	91/95	30/38	28/38	16/19	74/95	22/38	22/38	12/19	56/95	
%	100	95	89	96	79	74	84	78	58	58	63	59	

* This lab found a very high level of homocystine by ion-exchange which makes the correctness of the analytical result doubtful.

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 was set at the SAB meeting in November.

10. Annual meeting

The annual meeting of participants of the 5 DPT centres took place during the SSIEM symposium in Geneva on Tuesday, August 30, 2011 at 9.00

11. Changes planned for 2012

No changes are planned for this year as we will continue with submission of results online to our website. The samples for the Basel scheme will again be distributed by the CSCQ but we will remain responsible for the scientific and evaluation aspects of the scheme.

12. Tentative schedule and fee in 2012

We hope to follow a similar time schedule to that used for 2011. Exact details will follow.

Sample distribution	End April 2012
Start of analysis of Survey 2011/1	May 07, 2012, Monday
Survey 2011/1 - Results submission	May 28, 2012, Monday
Survey 201/1 - Reports	June 11, 2012, Monday
Start of analysis of Survey 2011/2	June 18, 2012, Monday
Survey 2011/2 – Results submission	July 09, 2012, Monday
Survey 2011/2 - Reports	August 03, 2012, Friday
Annual meeting of participants	Sept 04, 2012, in Birmingham at SSIEM
Annual Report 2011	December

The fee for 2012 has been increased slightly and has been set at €334.

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, February 2012.

Brian Fowler
Scientific advisor

Marianne Zaugg
Scheme organiser