0000000287876

Name:		**Age/Gender:**	34 years 9 months 0 days /Male
Referred By:	N.A	**Client Name:**	N.A
Collection Date:	13-05-2017 00:00:00	**Report Release Time:**	23-05-2017 19:34:24

Result Summary

Very high excretion of sugars were seen in the provided urine sample which indicated severely impaired glucose tolerance and poor glycemic control or parenteral glucose administration. Increased excretion of 4-hydroxyphenylacetate (4HPA) may be due to possibility of gastrointestinal malabsorption or diabetes related gut dysmotility or alteration of the composition or stability of normal microbial flora.

It is recommended to use Prebiotics, Probiotics for enhancement of beneficial commensal organisms. You may use of digestive enzymes and prophylactic and corrective B-complex vitamin supplementations. It is advised to send the repeat urine sample withholding intravenous fluids (if any) and controlling the current glycosuria in the context of diagnosis, treatment and prognosis of neurodevelopmental disorders.

Please correlate these findings through your family doctor or nutritionist and get them correlated with supplementary diet plan, drug treatment, previous metabolic profile and other clinical and diagnostic findings.

Sr.No	Investigation	Observed Value	Reference Range	Risk Graph

Metabolite Results

Urinary Markers

1	HVA	4.65	0.08 - 5.17	
2	4HPA	18.65	0.13 - 8.66	
3	VMA	2.26	0.03 - 2.96	
4	Citrate	2.45	0.0 - 4.12	
5	TG1	0	0.0 - 0.1	
6	pseudouridine	4.26	0.0 - 7.77	
7	4HPL	0.26	0.0 - 0.1	
8	Gly1	0.84	0.12 - 7.72	

Sr.No	Investigation	Observed Value	Reference Range	Risk Graph

Metabolite Results

Urinary Markers

9	2KG	0.03	0.0 - 0.1	
10	Phosphate	12.28	2.62 - 18.51	
11	Indole3AA	0	0.0 - 0.1	
12	Cresol	0	0.0 - 5.56	
13	tryptophan1	0.02	0.0 - 0.1	
14	hippurate1	5.61	0.0 - 13.81	
15	Cisaconate	2.49	0.0 - 2.95	
16	5HM2F	2.24	0.0 - 4.36	
17	5HindoleAA	0.01	0.0 - 0.1	
18	4HBA	2.45	0.0 - 3.63	
19	Indollactic aicd	0	0.0 - 0.1	
20	Arabitol	19.65	0.38 - 8.52	
21	tartarate	0	0.0 - 0.1	
22	Succinate	1.49	0.03 - 2.68	
23	Tyr1	6.25	0.0 - 15.4	
24	kynurate	2.48	0.0 - 4.48	
25	Benzonate	1.57	0.0 - 3.78	
26	3HP3HP	3.65	0.0 - 4.35	

End Of Report

CRM No :287876
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Patient Name :
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Understanding the report

In the technical report section, you will see the graphic representation of all metabolic markers in the scope of the test conducted on your sample(s) and interpreted by our metabolic experts.

Definitions

Metabolites - Metabolites in your blood/urine samples are the Markers of Metabolism and act as the ' health indicators'. They characterize your state of metabolism and help make inferences in case of non-specific health conditions which can be an outcome of problems with your metabolism. Tracking the levels of these metabolites is important to ensure that early signals of diabetes related complications can be picked up.

ControlValues - The 'Normal Limit' within which the value of a metabolic marker should ideally fall.

Observed *(your)* Value - The 'Actual Value' of a Metabolic Marker in your sample.

Understanding the Risk-Bar

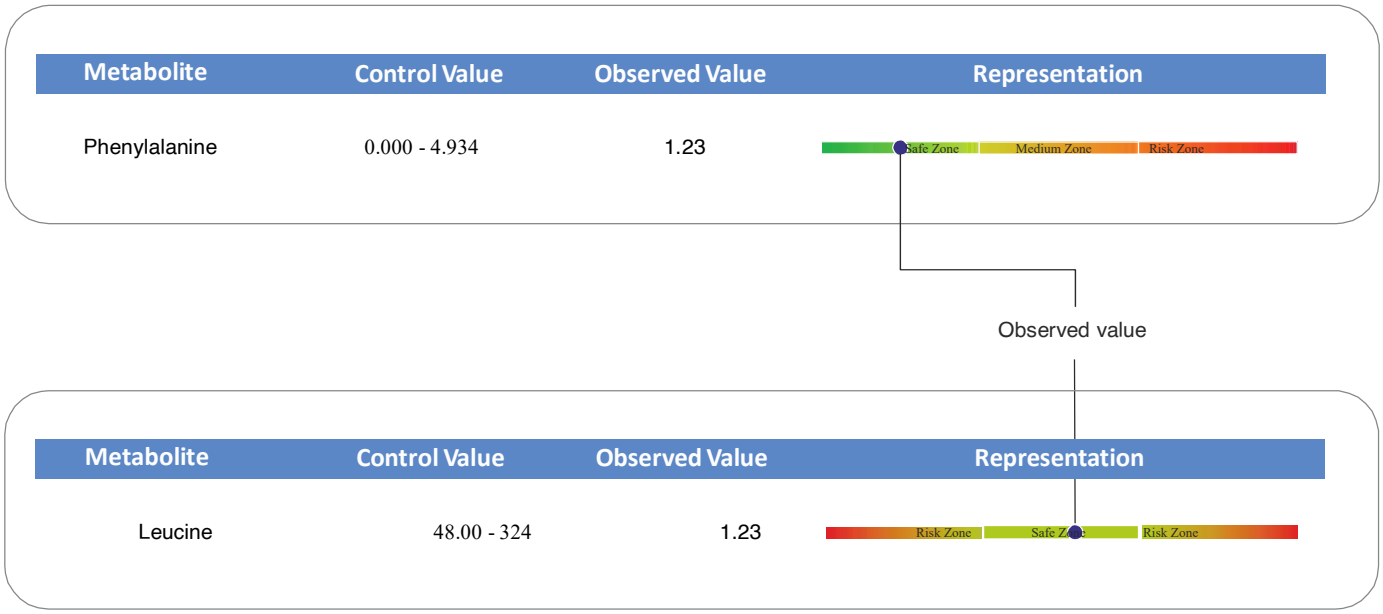
RiskBar - The horizontal bar as a pictorial representation of the observed values of the metabolic markers against the control values.

Safe Zone *(Green Color)*- If the value of markers measured in your sample fall in this region *(signified by the green zone)*, you can relax and maintain the lifestyle you have.

Risk Zone *(Red Color)* - If the value of marker(s) measured in your sample falls in this region *(signified by the red zone)*, it will be a matter of concern. You must consult your family physician or a metabolism expert.

Medium Zone *(color transition zone)* - If the value of a marker measured in your sample falls in this region *(signified by the color transition from green to red)*, you may need to bring in changes in your lifestyle, diet or medication, depending on the particular case. Any modifications, however, have to be routed through a medical practitioner.

Pointer - The 'blue dot' on the risk bar. It represents the actual value of a particular metabolic marker found in your sample.



A. Autism

ASD are complex neurodevelopmental disabilities with onset before 36 months and characterized by impairments in reciprocal social interactions, verbal and non-verbal communication skills, interests and activities. Individuals with autism vary widely in abilities, intelligence, and behaviors.

B.The analysis

ASD are known to be influenced by genetic and environmental factors. A range of gastrointestinal disorders have been linked in pathophysiology of ASD and studies have found that the condition is associated with abnormal gut flora.
There is also the possibility of previously unrecognized etiologic connections between dysbiosis and childhood developmental problems. Individuals with ASD if exposed to repeated courses of multiple antibiotic therapies may contribute to the complex relationships between gastrointestinal dysbiosis and ASD by altering the composition or stability of their normal microbial flora.

It has been observed that such altered gut microorganisms produce toxic metabolites and they are absorbed in the portal circulation and accumulate in the neural tissue. These metabolites interfere the neurotransmitter metabolism and these typical metabolites excreted in large amount in urine in children. Moreover, the effect impact of these metabolites manifest in autistic symptoms. Therefore urine metabolic analysis is used for diagnosis, treatment and prognosis of babies with autistic symptoms.

This analysis uses Gas chromatography/mass spectrometry (GC/MS) technology with subsequent multivariate statistical data interpretation for differential metabolic pathway profiling, disease diagnosis and prognosis. The relevant metabolites in ASD taken under consideration are intermediates of amino acid metabolism, antioxidants including nicotinic acid metabolism, sugar metabolism, mitochondrial metabolism, neurotransmitter metabolism.

B.1 AMINO ACID METABOLISM

Evidence suggests that diet and protein assimilation play important roles in autism. Amino acids are the chemical building blocks of key neurotransmitters that act at specific sites of the brain to influence mood and behavior. For this reason, the proper balance of these nutrients is essential for healthy emotional and cognitive development in children. The Amino Acid profile provides a comprehensive picture of the amino acid neurotransmitter imbalances.

B.2DIGESTIVE FUNCTION

Autistic children often exhibit chronic digestive problems that may be linked to changes in mood and behaviour. Gut enzyme deficiencies, malabsorption, and yeast overgrowth are common findings in autism. Abnormal gut function gives rise to elevation of typical metabolites in urine. Such urine profile associated with abnormal GI function provides an assessment of microbial balance (including yeast and bacterial growth), digestive function, and absorption. This test helps to develop individualized treatment strategies to improve function and reduce gastrointestinal symptoms.

B.3INTESTINAL PERMEABILITY

The integrity of the intestinal lining plays a critical role in absorbing nutrients and prevent toxins, allergens and other potentially harmful molecules from penetrating into the systemic circulation. Studies have found significantly increased gut floral permeability in autistic children. This serves as a common link between autism and autoimmune dysfunction, food allergies, gastrointestinal imbalances, bacterial and fungal overgrowth, and nutrient deficiencies. This Intestinal Permeability Assessment test measures urinary clearance of non-metabolized sugar molecules assess intestinal integrity and absorptive function.

B.4ANTIOXIDANTS

Children with ASD may have different redox abnormalities which may arise from various sources. The level of antioxidant excreted in urine was found to be significantly lower than normal in autistic children. The urine assessment calculates the urinary oxidative stress marker which is helpful to judge severity and prognosis of ASD patients.

References

1. Whitman WB, Coleman DC, Wiebe WJ. Prokaryotes: the unseen majority. Proceedings of the National Academy of Sciences of the United States of America. 1998;95:6578–6583. [PMC free article] [PubMed]
2. Gill SR, et al. Metagenomic analysis of the human distal gut microbiome. Science. 2006;312:1355–1359. [PMC free article] [PubMed]
3. Zhu B, Wang X, Li L. Human gut microbiome: the second genome of human body. Protein & cell. 2010;1:718–725. [PMC free article] [PubMed]
4. Clemente JC, Ursell LK, Parfrey LW, Knight R. The impact of the gut microbiota on human health: an integrative view. Cell. 2012;148:1258–1270. [PMC free article] [PubMed]
5. Peterson J, et al. The NIH Human Microbiome Project. Genome research. 2009;19:2317–2323. [PMC free article] [PubMed]
6. O'Hara AM, Shanahan F. The gut flora as a forgotten organ. EMBO reports. 2006;7:688–693. [PMC free article] [PubMed]
**7. A framework for human microbiome research. Nature. 2012;486:215–221. One of the initial publications from the Human Microbiome Project, showing the diversity of data contained in the microbiome from different body sites. [PMC free article] [PubMed]
8. Cummings JH, Macfarlane GT. Role of intestinal bacteria in nutrient metabolism. JPEN Journal of parenteral and enteral nutrition. 1997;21:357–365. [PubMed]
9. Staley JT, Konopka A. Measurement of in situ activities of nonphotosynthetic microorganisms in aquatic and terrestrial habitats. Annual review of microbiology. 1985;39:321–346. [PubMed]
10. Wade W. Unculturable bacteria--the uncharacterized organisms that cause oral infections. Journal of the Royal Society of Medicine. 2002;95:81–83. [PMC free article] [PubMed]
11. Duncan SH, Louis P, Flint HJ. Cultivable bacterial diversity from the human colon. Letters in applied microbiology. 2007;44:343–350. [PubMed]
12. Eckburg PB, et al. Diversity of the human intestinal microbial flora. Science. 2005;308:1635–1638. [PMC free article] [PubMed]
13. Shendure J, Ji H. Next-generation DNA sequencing. Nature biotechnology. 2008;26:1135–1145. [PubMed]
14. Pace NR. A molecular view of microbial diversity and the biosphere. Science. 1997;276:734–740. [PubMed]
15. Venter JC, et al. Environmental genome shotgun sequencing of the Sargasso Sea. Science. 2004;304:66–74. [PubMed]
16. Momozawa Y, Deffontaine V, Louis E, Medrano JF. Characterization of bacteria in biopsies of colon and stools by high throughput sequencing of the V2 region of bacterial 16S rRNA gene in human. PloS one. 2011;6:e16952. [PMC free article] [PubMed]
**17. Arumugam M, et al. Enterotypes of the human gut microbiome. Nature. 2011;473:174–180. The first paper with data to support the idea of characteristic gut microbiomes, classified as "enterotypes.". [PMC free article] [PubMed]
18. Wu GD, et al. Linking long-term dietary patterns with gut microbial enterotypes. Science. 2011;334:105–108. [PMC free article] [PubMed]
*19. Koenig JE, et al. Succession of microbial consortia in the developing infant gut microbiome. Proceedings of the National Academy of Sciences of the United States of America. 2011;108(Suppl 1):4578–4585. This study measured the developing infant gut microbiome by sampling stool from a single healthy infant 60 times, from birth to 2.5 years of age. [PMC free article] [PubMed]
20. Breitbart M, et al. Viral diversity and dynamics in an infant gut. Research in microbiology. 2008;159:367–373. [PubMed]
21. Dominguez-Bello MG, et al. Delivery mode shapes the acquisition and structure of the initial microbiota across multiple body habitats in newborns. Proceedings of the National Academy of Sciences of the United States of America. 2010;107:11971–11975. [PMC free article] [PubMed]
*22. Schwartz S, et al. A metagenomic study of diet-dependent interaction between gut microbiota and host in infants reveals differences in immune response. Genome biology. 2012;13:r32. This study compared both the developing gut microbiome and gene expression in 6 breastfed infants and 6 formula fed infants, finding systematic differences in the microbiome and in expression of genes relevant to immune system fucntion. [PMC free article] [PubMed]
23. Qin J, et al. A human gut microbial gene catalogue established by metagenomic sequencing. Nature. 2010;464:59–65. [PMC free article] [PubMed]
24. Bergman EN. Energy contributions of volatile fatty acids from the gastrointestinal tract in various species. Physiological reviews. 1990;70:567–590. [PubMed]
25. Walter J, Ley R. The human gut microbiome: ecology and recent evolutionary changes. Annual review of microbiology. 2011;65:411–429. [PubMed]
26. Chow J, Lee SM, Shen Y, Khosravi A, Mazmanian SK. Host-bacterial symbiosis in health and disease. Advances in immunology. 2010;107:243–274. [PMC free article]
27. Le Huerou-Luron I, Blat S, Boudry G. Breast- v. formula-feeding: impacts on the digestive tract and immediate and long-term health effects. Nutrition research reviews. 2010;23:23–36. [PubMed]

We welcome all questions and concerns. The questions pertaining to your analysis shall be answered by our experts (medical / nutritional).

You may post your queries on - genoroot@preventine.com. Please mention your Name, Date of Birth and the Customer ID in the query.

Customer satisfaction is our core goal. It is important for us to learn about what our customers think about our service and how we can improve it. If you have any suggestion or complaint, whatsoever, we request you to contact us and report it on:

Email: info@preventine.com
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