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WELLNESS STARTS WITH YOU

From:
Neurazon INC.
Precision Health Analysis Department - Children with Autism and Developmental Disorders
Quebec, Canada

Case no: 5101413163
First Name: Ahmad
Last Name: Odeh
DOB: 12.06.2021
Sex: Male

Test requested: Precision Health Analysis®

Results:

True Autism Spectrum Disorder Variants: NEGATIVE
Acquired Autism Spectrum Disorder Variants: POSITIVE
Attention-Deficit/Hyperactivity Disorder Variants: NEGATIVE
Information Processing and Brain Development Variants: NEGATIVE
White Matter Delays Variants: POSITIVE
Methylation Variants: NEGATIVE
Metabolic and Mitochondrial Function Variants: NEGATIVE
Neurotransmitters, Synaptic Health, and Behavior Variants: POSITIVE
Immune System Variants: NEGATIVE
Potential Digestive System Variants: NEGATIVE
Epigenetic Regulation Variants: NEGATIVE
Potential Uncontrolled Electrical Activity: NEGATIVE

Recommended Interpretation:

- Please note any clinically relevant variants detected that are associated with the described phenotype.
- Review all variants listed in the attached report, if applicable.
- We recommend reevaluating the sequence dataset every 12 months or when there are changes in the phenotype.
- Reassessment by a multidisciplinary team is advised every 3 months to monitor any observed phenotypic changes using artificial intelligence.
- A tailored intervention plan should be developed and supervised by a multidisciplinary team.

ADDITIONAL INFORMATION

This assessment has been developed and validated through the AI-powered bioinformatics platform of NEURAZON, which integrates advanced data analysis with input from partner laboratories worldwide. All results are carefully reviewed, interpreted, and compiled by NEURAZON's team of scientific experts. For full details, please refer to the attached report.

> Contact Details

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Recommended Homeopathy Plan

Step: 1st step
Name: Ahmad Odeh
DOB: 12.06.2021

Please refer to external report

ADDITIONAL INFORMATION

Recommendations: The provided guidance includes safe, non-drug, natural treatments intended to support the child's development. These recommendations are not a replacement for professional rehabilitation and training sessions and should always be followed under the supervision of a qualified specialist. Progress and improvement depend on the individual child, the underlying causes, and their response to the interventions, with recommendations adjusted based on regular evaluations every three months. If any signs of an allergic reaction occur, discontinue the treatment immediately and consult with a healthcare provider to modify the treatment plan accordingly.

Reevaluation: A reevaluation is necessary after the specified duration to reassess development using the AI model or as recommended by the professional team. This test doesn't replace a full clinical assessment by a physician.

Next Steps: Begin the second phase of the protocol after the time-frame indicated in the treatment plan.

This assessment has been developed and validated through the AI-powered bio-informatics platform of NEURAZON, which integrates advanced data analysis with input from partner laboratories worldwide. All results are carefully reviewed, interpreted, and compiled by NEURAZON's team of scientific experts. For full details, please refer to the attached report. Neurazon is a bio-informatics platform that analyzes data, not a laboratory; however, it partners with leading international laboratories to ensure the highest level of accuracy.

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Order no: 5101413163
Report Type: Final report

Case no: 5101413163
First Name: AHMAD
Last Name: ODEH
DOB: 12.06.2021
Sex: Male

Requested Test: WES (Whole Exome Sequencing)
Clinical Information/Indication: The analysis was performed according to the demand of referral center to identify variants in relevant genes associated with neurological disorders.
Result:

Main Findings:
A variant of unknown significance (VUS); c.514_515del / p.Ser172* in the BAIAP2L1 gene was identified.
A variant of unknown significance (VUS); c.1681G>C / p.Asp561His in the TBC1D23 gene was identified.
A variant of unknown significance (VUS); c.778G>C / p.Val260Leu in the MLC1 gene was identified.
A variant of unknown significance (VUS); c.2747C>A / p.Thr916Lys in the GIGYF1 gene was identified.

Gene (Transcript)	Location	Nucleotide (protein) dbSNP	Zygosity	Variant Classification	Disease (OMIM#, Inheritance)
BAIAP2L1 NM_018842.5	Exon 7	c.514_515del p.Ser172*	Heterozygous	Variant of Unknown Significance (VUS)	Autism Related Disorder [1]
TBC1D23 NM_001199198.3	Exon 16	c.1681G>C p.Asp561His	Homozygous	Variant of Unknown Significance (VUS)	Pontocerebellar Hypoplasia Type 11 OMIM: 617695 Autosomal Recessive
MLC1 NM_015166.4	Exon 10	c.778G>C p.Val260Leu rs143061714	Homozygous	Variant of Unknown Significance (VUS)	Megalencephalic Leukoencephalopathy with Subcortical Cysts Type 1 OMIM: 604004 Autosomal Recessive
GIGYF1 NM_001375765.1	Exon 24	c.2747C>A p.Thr916Lys	Heterozygous	Variant of Unknown Significance (VUS)	Autism Related Disorder[2]

Details About Gene and Variants:
BAIAP2L1: This gene encodes a member of the IMD (IRSp53/MIM homology domain) family. Members of this family can be subdivided in two groups, the IRSp53-like and MIM-like, based on the presence or absence of the SH3 (Src homology 3) domain. The protein encoded by this gene contains a conserved IMD, also known as F-actin bundling domain, at the N-terminus, and a canonical SH3 domain near the C-terminus, so it belongs to the IRSp53-like group. This protein is the substrate for insulin receptor tyrosine kinase and binds to the small GTPase Rac. It is involved in signal transduction pathways that link deformation of the plasma membrane and remodeling of the actin cytoskeleton. It also promotes actin assembly and membrane protrusions when overexpressed in mammalian cells, and is essential to the formation of a potent actin assembly complex during EHEC (Enterohemorrhagic Escherichia coli) pedestal formation. [provided by RefSeq, Oct 2009]

The BAIAP2L1 variant c.514_515del / p.Ser172* was detected in heterozygous state. It is classified as Variant of Unknown Significance (VUS) according to the recommendations of ACMG. This variant is associated with Autism Related Disorder[1].

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Last Name: ODEH
DOB: 12.06.2021
Sex: Male

[1] Iossifov, Ivan et al. "The contribution of de novo coding mutations to autism spectrum disorder." *Nature* vol. 515,7526 (2014): 216-21. doi:10.1038/nature13908.

TBC1D23: Involved in brain development; retrograde transport, endosome to Golgi; and vesicle tethering to Golgi. Located in WASH complex; cytoplasmic vesicle; and trans-Golgi network. Implicated in pontocerebellar hypoplasia type 11. [provided by Alliance of Genome Resources, Jul 2025]

The TBC1D23 variant c.1681G>C / p.Asp561His was detected in homozygous state. It is classified as Variant of Unknown Significance (VUS) according to the recommendations of ACMG. This variant is associated with autosomal recessive Pontocerebellar Hypoplasia Type 11 (OMIM: 617695).

MLC1: The function of this gene product is unknown; however, homology to other proteins suggests that it may be an integral membrane transporter. Mutations in this gene have been associated with megalencephalic leukoencephalopathy with subcortical cysts, an autosomal recessive neurological disorder. Alternatively spliced transcript variants encoding different isoforms have been identified. [provided by RefSeq, Jul 2008]

The MLC1 variant c.778G>C / p.Val260Leu was detected in homozygous state. It is classified as Variant of Unknown Significance (VUS) according to the recommendations of ACMG. This variant is associated with autosomal recessive Megalencephalic Leukoencephalopathy with Subcortical Cysts Type 1 (OMIM: 604004).

GIGYF1: This gene encodes a member of the gyf family of adaptor proteins. The encoded protein contains a gyf protein interaction domain. It binds growth factor receptor bound 10, another adaptor protein that binds activated insulin-like growth factor 1 and insulin receptors and regulates receptor signaling. [provided by RefSeq, Apr 2017]

The GIGYF1 variant c.2747C>A p.Thr916Lys was detected in heterozygous state. It is classified as Variant of Unknown Significance (VUS) according to the recommendations of ACMG. This variant is associated with Autism Related Disorder[2].

[2]Two de novo likely gene-disruptive/protein-truncating variants in the GIGYF1 gene (both frameshift) were identified in ASD probands from the Simons Simplex Collection (PMID 25363768). Additional de novo likely gene-disruptive/protein-truncating variants in GIGYF1 were identified in ASD probands from the SPARK cohort (Feliciano et al., 2019) and the Autism Sequencing Consortium (Satterstrom et al., 2020); six protein-truncating variants in this gene were also observed in case samples from the Danish iPSYCH study in Satterstrom et al., 2020. Furthermore, independent TADA analyses in Feliciano et al., 2019 and Satterstrom et al., 2020 identified GIGYF1 as an ASD candidate gene with a false discovery rate (FDR) 0.01.

Recommendations:
- It is recommended to evaluate this patient's result together with clinical and laboratory findings.
- Genetic screening and clinical evaluation are recommended for family members.
- Genetic counseling is recommended.

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Report Type: Final report

Variant Quality	99%

Method Limitations:
Polymorphisms in primary binding and regions, CNV duplications and somatic microsatellite variations, tissue mosaic, high GC nucleotide content can lead to false positive / negative results. False positive findings may occur due to large deletion / point mutation combined heterozygosity. Variants in transcripts other than canonical transcripts cannot be eliminated, and differences may occur between exon numbers and mutation positions depending on the transcript type sequenced. This method does not show heterozygous deletions and duplications in rare exons and nucleotide changes in other regions of the gene.

Variants with the allele fraction below 30% for regions with a sensitivity of this test of 50X read count are not reported. Additionally, due to technology limitations, some regions may be either not covered or poorly covered. Variables in these regions cannot be reliably detected. Areas with extremely low readings are considered artifacts as a result of validation studies, and they were not taken into account during the analysis. Copy number changes, inversions, translocations and repeat sequence increases cannot be detected by the NGS method.

The genetic results are interpreted in the context of the provided clinical findings, family history, and other laboratory data. Only variants in genes potentially related to the proband's medical condition are reported. Misinterpretation of results may occur if the provided information is inaccurate and/or incomplete. If the obtained genetic results do not concur with the clinical findings, additional testing should be considered.

Reference:
[1] Richards et al.Standarts and guidelines for the interpretation of sequence variants 17(5):405-424. doi:10.1038/gim.2015.30 GENETICS in MEDICINE,2015.

This test was analyzed by Neurazon, Canada.

Digitally Approved by:
Dr. Carmel Katz
Lead Scientist and Geneticist
Lead Bioinformatics Specialist
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