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DETECTION AND PHYLOGENETIC ANALYSIS OF *CLOSTRIDIUM VENTRICULI* IN AUTISTIC CHILDREN

A.H. Hasan, H.H. Lazim

Ibn Sina University of Medical and Pharmaceutical Sciences, Baghdad, Iraq

Abstract. *Background*. Autism spectrum disorder (ASD) is characterized by repetitive behaviors. There is evidence that gut flora imbalance may cause GI difficulties in autistic people. Gastrointestinal (GI) issues are associated with *Clostridium ventriculi* (*C. ventriculi*). The aim of this study was to use 16S rRNA gene sequencing to identify and genetically describe *Clostridium ventriculi* in fecal samples from children with autism. *Materials and methods.* A case-control study was done on fecal samples collected from 50 children diagnosed with autism. Also, samples were taken from 50 children who were not autistic as a control group. Using the FavorPrep Genomic DNA Mini Kit, DNA was extracted. PCR was used to amplify the 16S rRNA gene using the universal primers 27F and 1492R. After the PCR products were sequenced, BLAST and BioEdit tools were used to check the sequences for homology. The MEGA program was used for phylogenetic analysis. *Results.* Based on PCR results, 10% (5/50) of the 50 samples of autistic children that were examined proved positive for *C. ventriculi*, and all control group were negative for this bacteria. Genetic polymorphisms were indicated by specific nucleotide transitions and transversions that were discovered by sequencing. The Iraqi isolates and global samples exhibited a high level of genetic similarity (99%) according to phylogenetic analysis, indicating a recent common ancestor and potential clonal expansion. *Conclusion*. The discovery of *C. ventriculi* in autistic children raises the possibility of a connection between this bacteria and gastrointestinal problems linked to ASD.

Key words: *Clostridium ventriculi, autism spectrum disorder, phylogenetic analysis, 16S rRNA gene.*

ВЫЯВЛЕНИЕ И ФИЛОГЕНЕТИЧЕСКИЙ АНАЛИЗ *CLOSTRIDIUM VENTRICULI* У ДЕТЕЙ С АУТИЗМОМ

Хасан А.Х., Лазим Х.Х.

Университет медицинских и фармацевтических наук Ибн Сины, Багдад, Ирак

Введение. Расстройство аутистического спектра (РАС) характеризуется повторяющимся поведением. Существуют доказательства того, что дисбаланс кишечной флоры может вызывать проблемы с желудочно-кишечным трактом (ЖКТ) у людей с аутизмом. Проблемы с ЖКТ связаны с *Clostridium ventriculi* (*C. ventriculi*). Целью данного исследования было использование секвенирования гена 16S рРНК для идентификации и генетического описания *Clostridium ventriculi* в образцах кала детей с аутизмом. *Материалы и методы.* Исследование случай-контроль было проведено на образцах кала, собранных от 50 детей с диагнозом аутизм. Кроме того, образцы были взяты у 50 детей, не страдающих аутизмом (контрольная группа). Образцы ДНК выделяли с применением набора FavorPrep Genomic DNA Mini Kit. ПЦР использовалась для амплификации

Адрес для переписки:
Хусам Хусейн Лазим
Университет медицинских и фармацевтических наук Ибн Сины, Багдад, Ирак.
Тел.: 009647801612798. E-mail: hlazim@ibnsina.edu.iq

Contacts:
Husam Hussein Lazim
Ibn Sina University of Medical and Pharmaceutical Sciences, Baghdad, Iraq.
Phone: 009647801612798. E-mail: hlazim@ibnsina.edu.iq

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гена 16S рРНК с использованием универсальных праймеров 27F и 1492R. После секвенирования продуктов ПЦР были использованы базы данных BLAST и BioEdit для проверки последовательностей на гомологию. Программа MEGA использовалась для филогенетического анализа. *Результаты.* На основании результатов ПЦР 10% (5/50) из 50 обследованных образцов детей-аутистов оказались положительными на *C. ventriculi*, а все образцы контрольной группы были отрицательными. Генетические полиморфизмы были выявлены с помощью специфических нуклеотидных переходов и трансверсий, обнаруженных при секвенировании. Иракские изоляты и зарубежные образцы показали высокий уровень генетического сходства (99%) согласно филогенетическому анализу, что указывает на недавнего общего предка и потенциальное клональную экспансию. *Выводы.* Обнаружение *C. ventriculi* у детей-аутистов повышает вероятность связи между этой бактерией и желудочно-кишечными нарушениями, связанными с РАС.

Ключевые слова: *Clostridium ventriculi, расстройство аутистического спектра, филогенетический анализ, ген 16S рРНК.*

Introduction

Repetitive habits, limited interests, difficulties interacting with others, and communication difficulties are all hallmarks of autism spectrum disorder (ASD). It is a condition with genetic origins [4].

The community of around 10^{14} bacteria makes up the gastrointestinal (GI) microbiota. There are 100 times as many genes in these bacteria's genetic material, or microbiome, as there are in the human genome. More than 10^3 species can be found in the human gut ecology. The GI microbiota influences the onset of disease and contributes to health maintenance [5].

This microbial community helps break down components of our diet in the gut and offers protection against viruses, supports immune system training, and impacts gastrointestinal maturation. Additionally, microbes have a major role in the emergence of many diseases [3, 36].

The composition of the gastrointestinal (GI) microbiota is primarily ascertained by age, genetic factors, and nutrition [27].

Several individuals with autism display various gastrointestinal disorders, such as diarrhea, constipation, gas retention, abdominal pain, and discomfort. It is possible that aberrant gut microflora contributes to these issues. Multiple publications have shown an association between *Clostridium* and these difficulties [10].

Clostridium ventriculi (C. ventriculi), alternatively referred to as *Sarcina ventriculi (S. ventriculi)*, is a rare, anaerobic, non-motile, Gram-positive coccus that ferments carbohydrates. It thrives and proliferates in acidic environments [31].

C. ventriculi, a member of the family *Clostridiaceae*, is derived from the Latin word "sarcina", which means "package", due to its typical formation of tetrads or octets. The precise pathogenic role of *C. ventriculi* in humans remains uncertain [22].

This bacterium is linked to serious gastrointestinal issues, especially in people who have obstruction of the gastric outlet or delayed stomach emptying. It is associated with acute blood loss anemia, stomach perforation, emphysematous gastritis, and, infrequently, gastric or colorectal cancer in adults [6, 8, 11].

Although less frequent, pediatric cases exhibit comparable symptoms, with documented occurrences of emphysematous gastritis-related fatal stomach rupture, frequently preceded by persistent vomiting and gastric distension [22, 29].

The bacterium's capacity to digest carbohydrates and produce gas that worsens tissue necrosis and ischemia puts both groups at risk for potentially fatal consequences [14, 29].

Histopathological identification of its characteristic cuboid tetrads in biopsies is necessary for diagnosis [22, 29].

The usual course of treatment includes acid suppression, management of underlying motility issues, and antibiotics (such as metronidazole and penicillins) [6, 9, 14].

Through toxin-mediated pathways and dysbiosis of the gut microbiota, *Clostridium* species are linked to the onset and severity of autism spectrum disorder (ASD). According to research, children with ASD have higher levels of *Clostridium* (e.g., *Clostridium perfringens, Clostridium bolteae,* and *Clostridium histolyticum*) than controls do. This is especially true for strains that produce β2-toxin and neurotoxic metabolites like 3-(3-hydroxyphenyl)-3-hydroxypropionic acid (HPHPA), which interfere with dopamine metabolism and deplete catecholamines in the brain [3, 15, 17].

By cleaving proteins like synaptobrevin, clostridial toxins, such as *C. tetani*'s tetanus neurotoxin (TeNT), can travel through the vagus nerve and enter the central nervous system, affecting synaptic function and resulting in behavioral and neurodevelopmental abnormalities [18].

Furthermore, *Clostridium* species generate phenolic compounds and short-chain fatty acids (like propionic acid) that worsen intestinal permeability, neuroinflammation, and oxidative stress, which promotes the systemic spread of neuroactive metabolites [3, 15, 18].

The gut-brain axis may be further disrupted by environmental variables, such as glyphosate exposure, which may selectively suppress good gut bacteria and encourage the growth of *Clostridium* [3].

Vancomycin and fecal transplants, two interventions that target the gut microbiota, temporarily low-

er *Clostridium* levels and alleviate symptoms of ASD, highlighting their pathogenic involvement [17, 18].

In children with autism spectrum disorder (ASD), gastrointestinal issues have been closely linked to *Clostridium* species, especially *Clostridium histolyticum* (clusters I and II), *Clostridium bolteae, Clostridium perfringens, Clostridium paraputrificum*, and *Clostridium difficile*. Research has repeatedly demonstrated that ASD patients' stools contain higher concentrations of these species than those of neurotypical controls. For instance, children with gastrointestinal symptoms who had ASD were far more likely to have *C. perfringens* strains that produced β_2-toxin genes, which may be a contributing factor to the gut problems associated with ASD. Furthermore, whereas neurotypical youngsters had distinct *Clostridium* profiles, ASD patients were the only ones to have *C. bolteae* and *C. paraputrificum* [3, 19].

Molecular approaches in microbial ecology allow for the examination of intestinal flora composition without the need for culturing. This is accomplished using particular primers, yielding extremely sensitive, practical, and quick results [26].

The purpose of this study was to use 16S rRNA gene sequencing to identify and genetically describe *Clostridium ventriculi* in fecal samples from children with autism.

Materials and methods

Specimens. The sample for this case-control study included 50 patients with ASD diagnoses, 40 of whom were male and 10 of whom were female, ages 2 to 8. They were chosen from two nearby facilities that specialize in treating autism. Samples were also taken from 50 children who were not autistic as a control group.

The study excluded children with serious head traumas, neurological disorders, severe physical abnormalities, and gastrointestinal problems like bloating, gas, indigestion, constipation, or recurrent diarrhea. Additionally, the children had not been administered antibiotics or functional foods (probiotics, prebiotics) for at least a month prior to the sampling.

Sampling. The fecal sample was taken before breakfast after each child fasted overnight. Three successive fecal samples were collected from each child. The samples were placed in containers made of sterile plastic, then suspended and stored at a temperature of –70°C for subsequent DNA extraction.

DNA Extraction. Following the manufacturer's recommendations, genomic DNA was extracted from fecal samples using the QIAamp® DNA Stool Mini Kit (Qiagen, Germany). Electrophoresis on a 1% agarose gel was used to verify the existence and quality of the isolated DNA.

PCR Amplification. The 16S rRNA gene was amplified by PCR with universal primers 27F (5′-AGAGTTTGATCCTGGCTCAG-3′) and 1492R (5′-TACGGYTACCTTGTTACGACTT-3′) (10). In a 25 µl reaction mixture, 1.5 µl of DNA, 12.5 µl of Green Master Mix PCR (Promega, USA), 1 µl of each primer, and nuclease-free water were used for PCR amplification. Thirty-five cycles of denaturation for 45 seconds, annealing at 55°C for 45 seconds, extension at 72°C for 45 seconds, and final extension at 72°C for 7 minutes were performed after the initial denaturation at 95°C for 3 minutes. A 1.5% agarose gel electrophoresis was used to separate the PCR products.

Sequencing. The Sanger sequencing method was used to sequence the amplified PCR products (25 µL) and 50 µL of primers, which were delivered to Macrogen (Seoul, South Korea). BioEdit software and NCBI's BLAST program were used to evaluate the resultant sequences. Homologous sequences were found by comparing them to the NCBI GenBank database. To evaluate the robustness of the tree topology, the phylogenetic tree was visually shown using the Molecular Evolutionary Genetics Analysis (MEGA) software version 6.0 and the neighbor-joining procedure with 1000 bootstrap replicates. The acquired 16S rRNA sequences were added to the NCBI GenBank database with the accession numbers listed below: OM943844.1, OM943845.1, OM943846.1, OM943847.1, OM943848.1.

Results

The results presented in this study were based on analyses of data from a total of 50 children with autism. The findings indicated a higher risk of autism in males (40, 80%) compared to females (10, 20%).

To identify the 16S rRNA gene, 50 samples were subjected to PCR analysis. PCR analysis showed that 10% (5/50) of the samples had positive *Clostridium ventriculi* findings, distributed as 3 males and 2 females. All control group samples were negative for this bacterium.

Certain genetic alterations in *Clostridium ventriculi* are revealed by the 16S rRNA gene sequencing analysis. At nucleotide positions 63 and 43, respectively, these alterations consist of one transition (C to T) and one transversion (T to A). Furthermore, at nucleotide positions 93 and 129, there is one transversion (G to C) and one transition (A to G). Additionally, there are three transversions (A to C) at nucleotide positions 157, 195, and 207, as well as a transition (T to A). Moreover, there is one transition (C to T) at nucleotide position 227, and one transition (C to T) and one transversion (C to A) at nucleotide positions 306 and 328.

Analysis of the GenBank revealed a segment of the 16S rRNA gene that has 99% similarity with the 16S rRNA gene sequence in NCBI, as shown in Table.

The sequences with the highest degree of similarity and the most extensive overlap were discovered by comparing isolates acquired from dialysis patients.

Table. Type of polymorphisms of 16S rRNA gene

No. of sample	Type of substitution	Location	Nucleotide change	Reference Sequence	Sequence ID submission
1	Transvertion	43	T/A	ID: MG733966.1	ID: OM943844.1
	Transition	63	C/T		
2	Transition	93	A/G	ID: MG733966.1	ID: OM943845.1
	Transvertion	129	G/C		
3	Transvertion	157	A/C	ID: MG733966.1	ID: OM943846.1
	Transvertion	195	A/C		
	Transvertion	207	T/A		
4	Transition	227	C/T	ID: MG733966.1	ID: OM943847.1
5	Transition	306	C/T	ID: MG733966.1	ID: OM943848.1
	Transvertion	328	C/A		

Note. Abbreviations: T: Thymine, A: Adenine, C: Cytosine, G: Guanine.

A neighbor-joining tree was created for the phylogenetic analysis. The genetic relatedness between Iraqi isolates and isolates from various regions worldwide is illustrated using the phylogenetic tree. According to hierarchical cluster analysis, there are multiple clusters. The main cluster is separated into several subgroups. Many global isolates showed a high degree of similarity to the Iraqi isolates studied in this research, as shown in Fig.

Discussion

Researchers have found that people with ASD have more of the bacterium *Clostridium ventriculi* in their digestive tracts [25]. Because there is a link between *Clostridium ventriculi* and autism, scientists are studying how gut bacteria affect the behavior and symptoms of kids with autism.

De Angelis employed the 16S rRNA gene, a bacterial species marker, to detect *Clostridium ventriculi* in individuals with autism [9], The study's findings may be consequential in clarifying the influence of *Clostridium ventriculi* on the gut microbiota of individuals with autism. If a consistent association between this bacterium and autism is established, research into the influence of gut bacteria on the behavioral manifestations of autism may be stimulated [9]. Research indicates that individuals with ASD exhibit alterations in gut microbiota, suggesting that imbalances, such as an excess or deficiency of specific bacteria, may exacerbate ASD symptoms [11, 30].

Several studies have shown that people with ASD have higher levels of *Clostridium* than people who don't have ASD, these studies also indicate that *Clostridium* may generate neurotoxins that could affect the etiology of ASD [3, 10].

The 16S rRNA gene presents numerous significant benefits for the phylogenetic analysis of *Clostridium ventriculi*, The 16S rRNA gene is mostly the same in all organisms, but it does have some parts that are different from others that help tell species apart. This dual nature makes it feasible to draw evolutionary connections between *Clostridium ventriculi* and other genus members. Even in preserved areas, research has shown that there is enough variation to effectively resolve phylogenetic relationships [17, 33]. A strong basis for comparison is provided by the large collection of 16S rRNA sequences. Because there are several sequences from related *Clostridium* species, researchers may accurately position *C. ventriculi* into the wider clade, increasing the validity of phylogenetic findings [7, 14].

The distribution of the Iraqi samples throughout several phylogenetic tree branches indicates genetic variety. According to the study of relationships, there may be variance within this group, as certain Iraqi isolates (such as OM943844.1 and OM943845.1) have closer genetic distances than others (like OM943846.1). The genetic diversity seen in Iraqi *Clostridium ventriculi* isolates may be due to differences in how well they adapt to different environments and how well they fit into different niches in the gastrointestinal tract or other places, as shown by how they are spread out on the phylogenetic tree [2].

The distribution of *Clostridium ventriculi* isolates from Iraq over various branches of the phylogenetic tree indicates genetic variation among these isolates. Some Iraqi isolates, like OM943844.1 and OM943845.1, form tight clusters with little genetic separation. Others, like OM943846.1, spread out more, which suggests that there is some variation within the group, this diversity may arise from environmental influences, host-specific adaptations, or evolutionary divergence over time. Bacterial phylogenetic studies frequently highlight the influence of ecological niches and geographic isolation on genetic variation within species [24, 28].

The genetic similarity seen in the isolates from Iraq may be due to the ancestor's spread, as well as genetic drift, a small population size, or a lack of selective forces that encourage diversity. Consequently, the genetic diversity of the strains may be limited [18, 20].

The phylogenetic tree revealed a close genetic relationship among a group of isolates from the Czech Republic, Saudi Arabia, Japan, the UK, Norway, Ireland, and Russia. Some isolates from Iraq belong to a distinct branch, whereas others are located within this cluster, indicating a lesser degree of re-

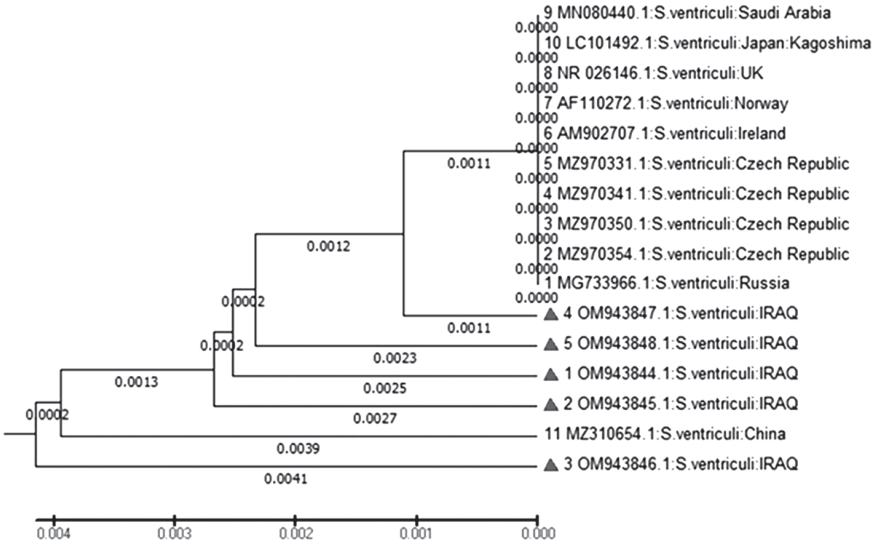

Figure. Neighbor-joining tree of *Clostridium ventriculi* based on the 16S rRNA gene

Note. Triangles indicate the positions of the Iraqi isolates within the phylogenetic tree.

latedness. The complicated connections between *C. ventriculi* strains from different parts of the world are made even clearer by the fact that a single isolate from China has appeared on a separate branch. Horizontal gene transfer, environmental factors, and historical patterns of core genome migration all work together to explain why bacterial strains from different countries are genetically similar. These components enhance the intricate relationships elucidated in bacterial phylogenetics, allowing populations from various geographic regions to exhibit significant genetic similarities [23, 28, 34].

Conclusion

The examination of *Clostridium ventriculi* and ASD highlights the increasing focus on the gut-brain-microbiota axis and its possible link to neurological disorders. Research demonstrates that individuals with ASD show considerable changes in their gut microbiota composition, marked by increased levels of *Clostridium ventriculi*, this bacterium may exacerbate symptoms of ASD through mechanisms such as neurotoxic production and intestinal dysbiosis, which can affect behavior and cognitive function.

Phylogenetic analysis of the 16S rRNA gene has elucidated the genetic diversity of *Clostridium ventriculi* isolates from Iraq and other regions. The differences between isolates could be due to evolutionary divergence, factors that are specific to the host, or changes in the environment, these findings indicate that ecological and geographical factors influence the genetic composition of bacterial strains.

While the link between *Clostridium ventriculi* and ASD is promising, more research is needed to find out what causes it and how it works, if validated, this correlation may enhance therapeutic approaches targeting gut microbiota to mitigate ASD symptoms. In conclusion, the study highlights the significance of comprehending the role of gut microbiota in neurological disorders and the prospects of microbiome-based therapies.

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Авторы:

Хасан А.Х., доктор философии в области микробиологии, преподаватель, Университет медицинских и фармацевтических наук Ибн Сины, Багдад, Ирак;
Лазим Х.Х., доктор философии в области микробиологии, преподаватель, Университет медицинских и фармацевтических наук Ибн Сины, Багдад, Ирак.

Authors:

Hasan A.H., PhD (Microbiology), Lecturer, Ibn Sina University of Medical and Pharmaceutical Sciences, Baghdad, Iraq;
Lazim H.H., PhD (Microbiology), Lecturer, Ibn Sina University of Medical and Pharmaceutical Sciences, Baghdad, Iraq.

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