Assessment of Genetic Mutations in Genes DSM-IV, DRD4, SERT, HTR1B, SNAP25, GRIN2A, ADRA2A, TPH2 and BDNF Induced Attention Deficit Disorder and Hyperactivity in Children

Shahin Asadi1, Mahsa Jamali2, Zahra Gholizadeh2, Mina Niknia1 and Maryam Sati2

1Research Center for Stem Cell and Drug Applied Research Center, Tabriz University of Medical Sciences in Modern Biology, Tabriz, Iran
2Young Researchers and Elite Club, Islamic Azad University, Tabriz, Iran

Corresponding author: Shahin Asadi, Student of Molecular Genetics, Research Center for Stem Cell and Drug Applied Research Center, Tabriz University of Medical Sciences in Modern Biology, Tabriz, Iran. Tel: +989379923364; E-mail: shahin.asadi1985@gmail.com

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Abstract

In this study we have analyzed 1200 people, 580 ADHD and 620 control groups. The genes DSM-IV, DRD4, SERT, HTR1B, SNAP25, GRIN2A, ADRA2A, TPH2 and BDNF analyzed in terms of genetic mutations made. In this study, people who have genetic mutations were targeted, with nervous disorders, ADHD. In fact, of all people with ADHD, 360 children had a genetic mutation in the genes DSM-IV, DRD4, SERT, and HTR1B. And 100 children had a genetic mutations in SNAP25, GRIN2A genes were a genetic mutations in the genes ADRA2A, TPH2 and BDNF and 76 children, respectively. Any genetic mutations in the target genes control group, did not show.

Hypothesis and objectives: In this study, we further understand the genes involved in ADHD children with the genetic mutations discussed. The aim of this study was to evaluate genetic and epigenetic closer to induce hyperactivity disorder in children.

Keywords: Genetic study; ADHD; Mutations; The genes DSM-IV; DRD4; SERT; HTR1B, SNAP25; GRIN2A; ADRA2A; TPH2; BDNF

Introduction

Today, hyperactivity or attention deficit disorder in children, neurological disorders are incurable and usually fatal disease. Hyperactivity always caused by genetic mutations that have the potential to be transmitted from mother to child. If the mother during pregnancy, the drug as well as strong antibiotics and sedatives as well as taking abortion drugs, the probability that a child is hyperactive, very much.

Attention deficit hyperactivity disorder (ADHD)

Is a mental disorder of the neurodevelopmental type [1,2]. It is characterized by problems paying attention, excessive activity, or difficulty controlling behaviour which is not appropriate for a person’s age [3]. These symptoms begin by age six to twelve, are present for more than six months, and cause problems in at least two settings (such as school, home, or recreational activities) [4,5]. In children, problems paying attention may result in poor school performance [3]. Although it causes impairment, particularly in modern society, many children with ADHD have a good attention span for tasks they find interesting [6].

Despite being the most commonly studied and diagnosed mental disorder in children and adolescents, the cause is unknown in the majority of cases [7]. The World Health Organization (WHO) estimated that it affected about 39 million people as of 2013 [8]. It affects about 5% to 7% of children when diagnosed via the DSM-IV criteria [9,10] and 1% to 2% when diagnosed via the ICD-10 criteria [11]. Rates are similar between countries and depend mostly on how it is diagnosed [12]. ADHD is diagnosed approximately three times more often in boys than in girls [13,14]. About 30% to 50% of people diagnosed in childhood continue to have symptoms into adulthood and between 2% to 5% of adults have the condition [15,16]. The condition can be difficult to tell apart from other disorders, as well as to distinguish from high levels of activity that are still within the normal-range [5].

Materials and Methods

In this study, 580 patients with ADHD and 620 healthy controls were studied. Peripheral blood samples from patients and parents with written permission control were prepared. After separation of serum, using Real Time-PCR technique of tRNA molecules was collected. To isolate Neuroglial cells erythrocytes were precipitated from hydroxyethyl starch (HES) was used. At this stage, HES solution in a ratio of 1 to 2 on ficole was poured in the 480G was centrifuged for 34 minutes. Mono nuclear Neuroglial cells were studied. Peripheral blood samples from patients and parents with written permission control were prepared. After separation of serum, using Real Time-PCR technique of tRNA molecules was collected. To isolate Neuroglial cells erythrocytes were precipitated from hydroxyethyl starch (HES) was used. At this stage, HES solution in ratio of 1 to 5 with the peripheral blood of patients and controls were mixed. After 60 minutes of incubation at room temperature, the supernatant was removed and centrifuged for 14 min at 400 Gera. The cell sediment with PBS (phosphate buffered saline), pipetazh and slowy soluble carbohydrate ratio of 1 to 2 on ficole (Ficoll) was poured in the 480G was centrifuged for 34 minutes. Mono nuclear Neuroglial cells also are included, has a lower density than ficole and soon which they are based. The remaining erythrocytes have a molecular weight greater than ficole and deposited in test tubes.

The supernatant, which contained the mononuclear cells was removed, and the 400 Gera was centrifuged for 12 minutes. Finally, the sediment cell, the antibody and Neuroglial cells was added after 34 minutes incubation at 5°C, the cell mixture was passed from pillar LSMAC. Then the cells were washed with PBS and attached to the
column LSMACSS pam Stem cell culture medium containing the transcription genes DSM-IV, DRD4, SERT, HTR1B, SNAP25, GRIN2A, ADRA2A, TPH2 and BDNF were kept (Figures 1-8).

Figure 1: SNAP25 gene pattern band formation in patients with ADHD.

Figure 2: TPH2 gene pattern band formation in patients with ADHD.

To determine the purity of Neuroglial cells are extracted, flow cytometry was used. For this purpose, approximately $4.5 \times 10^3$ Neuroglial cells were transferred to a new microtube and to the one times the volume of cold ethanol was added. The resulting mixture for 24 hours at -20°C was incubated.

2) Then for 45 min at 4°C, it was centrifuged at 12000 rpm era. Remove the supernatant and the white precipitate, 1 ml of cold 75% ethanol was added to separate the sediment from micro tubes were vortex well. The resulting mixture for 20 min at 4°C and by the time we were centrifuged 12000 rpm. Ethanol and the sediment was removed and placed at room temperature until completely dry deposition.

The precipitate was dissolved in 20 μl sterile water and at a later stage, the concentration of extracted mRNA was determined.

Figure 3: DRD4 gene pattern band formation in patients with ADHD.

Figure 4: SERT gene pattern band formation and diagram in patients with ADHD.

To assess the quality of mi-RNAs, the RT-PCR technique was used. The cDNA synthesis in reverse transcription reaction (RT) kit (Fermentas K1622) and 1 μl oligo primers 18(dT) was performed. Following the PCR reaction 2 μM dNTP, 1μg cDNA, Fermentas PCR buffer 1X, 0.75 μM MgCl$_2$, 1.25 U/μl Tag DNA at 95°C for 4 min, 95°C for 30s, annealing temperature 58°C for 30s, and 72°C for 30 seconds, 35 cycles were performed. Then 1.5% agarose gel, the PCR product was dumped in wells after electrophoresis with ethidium bromide staining and color was evaluated.
Discussion and Conclusion

According to the results of sequencing the genome of patients with ADHD, and the genetic mutations DSM-IV, DRD4, SERT, HTR1B, SNAP25, GRIN2A, ADRA2A, TPH2 and BDNF induced Attention Deficit Disorder and Hyperactivity in Children. J Neurol Disord 4: 324. doi:10.4172/2329-6895.1000324
about 91% of patients with ADHD, they have these genetic mutations. Patients with ADHD, unusual and frightening images in the process of ADHD experience. Lot epigenetic factors involved in ADHD. But the most prominent factor to induce ADHD, mutations is DSM-IV, DRD4, SERT, HTR1B, SNAP25, GRIN2A, ADRA2A, TPH2 and BDNF genes. These genes can induce the birth and can also be induced in adulthood. This study is the largest genetic study of ADHD in the country and one of the largest global research in the genetics of psychiatric illness is more active. This study shows that many genes and lifestyle, and diet of the mother during pregnancy induced hyperactivity disorder in children is involved. We hope researchers in the field as well as additional genes are examined for the disease (Figures 1-8).

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