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Hereditary factors of bruxism

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ABSTRACT

Background: Bruxism is a parafunctional disorder. The prevalence of this rhythmic activity of rodent muscles is reported to be about %8. This disease can compromise the life quality of a person's general performance. The aim of this study is to gather information upon genetic factors, which contribute to the pathogenesis of the disease.

Materials and Methods: All related articles published in 1966 onward from google scholar such as ISI, PubMed, Scopus and Ovid within the databases were searched using English keywords 'Bruxism and Genetics'. 300 articles were found. 252 articles were removed due to content duplication and irrelevance.

Results: The review of selected articles finally showed that in addition to other factors such as psychological factors, local factors, systemic factors, etc., the genetic factors also play a significant role in pathogenesis of bruxism. Among the influential genes are rs6313 polymorphism from the 5HT2A gene and rs6313 polymorphism from the HTR2A gene.

Conclusion: Evidence suggests that genetic factors play an important role in the pathology and development of bruxism, however the main causing mechanism still largely remains unknown.

Keywords: Genetic factors; Bruxism; Article review; Polymorphism.

Introduction

Bruxism mostly occurs during sleep, however the phenomenon can be observed in awake periods too. Both adults and children can be affected. Simultaneous bruxism can cause tooth erosion (severe tooth sensitivity) and shortening (especially in the anterior teeth); gingival damage, enamel fractures and damage to dental fillings, jaw joint problems, tooth loss followed by bone loss, reduction in facial vertical height, inadequate jaws shapes; mechanical implant complications; masseter muscle hypertrophy and masticatory fatigue; increase migraine risk and pain and impairment of Temporomandibular joint (TMJ) [1]. Bruxism data has been reviewed worldwide and reported by medical and dental researchers, with overall prevalence of sleep bruxism reported to be 15.9% and awake bruxism 23.8% [2]. Sleep bruxism complaints have been reported in 14% of children, 8% of adults, 3% of people over 60 years of age.In one study, the prevalence of bruxism in children was reported to be 5-46%.

The prevalence of bruxism in 600 Iranian children aged 4-12 years has also been reported 26.2%. There is no gender difference observed in this complication. But some studies have reported it to be more common in women than in men [3]. Evidence from dental practices suggest that bruxism may be hereditary as most people who visited their dentist have reported one or more family members with bruxism [4]. Studies have shown that inheritance is partially involved in bruxism [5]. 21–5% of children with bruxism have first-degree relatives with sleep bruxism during their lifetime [6]. Since bruxism usually occurs at night, sleep physiology, especially the "arousal response" has been studied. This response is associated with basic body movements, increased heart rate, respiratory changes, and increased muscle activity. One article reported that 86% episodes of bruxism were associated with arousal response during involuntary leg movements, indicating that bruxism is indeed part of arousal response [7,8]. There is growing scientific evidence for bruxism disorders that suggest genetic factors do play an important role in the pathology of the disorder. Bouchi et al investigated the prevalence of sleep bruxism in patients with IBD and its association with other dental disorders and patients' life quality. Sleep disorder and enamel attrition lesions were more frequent in patients with Crohn's disease than in patients with colitis and control cases. There was no significant difference between groups of enamel hypoplasia, temporomandibular disorders, pulmonary aphthous stomatitis, depression and sleep quality [9].

In addition a Finnish twin study investigated the genetic factors and phenotypic differences of bruxism in relation to sleep apnea in adolescents, where they stated that genetic factors have a significant influence on phenotypic variation of responsibility for bruxism in sleep, with gender having no influence on its prevalence [10]. A thorough examination on the prevalence of bruxsim's phenotype, psychosis and genotype was carried out. The purpose of this study was to determine the relationship between extraversion, anxiety, and neuroticism with psychomorphism of 6313 rs polymorphism of gene HTR2A and prevalence of bruxism. Homogeneous analysis of genotype showed no significant difference in allele frequency among the four groups On the other hand, there was no significant difference between the SNPs6313rs (HTR2A gene) groups [11]. An investigation on genetic polymorphism in the serotonergic system associated with the manifestations of tooth grinding suggested that the CNS and their genes may be involved in the development of bruxism and thus serotonin can be associated with the pathogen of tooth grinding. The data showed that there were significant differences in allele frequency for the 2770304rs polymorphism of the gene HTR2A and occurrence of bruxism. The results of this research group show that polymorphisms are involved in serotonergic pathways in teeth grinding [12]. The rs6313 polymorphism of the HTR2A gene is significantly associated with an increased risk of sleep bruxism. Moreover, in a publication by Abbey et al. they reported on a study identifying genetic markers for bruxism and concluded that genetic factors can contribute to the etiology of sleep bruxism [13]. Hereditary factors in the evaluation of Bruxsim and its different diagnosis were found, especially in first-degree relatives and the rs6313 polymorphism of the HTR2A gene whose association with stress, anxiety, and neurological disorders have been documented, with the association of the rs6313 polymorphism of the HTR2A gene with the changes that these emotions cause in the serotonergic system [14].

Wiga et al. studied Bruxism and data were searched using PubMed and ScienceDirect databases using 'Bruxism' keywords, causes and symptoms, wear and tear, dental injuries, musculoskeletal disorder and TMJ dysfunction. Following the entire abstract analysis of the 102 articles, 34 articles and 4 textbooks were included. The results showed that the main consequences of bruxism are as follows: muscle fatigue, dental pain, severe use of occlusal dental surfaces, and in severe cases tooth loss, headache, periodontal lesions, and TMJ disorders [15]. In 1970, Chorskin et al. studied the effect of nutritional supplements on bruxism and dental compression. In conclusion patients who used calcium and vitamin B5 supplements showed improvement [16,17]. Four years later, Lehvila et al reported a significant decrease in the activity and duration of bruxism episodes in 6 patients who consumed one tablet daily of vitamins and minerals including 25mg magnesium in children and 100mg adult dose for at least 5 weeks. As a result, magnesium supplements have been suggested to be used in the treatment of Bruxism [18,19].

Material and Methods

In order to address the issue of bruxism, a narrative review was done, with the help of (PubMed, Google Scholar, ISI, Scopus, Ovid, Up to date) foreign databases. We have also considered time limitations from 1995 to 2018 using the English keywords; (Genetic factors, Bruxism) were searched and 300 studies have been found. After excluding duplicate articles (130 articles), 170 remaining articles were excluded from the

study after reviewing 77 article titles due to having irrelevant article titles to our research review topic. Furthermore, of the remaining 93 articles, after reviewing the abstracts, a number of them were excluded too due to the lack of abstraction and summarization with the subject of our review. In summary: Articles were excluded based on topics, abstracts, repetition, and non-topic relevance. As a result, 48 full-text interventional articles in the field of genetic factors and its role in Bruxism disease were reviewed which matched our case study.

The criteria for selecting articles in this study were as follows:

A) Naming the genes identified in Bruxism.

B) They were published as research articles, not review and edited articles.

C) Bruxism with characteristics of gene mutations or family history.

D) We made English-language articles as the standard for the search.

Results

Table 1 suggests that investigation of selected articles show that in addition to genetic factors other factors such as psychological factors, local factors, etc. have a significant role in the incidence and pathogenesis of Bruxism. Among the affecting genes are rs6313 polymorphism of 5HT2A gene and rs6313 polymorphism of HTR2A genes. Overall, these genes and their associated polymorphism "inheritance" can confirm the genetic impact for the onset of Bruxism. From the mentioned studies (Table 1) , it was concluded that the polymorphisms (rs6313) of the HTR2A gene and the polymorphism (rs6313) of the 5HT2A gene were the most effective polymorphisms in causing Bruxism.

Authors	*The Relationship between Bruxism and Genetics*	*Prevalence by age*	*Prevalence by gender*	*The Relationship between Bruxism and Family History*	*Effective gene polymorphisms*
Reding et al. 1966	*N/A*	*N/A*	*N/A*	*N/A*	*N/A*
Cheraskin et al. 1970	*N/A*	*N/A*	*N/A*	*N/A*	*N/A*
Lehivila et al. 1974	*N/A*	*N/A*	*N/A*	*N/A*	*N/A*
Pavone1985	*N/A*	*N/A*	*N/A*	*N/A*	*N/A*
Pierce et al. 1995	*N/A*	*N/A*	*N/A*	*N/A*	*N/A*
Tan EK 2000	*N/A*	*N/A*	*N/A*	*N/A*	*N/A*
Nissan i et al 2001	*N/A*	*N/A*	*N/A*	*N/A*	*N/A*
Macfarlane et al. 2002	*N/A*	*N/A*	*N/A*	*N/A*	*N/A*
Wilhelm et al. 2007	*YES*	*N/A*	*N/A*	*YES*	*(HTR2A) rs6313*
Schneider et al. 2007	*N/A*	*N/A*	*N/A*	*N/A*	*N/A*
Donnellan et al. 2008	*YES*	*N/A*	*N/A*	*N/A*	*(5HT2A) rs6313*
Oakley et al. 2008	*N/A*	*N/A*	*N/A*	*N/A*	*N/A*
Ribeiro-Dasilva et al 2009	*N/A*	*N/A*	*N/A*	*N/A*	*N/A*
Gungormus et al. 2009	*N/A*	*N/A*	*N/A*	*N/A*	*N/A*
Shetty et al. 2010	*N/A*	*N/A*	*N/A*	*N/A*	*N/A*
Pakpour et al. 2011	*N/A*	*N/A*	*N/A*	*N/A*	*N/A*

Abey et al. 2012	YES	N/A	N/A	N/A	(HTR2A) rs6313 HTR24
Bayar et al. 2012	N/A	N/A	N/A	N/A	N/A
Nixdorf et al. 2012	N/A	N/A	N/A	N/A	N/A
Arzul et al. 2012	N/A	N/A	N/A	N/A	N/A
Rintakoskik et al. 2012	YES	N/A	N/A	N/A	UNKNOWN
Fraley et al. 2013	YES	N/A	N/A	N/A	(5HT2A) rs6313 (HTR2A) rs6313
Ahlberg et al. 2013	N/A	N/A	N/A	N/A	N/A
Durham et al. 2013	N/A	N/A	N/A	N/A	N/A
White ford et al. 2013	N/A	N/A	N/A	N/A	N/A
Swaminathan et al. 2014	N/A	N/A	N/A	N/A	N/A
Esquerra-trabalon et al. 2014	YES	N/A	N/A	N/A	(HTR2A) rs6313
Lobbezoo et al. 2014	YES	N/A	N/A	N/A	UNKNOWN
Lucas et al.2014	N/A	N/A	N/A	N/A	N/A
Dalewski et al. 2015	N/A	N/A	N/A	N/A	N/A
Souzava et al 2015	N/A	N/A	N/A	N/A	UNKNOWN
Veiga et al. 2015	N/A	N/A	N/A	N/A	N/A
Chercanovic et al. 2015	N/A	N/A	N/A	N/A	N/A
Murali et al. 2015	N/A	N/A	N/A	N/A	UNKNOWN
Wieskopt et al. 2015	N/A	N/A	N/A	N/A	N/A
Emami Moghadam et al. 2015	N/A	N/A	N/A	N/A	N/A
Gorski 2015	YES	N/A	N/A	N/A	UNKNOWN
Durham et al. 2016	N/A	N/A	N/A	N/A	N/A
Khaled et al. 2016	N/A	N/A	N/A	N/A	N/A
Oporto et al. 2016	YES	N/A	N/A	N/A	(HTR1A) rs6295 (HTR2A) rs2770304 (HTR2A) rs6313 HTR2C) (rs17260565 SLC6A4) rs6374904
Butler et al. 2016	N/A	N/A	N/A	N/A	N/A
Yuce et al. 2017	N/A	N/A	N/A	N/A	N/A
Xue X – T et al. 2017	N/A	N/A	N/A	N/A	N/A
Deoss. et al 2017	N/A	N/A	N/A	N/A	N/A

Jimenez-silva et al. 2017	*YES*	*N/A*	*N/A*	*N/A*	*UNKNOWN*
Bucci et al. 2018	*N/A*	*N/A*	*N/A*	*N/A*	*N/A*
Cruz-Fierroet al. 2018	*YES*	*N/A*	*N/A*	*N/A*	*(HTR2A) rs6313 SNPs*
Ondo et al 2018	*N/A*	*N/A*	*N/A*	*N/A*	*N/A*

Table 1. Summary of reviewed articles from 1966 to 2018.

Discussion

To date, the exact causes of bruxism have not been identified. It is one of the most common sleep disorders and one of the unconscious and involuntary neuromuscular activities and causes pain and dysfunction of the TMJ; with other complications such as myofascial muscle pain, head and neck pain and burnout, wear and tear, tooth decay, destruction of structures that support dental complex cause muscle pain and spasms and also creates aesthetic problems. In severe cases, this habit can lead to osteoarthritis of the jaw joint [20]. Early detection of the disorder stops its progression in the early stages and also reduces associated treatment costs. The need to determine its prevalence in different communities and to identify its associated factors has been emphasized. Several studies have shown that for this disorder, symptoms such as oral pain are very common, in which young adults are at higher risk being affected, and these symptoms can have adverse effects on physical, social, psychological and personality performance. Common consequences for bruxism include avoiding certain types of foods, taking medication, sleep disorders, inability to work, the need to rest and reduced social engagements [21].

Prevention modalities and treatment for bruxism include patient education and early self-care. If a person is more likely to be infected than the general population, they can be informed from an early age especially those with susceptible polymorphisms. The rationale for selecting behavioral therapies stems from the fact that parafunctional activities and social and psychological factors play an important role in causing muscle pain [22]. The purpose of these trainings are to reassure the patients, explain parafunctional nature and its possible causes. Early disease detection and ultimately reducing repetitive muscle tensions such as teeth grinding, encourage and strengthen the vulnerable individuals, teach them relaxation and control related muscle activities [23]. Various causes and factors that may have been involved in the development of Bruxism have been discussed, however the genetic factor contribution is also emphasized and confirmed, and as a result some researchers and even psychologists characterize several genes for the expression of a unique or specific existence with bruxism in anxious individuals and individuals with neurological disorders; indicating association with specific polymorphism rs6313 of the 5HT2A gene [24,25]. Indeed, various psychological factors have a contributory role [26,27,28,29].

In various evaluations and diagnoses, we also came across hereditary factors [30], especially between first-degree relatives and specific gene function that has been shown to be physical and behavioral throughout this period [31]. The polymorphism rs6313 of the HTR2A gene has been shown to be associated with stress, anxiety, and neurological disorders, along with the association of the rs6313 gene polymorphism with the HTR2A gene with changes in these sensations in the serotonergic system [25,32]. It also plays an important role in relation to sleep bruxism [33]. There are thirteen polymorphisms within the four genes involved in serotonergic neurotransmitters (SLC6A4, HTR1A, HTR2A, and HTR2C). These factors have been selected and predicted to compare the case (sleep bruxism) and control group. Five predictions of our choice: Epworth›s sleep scale, leg-restlessness, foot restlessness symptoms such as stress, anxiety, neurological and mental disorders, rs6313, rs2770304, rs4941573. Several variables of step-by-step analysis between the chosen ones were predicted and the state of sleep bruxism was adjusted in them. Whether these genes affect sleep bruxism or not? Analysis showed that only allele C carries the rs6313 polymorphism of the HTR2A gene (102C>T), which is significantly associated with an increased risk or risk of sleep bruxism. The findings suggest that genetic causes may contribute strongly to the development of sleep bruxism (odds ratio=4.250 95% confidence interval: 1.599-11.297 P=0.004) [33]. DNA analysis has also been described in one of the publications. The study was performed by identifying genetic markers for sleep bruxism. A case-control study

was used focusing on genetic factors associated with serotonin metabolism and neurotransmitters that are believed to play a role in the etiology of bruxism. A specific type of HTR24 gene has been found on chromosome 13, which encodes for serotonin receptors, it has been linked to a 4.25-fold increase in risk of sleep bruxism. Based on this, the researchers concluded that genetic factors could contribute to the etiology of sleep bruxism [33]. The 5 HT2A gene (5-hydroxytryptamine), is responsible for circadian rhythms, maintaining arousal, and regulating muscle tone and respiration, and is a contributor for sleep bruxism. These studies show that the interaction of serotonin (HT-5) has been reported as a neurotransmitter in behavioral responses to environmental stimuli, especially in the social environment. By doing so, it has played an important role in social responses and has also been reported to be associated with stress response and coping with stress [32,34].

SLC6A4 polymorphisms are associated with sleep disorders and apnea syndrome located on chromosome 17q11.1-17q12 [35,36]. The receptors on the polymorphisms of each gene are encoded or encrypted by the same gene involved, such as rs6295 polymorphism, by the HTR1A gene itself on the 5q11.2-q13 chromosome. Similarly, the polymorphisms of each gene are encoded on their own genes. In this regard, we point out that the HTR2A gene is located on the 13q14-q21 chromosome and the HTR2C gene is on the Xq24 chromosome [3].

Conclusion

There is increasing scientific evidence suggesting the important role that Genetics plays in the occurrence of Bruxism. However the exact mechanism for the disorder and its resultant pain is largely unknown. Therefore, we conclude from this review study, inheritance has a great role in bruxism, which is most influenced by the rs6313 polymorphisms of the 5HT2A gene and the rs6313 of the HTR2A gene. Thorough studies on these genes can be useful in preventing this disorder and its complications. Future research should consider objective diagnostic methods for the diagnosis of Bruxism in order to have more reliable results and also to identify the exact genes associated with Bruxism.

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Conflict of Interest

There is no conflict of interest to declare.

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