

The Effects of Tooth Loss on the Brain

Sri Veludandi¹, Swati Chitre^{2*}

¹Michigan State University, USA

²University of Detroit Mercy School of Dentistry, USA

*Corresponding author: Swati Chitre, University of Detroit Mercy School of Dentistry, 2700 Martin Luther King Jr Blvd, Detroit, MI 48208, USA. Tel: +13134946783; Fax: +13134946781; Email: chitresd@udmercy.edu

Citation: Veludandi S, Chitre S (2017) The Effects of Tooth Loss on the Brain. Dent Adv Res 2: 141. DOI: 10.29011/2574-7347.100041

Received Date: 27 November, 2017; Accepted Date: 11 December, 2017; Published Date: 19 December, 2017

Introduction

Teeth connect with other organs in the body via nerves which aid in sensory reception and proprioception. Specifically, teeth have a correlation with parts of the brain. When observing the somatotopic representation and center of gravity of teeth in the postcentral gyrus, there is an overlap of sensory representation in the rostral-to-caudal progression. This is a possible indication that there is converging input from teeth. The center of gravity was also consistent with the sensory homonucleus in the brain [1]. Tooth loss is characterized with much impairment such as difficulty in mastication, speaking, stereognosis, and proprioception. Tooth loss also is associated with altered emotional functions [2]. In a representative sample of adults in the United States, who had six teeth or more removed experienced a comorbid of depression and anxiety [3]. Considering how tooth loss may lead to eventual nerve loss and several motor/emotional complications, this review analyzes how tooth loss affects the physical components of the brain as well as the cognitive impacts.

Objective

The objectives of this review are:

- 1) To evaluate whether tooth loss has significant physical and cognitive brain changes from analysis of literature
- 2) To determine gaps in found literature in relation to the topic being studied
- 3) to determine if this research and future research will be able to determine if tooth loss can be a predictor of certain diseases and utilize this to prevent the diseases.

Results

Tooth loss has long term changes in the brain. In rats that had their molar teeth extracted, there were sustained neuroplastic changes that lasted one to two months [4]. Specifically, this study examines general physical brain changes, specifically, white brain matter changes and Parkinson disease patients.

Physical Brain Changes Following Tooth Loss

Utilizing structural magnetic resonance imaging, voxel-wise analysis and mice with different genetic strains, volumetric differences of 160 brain regions after teeth extraction were studied. The study was done after twenty-one days on post-mortem mice, a control of sham operation and the variable of tooth extraction (3 right maxillary molar teeth were extracted) were maintained. 34 brain regions showed significant volumetric differences. These differences were in gray matter, a part of the central nervous system, which is involved in sensory perception and muscle control [5].

Decreases in Gray Matter Volume

There was a significant decrease in gray matter volume in the primary somatosensory, primary motor region, and premotor cortex [5]. The primary somatosensory processes afferent somatosensory input and contributes to movement by integrating sensory and motor signals [6]. The primary motor region is composed of neurons that are crucial components of independent finger movements. The premotor cortex is associated with learning arbitrary association and hand movement control [7]. The primary motor region and premotor region usually work in conjunction. The decrease in volume in premotor cortex can show a decrease in masticatory performance. This evaluation is accordant to findings of reduced tongue and jaw motor representation as well as diminished orofacial motor and somatosensory region excitability after tooth loss in rodents [8].

There was a decrease in gray matter volume in the basal ganglia (specifically, the striatum, globus pallidus, and nucleus accumbens) [5]. The basal ganglion is a group of the subcortical nuclei and contributes to motor control and learning and emotions [9]. The striatum, globus pallidus, and nucleus accumbens are regions that administer motivation and motor processing. The nucleus accumbens in particular, participates in reward processing and emotional memory formation. It also controls the motor recovery post injury and response to harmful stimuli in patients with chronic pain conditions [5]. The paraventricular nucleus of the hypothalamus is a significant source of projection in the nucleus accumbens

and its brain matter volume is decreased due to tooth loss [10]. The decrease in brain volume in the nucleus accumbens is correlated to a steep cognitive decline [11]. There was a decrease in brain matter volume in the frontal association cortex. This area is important for processing inputs to the cerebral cortex and motor planning, working memory, and problem-solving behaviors [5].

Increases in Gray Matter Volume

There was an increase in brain matter volume in the temporal limbic forebrain regions- specifically the amygdala, subcortical sensory and motor nuclei [5]. The amygdala regulates response to fear and learning/ associative processing [12]. The entorhinal cortex, which is located in the medial temporal lobe, relay object and spatial information to the hippocampus, is also increased in response to tooth loss [13]. These brain regions are usually enlarged in response to fearful conditions. The increased volume in the regions due to tooth loss may be associated excessive fear or anxiety [5].

There was an increase in gray matter volume in mammillary bodies and the anterior association cortex. The maxillary bodies play an important part in consolidating memory [5]. The anterior association cortex is associated with memory and cognitive functions [14].

Tooth loss affects nerves and brainstem regions. There was increased brain matter volume in brainstem regions including: the trigeminal motor nucleus, facial nucleus and nerve, the trigeminal sensory and solitary tract nuclei, pons, superior and inferior olivary complexes, and the cuneate nucleus. These regions are responsible for sensory and motor functions [5]. Regarding the trigeminal nerve, which provides sensation to the face and is the motor nerve associative mastication muscles [15], there is a decrease in brain matter volume in the trigeminal motor nucleus. However, there is an increase in the trigeminal spinal tract nucleus. Additionally, after a pulpectomy or acute dental stimulation, the neurons in the trigeminal brainstem sensory nuclei have increased excitability while neurons of the orofacial sensorimotor cortex have a decreased excitability [5].

Tooth Loss Association with Brain White Matter Change

This study analyzes tooth loss on the terms of it being a cerebrovascular risk factor. There is analysis of brain white matter change and silent infarction association with tooth loss. White brain matter change may be correlated with defects in memory. Humans were subjects and data were collected through interviews and evaluations (Brain CT). Variables such as hypertension, smoking, diabetes, age, and hyperlipidemia were taken into consideration as potential covariates in the evaluations. White brain matter change was defined as somewhat hypodense areas with diameters greater or equal to 5 mm in the subcortical area. The P value for significance was 0.01 for the analysis. The analysis was divided into subjects with 0-5 lost teeth, 6-10 lost teeth, and greater than 10 lost teeth. In an unadjusted analysis, the white brain matter/silent infarction subject odds ratio for 6-10 lost teeth was 2.3 with a 96 % confidence interval and p significance of 0.006 in comparison

to 0-5 lost teeth subjects. The white brain matter/silent infarction subject odds ratio for more than 10 lost teeth was 4.2 with a 95% confidence interval and P significance of 0.001 when compared to subjects with 0-5 lost teeth. Then an adjustment was made to the odds ratio to take in the effects of the covariates. The odds ratio was 1.7 for subjects with 6-10 lost teeth with a confidence interval of 95% and a P significance of 0.12. The odds ratio for

Subjects with more than 10 lost teeth were 3.9 with a 95% confidence interval and P significance less than 0.001. Overall, subjects that had white brain matter changes and/or silent infarctions had greater lost teeth than subjects without those conditions and had a normal brain CT. Although this study examines white brain matter change, there is an increased significance of tooth loss when regarded the covariates (age, hypertension, diabetes) [16]. This limits the actual determination as to if white brain matter change and tooth loss are strongly correlated.

Tooth Loss Association in Parkinson Disease Patients

Parkinson's disease pertains to loss of dopaminergic neurons. This causes behavior dysfunction and deterioration of movement. The study group had patients affected by Parkinson's disease and patients who were not affected by Parkinson's disease who went through periodontal deep socket investigation with the utilization of a dental probe and X-rays. Per patient with Parkinson's disease, the average number of missed teeth was 13 (total record of 330 teeth) with a range of 10-22 teeth. Per patient without Parkinson's disease, the average number of missed teeth was 9 (total record of 418 teeth) with a range of 8-23 teeth. The patients with Parkinson's disease had a greater number of lost teeth than patients without Parkinson's disease. The study also evaluates the incidence of periodontal disease, which is a gum inflammation that results in serious damage of soft tissue and bone support, its caused by plaque and contributing factors can be hormonal, diabetes, medications is increased in patients with Parkinson's disease and untreated caries had no significant difference between both groups [2]. This study relates the cognitive and movement deficiency consequences of Parkinson's disease as possible rationale for increased tooth loss, suggesting impaired manual dexterity may negatively influence oral hygiene and cause tooth loss. This study does not directly correlate tooth loss as a factor of loss of dopaminergic neurons which is associated with Parkinson's disease.

Conclusion

Teeth loss increases or decreases gray brain matter in specific regions of the brain which all control different aspects of brain function. There was an increase in gray matter volume for regions that controlled response to fear and learning, memory consolidation, and the trigeminal spinal tract nucleus. Regions of the brain that control movement, motor processing/control and emotions, working memory and problem-solving behavior, and the trigeminal motor nucleus had a decrease in gray matter volume. The finding of the study can be employed for tooth loss prevention and treatment [5]. Tooth loss may be utilized as a predictor of white brain matter

and silent infarction. Despite incomplete correlation and data, this study can be applied to educate patients on dental care and manage the covariates as well [16]. Tooth loss and incidence of periodontal disease is highly correlated to patients with Parkinson's disease. This can be used during dental checkups and take extra care in oral health of Parkinson's disease patients and to educate patients [2]. These studies show a correlation between tooth loss and brain changes and function, but do not explicitly state that tooth loss can predict brain change and disease onset. Further studies can be executed to examine if tooth loss is a predictor, this can be best done in a long-term study. Studies can be done over the course of a subjects' lifetime to determine the correlation between number of lost adult teeth to the type of diseases accumulated through the lifetime and what parts of the brain regions are affected. Another study can be conducted to determine if tooth loss can be treated. Osseoperception may be due to the primary sensorimotor cortex of the brain and the brain may compensate loss of region matter by engaging other parts of the brain [4]. Furthermore, the study can examine if the treatment will be able to restore original nerve conduction and revert brain changes.

References

1. Miyamoto JJ, Honda M, Saito DN, Okada T, Ono T, et al. (2006) The Representation of the Human Oral Area in the Somatosensory Cortex: A Functional MRI Study. *Cereb Cortex* 16: 669-675.
2. Ciccù M, Risitano G, Lo Giudice G, Bramanti E (2012) Periodontal Health and Caries Prevalence Evaluation in Patients Affected by Parkinson's Disease. *Parkinson's Disease* 2012: 541908.
3. Wiener RC, Wiener MA, McNeil DW (2015) Comorbid depression/anxiety and teeth removed: Behavioral Risk Factor Surveillance System 2010. *Community Dentistry and Oral Epidemiology* 43: 433-443.
4. Yan C, Ye L, Zhen J, Ke L, Gang L (2008) Neuroplasticity of edentulous patients with implant-supported full dentures. *European Journal of Oral Sciences* 116: 387-393.
5. Avivi-Arber L, Seltzer Z, Friedel M, Lerch JP, Moayed M, et al. (2016) Widespread Volumetric Brain Changes following Tooth Loss in Female Mice. *Frontiers in Neuroanatomy* 10: 121.
6. Borich MR, Brodie SM, Gray WA, Ionta S, Boyd LA (2015) Understanding the role of the primary somatosensory cortex: Opportunities for rehabilitation. *Neuropsychologia* 79: 246-255.
7. Chouinard PA, Paus T (2006) The Primary Motor and Premotor Areas of the Human Cerebral Cortex. *The Neuroscientist* 12: 143-152.
8. Avivi-Arber L, Lee JC, Sood M, Lakschevitz F, Fung M, et al. (2015) Long-term neuroplasticity of the face primary motor cortex and adjacent somatosensory cortex induced by tooth loss can be reversed following dental implant replacement in rats. *J Comp Neurol* 523: 2372-2389.
9. Lanciego JL, Luquin N, Obeso JA (2012) Functional Neuroanatomy of the Basal Ganglia. *Cold Spring Harbor Perspectives in Medicine* 2: a009621.
10. Kirouac GJ (2015) Placing the paraventricular nucleus of the thalamus within the brain circuits that control behavior. *Neuroscience & Biobehavioral Reviews* 56: 315-329.
11. de Jong LW, Wang Y, White LR, Yu B, van Buchem MA, et al. (2012) Ventral striatal volume is associated with cognitive decline in older people: a population based MR-study. *Neurobiology of Aging* 33: 424. e1-424.10.
12. Ressler KJ (2010) Amygdala Activity, Fear, and Anxiety: Modulation by Stress. *Biological Psychiatry* 67: 1117-1119.
13. Schultz H, Sommer T, Peters J (2015) The Role of the Human Entorhinal Cortex in a Representational Account of Memory. *Frontiers in Human Neuroscience* 9: 628.
14. Goodwin SJ, Blackman RK, Sakellaridi S, Chafee MV (2012) Executive Control Over Cognition: Stronger and Earlier Rule-Based Modulation of Spatial Category Signals in Prefrontal Cortex Relative to Parietal Cortex. *The Journal of Neuroscience* 32: 3499-3515.
15. Tewfik TL (2016) Trigeminal Nerve Anatomy: Gross Anatomy, Branches of the Trigeminal Nerve, Microscopic Anatomy. *Medscape*.
16. Minn YK, Suk SH, Park H, Cheong JS, Yang H, et al. (2013) Tooth Loss Is Associated with Brain White Matter Change and Silent Infarction among Adults without Dementia and Stroke. *Journal of Korean Medical Science* 28: 929-933.