Original Article

Aging-related Alteration of Trigeminal Ganglion Structures; A Histologic Study of Neuronal Population, Satellite Ganglionic Cells and Vascular Bed

Shahriar Ahmadpour and Arman Behrad

Neuroscience Educational and Research Center (NERC). Department of Anatomy, Faculty of Medicine, North Khorasan University of Medical Sciences, Bojnurd, Iran

ABSTRACT

Background: Aging is associated with signs that are similar to peripheral neuropathy and age-related decline in the peripheral sensory system and proprioceptive functions. Trigeminal ganglion (TG) is one of the sensory ganglion providing sensory information from the orofacial region. Ultrasstruchral alteration in neuronal population of TG has been reported.

Aim of Work: Examine the effects of aging on neuronal population, distribution, and satellite ganglionic cells (SGCs) and compare with the TG of young animal.

Materials and Methods: Old male Wistar rats (32 weeks old) (250±20gr) and young male (4weeks old) (150±20) were deeply anesthetized with chloroform. To avoid inadvertent mechanical dark neuron formation, each animal was transcardially perfused with 500ml of 4% paraformaldehyde in0.1 M phosphate-buffered saline. The skull cap was cut open and the TG was exposed and removed carefully. The sections were selected according to the systematic random sampling(SRS) and stained with H&E. From each section 10 fields(200×160µm) were selected randomly and the profile of neurons, neuronal shapes, SGCs, and the number of capillary profiles were studied.

Results: The measured profile of neurons in the TG of young (17.11± 4.8) and old animals (15.69±4.4) showed a meaningful level of differences(p<0.05). The comparison between the number of SGCs in young animals (16±8.8) and old animals (20±7.12) showed no significant level of difference(p>0.05). The number of capillary profiles in the TG of young animals (7±2.73) showed a meaningful difference with those of old animals (3.8±0.83) (p<0.05).

Conclusion: With aging neuro-glial population and vascular bed of TG undergo a series of quantitative and qualitative alterations. Precisely aging is associated with decrease in neuronal size and vascular bed while the number of SGCs remains without significant changes. Aging leads to changes in neuronal distribution and likely morphologic alterations in SGCs subpopulation.

Received: 14 February 2021, **Accepted:** 23 February 2021

Key Words: Aging, satellite, trigeminal ganglion.

Corresponding Author: Shahriar Ahmadpour, PhD, Neuroscience Educational and Research Center (NERC). Department of Anatomy, Faculty of Medicine, North Khorasan University of Medical Sciences, Bojnurd, Iran, **Tel.**: +989021232016, **E-mail:** Shahahmadpour@gmail.com

ISSN: 1110-0559, Vol. 45, No.1

INTRODUCTION

Aging is associated with signs that are similar to peripheral neuropathy[1]. Studies have documented age-related decline in the peripheral sensory system and proprioceptive functions[2-8]. One of the main reasons for the age-related decline in sensory functions has been considered to be the loss of primary sensory neurons[9]. Trigeminal ganglion (TG) is one of the sensory ganglion providing sensory information from the orofacial region. The trigeminal ganglion occupies Meckel's caves near the apex of the petrous part of the temporal bone. It comprises pseudounipolar neurons from the ophthalmic, maxillary, and mandibular divisions of the trigeminal nerve[10]. TG is composed of neurons and glial cells. The pseudounipolar neurons have been classified according to their size into the large, light A-type, and small B –type cells. Large A-type cells contain scattered clumps of Nissl substance and B –type cells contain coarser clump of Nissl substance[11,12]. Studies have shown that each type involves different sensory modalities[13,14]. Glial cells of TG which surround these neurons directly modulate neuronal function and activities[15,16]. Interestingly, neuroglia interactions are involved in inflammation and pain[17]. TG consists of two types of glial cells including satellite glial cells (SGCs) and Schwann cells. SGCs surround neuronal cell bodies in the peripheral ganglia, carry numerous neuroactive molecules such as adenosine triphosphate (ATP) and bradykinin. SGCs directly influence neuronal activity by controlling the microenvironment in the ganglion[16,18]. A previous study has provided compelling evidence about the ultrastructural changes in the neurons of TG in the aged animals[19]. Additionally, recent findings indicate the possible role of TG and neuroinflammation in the brain regions involved in memory[20]. The present histomorphometric study was conducted to examine the effects of aging on neuronal population, distribution, SGCs and vascular structure.

 DOI: 10.21608/ejh.2021.63142.1432

METHODS AND MATERIALS

All the experiments in this study were conducted in the educational and research laboratory of neuroscience (ERLN)of North Khorasan University of Medical Sciences (NKUMS). Old male Wistar rats (32 weeks old) (250±20g) and young male (4weeks old) (150±20 g) were obtained from the experimental animal facility of ERLN. All procedures were conducted according to the Institutional Animal Ethics Committee. The animals were kept in cages with not more than three animals in one cage. They were maintained at 12/12 light and dark cycles with free access to water and food. After deep anesthesia with chloroform. To avoid inadvertent mechanical dark neuron formation, each animal was transcardially perfused with 500ml of 4% paraformaldehyde in0.1 M phosphate-buffered saline[21]. The skull cap was cut open and the TG was exposed and removed carefully. Paraffin-embedded sections of 10µm thickness were cut on a microtome. The sections were selected according to the systematic random sampling (SRS) and stained with H&E. The TG was divided into three arbitrary concentric zones. From each section 10 fields (200×160µm) were selected randomly and the profile of neurons, SGCs, and the number of capillary profiles were counted according to the previous studies with the modified stereological method[22]. The diameters of the neurons from selected fields were measured using an Olympus microscope equipped with Cellsens software.

Statistics

All data are expressed as mean ± SD. Statistical comparison between the two groups was made using the Student t-test. The comparison between the right and left sides TG were made using Kruskal-Wallis and Mann-Whitney test. Statistically, a significant difference was accepted at the *P-value*<0.05 level (21,26)

RESULTS

Size and morphology of the neuron

The measured profile of neurons in the TG of young (17.11± 4.8) and old animals (15.69±4.4) showed a meaningful level of differences(p<0.05). Because of the complicated cytoarchitectonic picture of TG, we applied the hodologous method to classify neurons. Following types of neurons have been differentiated according to their shape and the size of their perikarya (Figures 1-3):

- Light – large sized with a round contour
- Light- medium-sized with a round contour
- Light-small sized with a round contour
- Dark- large-sized polygonal
- Dark- medium-sized polygonal
- Dark –small-sized polygonal
- Dark-small sized with a round contour
- Dark- medium-sized with a round contour
- Dark elongated
- Light elongated

In young animals, 5-6 layers of mainly light –large-sized (Type-A) with round contour and granular cytoplasm were noticed. Dispersed dark neurons (Type-B) with different size and forms among the type –A neurons were found. In the deeper zone, dark polygonal neurons of different sizes were the dominant microscopic feature. In old animals, mediums sized dark polygonal were dominant in peripheral zones, and in some regions, the peripheral strata were reduced to one layer of dark polygonal neurons.

Morphology and counting the SGCs

Following types of SGC were differentiated according to their nucleus shapes: dark round, dark, and light elliptical forms (Figures 4-6). The last form was only seen in young animals. The comparison between the number of SGCs in young animals (16±8.8) and old animals (20±7.12) showed no significant level of difference (p>0.05).

The counting of capillary profile

The number of capillary profiles in the TG of young animals (7±2.73) showed a meaningful difference with those of old animals (3.8±0.83) (p<0.05).

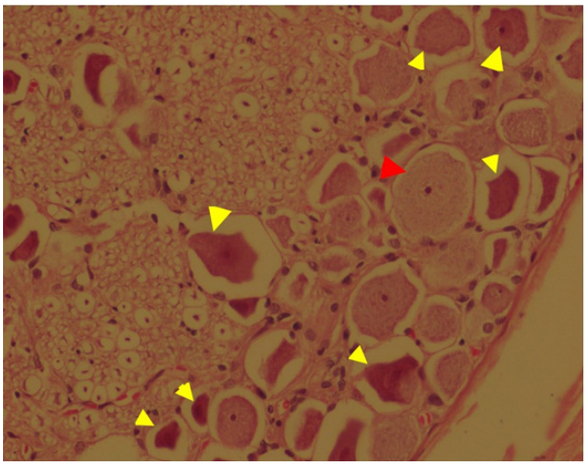

Fig. 1: Dark (type-B) neurons (yellow arrowheads) with different shapes and sizes are seen in peripheral region of TG . A light (type –A) neuron with light granular cytoplasm with prominent nucleolus (red arrowhead) among the type -B neuron.X40

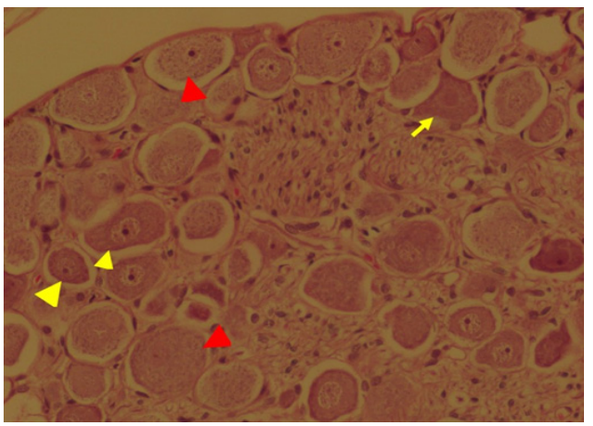

Fig. 2: Neuronal population of TG.Light (type-A) neurons(red arrowheads) with round contour and granular cytoplasm are scattered among elongated - round dark (yellow arrowheads) and medium-sized dark polygonal neurons (yellow arrow).X40

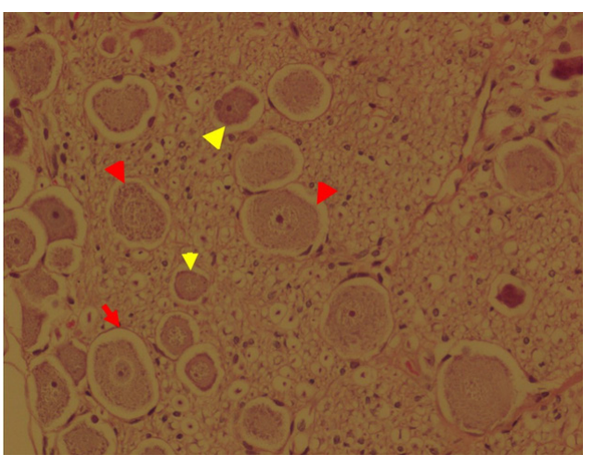

Fig. 3: Light neurons of TG with different size and shape. Light (type-A) neurons(red arrowheads) with round contour, elongated light neuron (arrow) and small light and dark neurons with round contour(yellow arrowheads) are seen .X40

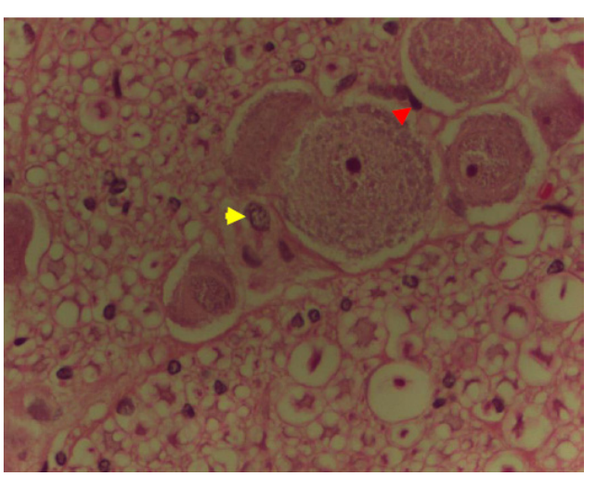

Fig. 4: Satellite ganglionic cells in the TG of old animals. Dense dark SGC (red arrowhead) and SGC with light larger appearance around the type -A neuron (yellow arrowhead). X40

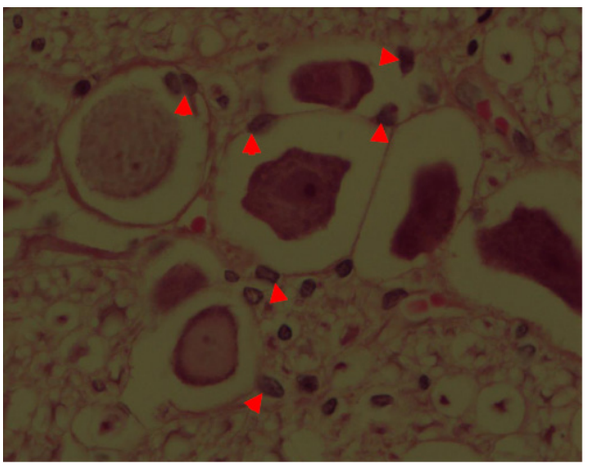

Fig. 5: Satellite ganglionic cells in the TG of young animals. SGCs (red arrowhead) around the different types of TG neurons. X40

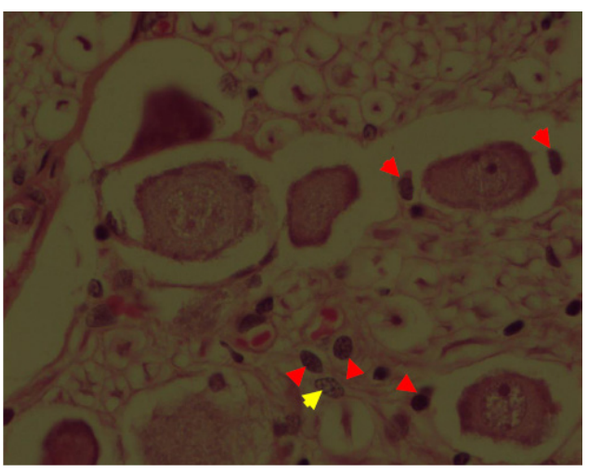

Fig. 6: Satellite ganglionic cells in the TG of the young animals. Dense dark SGC (red arrowhead) and SGC with a light larger appearance (yellow arrowhead). X40

DISCUSSION

In this study, we investigated whether aging is associated with neuroglia and vascular changes in the trigeminal ganglion. The results of our study showed that aging leads to a decrease in the size of neurons. Another set of our results revealed that SGCs are a heterogeneous population of the glial cells and aging is not associated with a decrease in the number of SGCs.Additionally, our findings revealed that aging leads to a decrease in the number of capillaries of TG. Besides the results of this study showed that aging is associated with the change in the cytoarchitecture of TG. Aging as a natural process is associated with sensory impairment. Recent studies have shown that age-related sensory decline affects multiple modalities[1,3-9]. TG is the main sensory relay station of orofacial regions. Structurally TG is composed of light type A-neuron, dark type B neurons, and glial cells. Glial cells of TG consist of Schwan cells and satellite ganglionic cells (SGCs). The results of this study are consistent with the previous report and suggest age-related alterations in the

TG. Seress *et al*[19] reported ultrastructural alteration in the mitochondria of B-type cells and a decrease in cell number in the TG of the old animals. They showed mitochondria swelling and crista disruption in only type B (not A-type, not SGCs) neurons. Because only B-type neurons are sensitive to different noxae, it seems aging to accelerate the process that leads to mitochondrial damage[23]. Various noxious agents such as oxidative stress and free radicals can induce mitochondrial damage and subsequently neuronal degeneration[24]. The result in old animals showed scattered degenerating neurons particularly small to medium B- type neurons in aging animals, while no degenerating neurons were found in the young animal group. Whereas we did not perform counting of the degenerating neurons, this sort of data should be interpreted cautiously. In another study, Biedenbach[25] showed that aging leads to changes in the size of neurons in TG. Interestingly our results in aged animals showed that the smaller size of the neuron is not coincident with a decrease in SGCs number. SGCs are known as glial cells that express GFAP proteins. SGCs are involved in maintaining ionic homeostasis and neuroinflammatory processes[16-18]. Our light microscopic findings indicate SGCs are a heterogeneous population and aging processes may affect particular types of SGCs. These results are supported by the study of Tongtako *et al* (2017)[26]. Their findings showed that SGCs represents an exceptional, intermediate glial cell population with phenotypical characteristics of oligodendrocytes and astrocytes and might possess intrinsic regenerative capabilities in *vivo*. Additionally, the quantitative results of the number of SGCs in the old animals can be considered as a compensatory mechanism in response to neuronal loss[26,28]. It seems through aging a dynamic adaptive change occurs in the neuro-glial population and cytoarchitecture of TG. The cytoarchitecture of TG has been investigated since Cajal[29]. Komer classified the sensory neurons of TG into four main types including large neurons with light cytoplasm, large with granulation, small with light, and small with dark cytoplasm[30]. Krastev *et al* (2013)[29] differentiate seven types of neurons according to their shape and size of their perikarya including; large light, medium light, small light, medium dark, small dark, elongated and polygonal. Our findings are also in line with the previous reports, indicating the heterogeneity of neuronal papulation of the TG. The diversity in size of neurons in the TG reflects their specific involvement in different sensory modalities[31]. To our knowledge, less has been dealt with the dark stained polygonal neurons in the TG. Considering the dominant presence of dark stained in the aging animals, it may be suggestive of the quiescent state of neurons. Previous studies have reported dark stained neurons in various pathological conditions including epilepsy, ischemia, cholestasis, and aging[32,33]. Therefore, it could be assumed that dark stained polygonal neurons likely represent a hibernate state[34], which in turn may reflect the plasticity of the TG[35]. Furthermore, our quantitative analysis showed that aging leads to a decrease in the number of capillary profiles in TG. The microvascular bed of TG provides blood supply to the sensitive ganglionic cells[36,37]. The TG vascular topography and microvascular bed have been implicated in the pathogenesis of TG related neurologic disorders such as trigeminal neuralgia(TN) and migraine[37]. Interestingly TN is more common in elderly patients and[38], therefore it would be reasonable to assume that adaptive aging-related cyto-vascular changes inadvertently increase the risk TG related pathologies. Further studies based on stereology and TEM methods are recommended to reveal further details of the TG cyto-structural changes in aging.

CONCLUSION

With aging neuroglial population and vascular bed of TG undergo a series of quantitative and qualititative alterations. Precisely aging is associated with decrease in neuronal size and vascular bed while the number of SGCs remains without significant changes. Additonally aging lead to changes in neuronal distribution and likely morphologic alterations in SGC subpopulation.

CONFLICT OF INTERESTS

There are no conflicts of interest.

REFERENCES

1. Cavazzana A, Röhrborn A, Garthus-Niegel S, Larsson M, Hummel T, Croy I. Sensory-specific impairment among older people. An investigation using both sensory thresholds and subjective measures across the five senses. PLoS One. 2018 Aug 27;13(8):e0202969. doi: 10.1371/journal.pone.0202969. PMID: 30148857; PMCID: PMC6110574.
2. Heft MW, Robinson ME. Somatosensory function in old age. J Oral Rehabil. 2017 Apr;44(4):327-332. doi: 10.1111/joor.12488. Epub 2017 Feb 17. PMID: 28130938.
3. Heine C, Browning C. The communication and psychosocial perceptions of older adults with sensory loss: A qualitative study. Ageing and Society. 2004;24(1):113 - 130.
4. Hummel T, Kobal G, Gudziol H, Mackay-Sim A. Normative data for the "Sniffin' Sticks" including tests of odor identification, odor discrimination, and olfactory thresholds: an upgrade based on a group of more than 3,000 subjects. Eur Arch Otorhinolaryngol. 2007 Mar;264(3):237-43. doi: 10.1007/s00405-006-0173-0. Epub 2006 Sep 23. PMID: 17021776.
5. Kaneda H, Maeshima K, Goto N, Kobayakawa T, Ayabe-Kanamura S, Saito S. Decline in taste and odor discrimination abilities with age, and relationship between gustation and olfaction. Chem Senses. 2000 Jun;25(3):331-7. doi: 10.1093/chemse/25.3.331. PMID: 10866991.
6. Lautenbacher S, Strian F. Similarities in age differences in heat pain perception and thermal sensitivity. Funct Neurol. 1991 Apr-Jun;6(2):129-35. PMID: 1916455.

7. Roth TN. Aging of the auditory system. Handbook of clinical neurology. 129: Elsevier; 2015. p. 357-73.
8. Seubert J, Laukka EJ, Rizzuto D, Hummel T, Fratiglioni L, Bäckman L, Larsson M. Prevalence and Correlates of Olfactory Dysfunction in Old Age: A Population-Based Study. J Gerontol A Biol Sci Med Sci. 2017 Aug 1;72(8):1072-1079. doi: 10.1093/gerona/glx054. PMID: 28444135; PMCID: PMC5861894..
9. Lindholm T, Risling M, Carlstedt T, Hammarberg H, Wallquist W, Cullheim S, Sköld MK. Expression of Semaphorins, Neuropilins, VEGF, and Tenascins in Rat and Human Primary Sensory Neurons after a Dorsal Root Injury. Front Neurol. 2017 Feb 21;8:49. doi: 10.3389/fneur.2017.00049. PMID: 28270793; PMCID: PMC5318460.
10. Messlinger K, Russo AF. Current understanding of trigeminal ganglion structure and function in headache. Cephalalgia. 2019 Nov;39(13):1661-1674. doi: 10.1177/0333102418786261. Epub 2018 Jul 10. PMID: 29989427; PMCID: PMC7007999.
11. Dodick D, Silberstein S. Central sensitization theory of migraine: clinical implications. Headache. 2006 Nov;46 Suppl 4:S182-91. doi: 10.1111/j.1526-4610.2006.00602.x. PMID: 17078850.
12. Lawson SN, Waddell PJ. Soma neurofilament immunoreactivity is related to cell size and fibre conduction velocity in rat primary sensory neurons. J Physiol. 1991 Apr;435:41-63. doi: 10.1113/jphysiol.1991.sp018497. PMID: 1770443; PMCID: PMC1181449..
13. McNeill DL, Coggeshall RE, Carlton SM. A light and electron microscopic study of calcitonin gene-related peptide in the spinal cord of the rat. Exp Neurol. 1988 Mar;99(3):699-708. doi: 10.1016/0014-4886(88)90186-0. PMID: 3257735..
14. Murata Y, Onda A, Rydevik B, Takahashi I, Takahashi K, Olmarker K. Changes in pain behavior and histologic changes caused by application of tumor necrosis factor-alpha to the dorsal root ganglion in rats. Spine (Phila Pa 1976). 2006 Mar 1;31(5):530-5. doi: 10.1097/01.brs.0000201260.10082.23. PMID: 16508546.
15. Watkins LR, Maier SF. Beyond neurons: evidence that immune and glial cells contribute to pathological pain states. Physiol Rev. 2002 Oct;82(4):981-1011. doi: 10.1152/physrev.00011.2002. PMID: 12270950.
16. Watkins LR, Milligan ED, Maier SF. Glial activation: a driving force for pathological pain. Trends Neurosci. 2001 Aug;24(8):450-5. doi: 10.1016/s0166-2236(00)01854-3. PMID: 11476884.
17. Hanani M. Satellite glial cells in sensory ganglia: from form to function. Brain Res Brain Res Rev. 2005 Jun;48(3):457-76. doi: 10.1016/j.brainresrev.2004.09.001. PMID: 15914252.
18. Chudler EH, Anderson LC, Byers MR. Trigeminal ganglion neuronal activity and glial fibrillary acidic protein immunoreactivity after inferior alveolar nerve crush in the adult rat. Pain. 1997 Nov;73(2):141-149. doi: 10.1016/S0304-3959(97)00088-2. PMID: 9415499.
19. Seress L, Szoke E, Czéh G. Age-related mitochondrial damage in the B-type cells of the rat trigeminal ganglia. Acta Biol Hung. 2002;53(1-2):167-75. doi: 10.1556/ABiol.53.2002.1-2.16. PMID: 12064769.
20. Kubo KY, Murabayashi C, Kotachi M, Suzuki A, Mori D, Sato Y, Onozuka M, Azuma K, Iinuma M. Tooth loss early in life suppresses neurogenesis and synaptophysin expression in the hippocampus and impairs learning in mice. Arch Oral Biol. 2017 Feb;74:21-27. doi: 10.1016/j.archoralbio.2016.11.005. Epub 2016 Nov 6. PMID: 27846401.
21. Ahmadpour SH, Haghir H. Diabetes mellitus type 1 induces dark neuron formation in the dentate gyrus: a study by Gallyas' method and transmission electron microscopy. Rom J Morphol Embryol. 2011;52(2):575-9. PMID: 21655645.
22. Royet JP. Stereology: a method for analyzing images. Prog Neurobiol. 1991;37(5):433-74. doi: 10.1016/0301-0082(91)90009-p. PMID: 1754688.
23. Barazzoni R, Short KR, Nair KS. Effects of aging on mitochondrial DNA copy number and cytochrome c oxidase gene expression in rat skeletal muscle, liver, and heart. J Biol Chem. 2000 Feb 4;275(5):3343-7. doi: 10.1074/jbc.275.5.3343. PMID: 10652323.
24. Richter C, Park JW, Ames BN. Normal oxidative damage to mitochondrial and nuclear DNA is extensive. Proc Natl Acad Sci U S A. 1988 Sep;85(17):6465-7. doi: 10.1073/pnas.85.17.6465. PMID: 3413108; PMCID: PMC281993.
25. Biedenbach MA, Kalu DN, Herbert DC. Effects of aging and food restriction on the trigeminal ganglion: a morphometric study. Mech Ageing Dev. 1992 Sep;65(2-3):111-25. doi: 10.1016/0047-6374(92)90028-c. PMID: 1434941.
26. Tongtako W, Lehmbecker A, Wang Y, Hahn K, Baumgärtner W, Gerhauser I. Canine dorsal root ganglia satellite glial cells represent an exceptional cell population with astrocytic and oligodendrocytic properties. Sci Rep. 2017 Oct 24;7(1):13915. doi: 10.1038/s41598-017-14246-7. PMID: 29066783; PMCID: PMC5654978.
27. Gunjigake KK, Goto T, Nakao K, Kobayashi S, Yamaguchi K. Activation of satellite glial cells in rat trigeminal ganglion after upper molar extraction. Acta Histochem Cytochem. 2009 Oct 30;42(5):143-9. doi: 10.1267/ahc.09017. Epub 2009 Sep 15. PMID: 19918323; PMCID: PMC2775105.

28. Nascimento DS, Castro-Lopes JM, Moreira Neto FL. Satellite glial cells surrounding primary afferent neurons are activated and proliferate during monoarthritis in rats: is there a role for ATF3? PLoS One. 2014 Sep 23;9(9):e108152. doi: 10.1371/journal.pone.0108152. PMID: 25247596; PMCID: PMC4172763.

29. Krastev DS, Apostolov A. Cytoarchitectonic study of the trigeminal ganglion in humans. Clujul Med. 2013;86(2):97-101. Epub 2013 May 9. PMID: 26527926; PMCID: PMC4462427.

30. Luo PF, Wang BR, Peng ZZ, Li JS. Morphological characteristics and terminating patterns of masseteric neurons of the mesencephalic trigeminal nucleus in the rat: an intracellular horseradish peroxidase labeling study. J Comp Neurol. 1991 Jan 8;303(2):286-99. doi: 10.1002/cne.903030210. PMID: 2013641.

31. Pk S, Ramar S, Al Dajah SB.. Morphological study of nociceptive neurons in the trigeminal ganglion. JIJHRS. 2017;5(1).

32. Kherani ZS, Auer RN. Pharmacologic analysis of the mechanism of dark neuron production in cerebral cortex. Acta Neuropathol. 2008 Oct;116(4):447-52. doi: 10.1007/s00401-008-0386-y. Epub 2008 Jun 3. PMID: 18521615.

33. Zimatkin SM, Bon' EI. Dark Neurons of the Brain. Neurosci Behav Physi .2018;48, 908–912

34. Horowitz JM, Horwitz BA. Extreme Neuroplasticity of Hippocampal CA1 Pyramidal Neurons in Hibernating Mammalian Species. Front Neuroanat. 2019 Feb 13;13:9. doi: 10.3389/fnana.2019.00009. PMID: 30814935; PMCID: PMC6381046.

35. Lynds R, Lyu C, Lyu GW, Shi XQ, Rosén A, Mustafa K, Shi TJS. Neuronal plasticity of trigeminal ganglia in mice following nerve injury. J Pain Res. 2017 Feb 9;10:349-357. doi: 10.2147/JPR.S120092. PMID: 28223844; PMCID: PMC5310634.

36. Ćetković M, Štimec BV, Mucić D, Dožić A, Ćetković D, Reçi V, Çerkezi S, Ćalasan D, Milisavljević M, Bexheti S. Arterial supply of the trigeminal ganglion, a micromorphological study. Folia Morphol (Warsz). 2020;79(1):58-64. doi: 10.5603/FM.a2019.0062. Epub 2019 Jul 8. PMID: 31282551.

37. Smoliar E, Smoliar A, Sorkin L, Belkin V. Microcirculatory bed of the human trigeminal nerve. Anat Rec. 1998 Feb;250(2):245-9. doi: 10.1002/(SICI)1097-0185(199802)250:2<245::AID-AR14>3.0.CO;2-O. PMID: 9489785.

38. Wijeratne T, Tang HM, Crewther D, Crewther S. Prevalence of Migraine in the Elderly: A Narrated Review. Neuroepidemiology. 2019;52(1-2):104-110. doi: 10.1159/000494758. Epub 2019 Jan 9. PMID: 30625476..

الملخص العربى

التعديلات المرتبطة بالشيخوخة في الهياكل العقدية الثلاثية التوائم؛ دراسة نسيجية لسكان الخلايا العصبية والخلايا العقدية الساتلية وسرير الأوعية الدموية

شهريار احمدپور، آرمان بهراد

مركز تعليم وبحوث علم الأعصاب (NERC)، قسم التشريح، كلية الطب،

جامعة شمال خراسان للعلوم الطبية، بوجنورد، إيران

الخلفية: الشيخوخة مرتبطة بعلامات مشابهة لاعتلال الأعصاب المحيطية والتدهور المرتبط بالعمر في الجهاز الحسي المحيطي ووظائف التحفيز التحسسي. العقدة الثلاثية التوائم (TG) هي إحدى العقدة الحسية التي توفر معلومات حسية من منطقة الفم والوجه. تم الإبلاغ عن التغيير فوق الصدري في السكان العصبية من TG.

هدف العمل: فحص آثار الشيخوخة على مجموعات الخلايا العصبية ، والتوزيع ، والخلايا العقدية الساتلية (SGCs) ومقارنتها مع TG للحيوان الصغير.

المواد والطرق: تم تخدير ذكور فئران Wistar القديمة (٣٢ أسبوعًا) (٢٥٠ ± ٢٠gr) والشباب الذكور (٤ أسابيع) (١٥٠ ± ٢٠) بعمق باستخدام الكلوروفورم. لتجنب تكوين الخلايا العصبية الميكانيكية الداكنة عن غير قصد ، تم ترطيب كل حيوان عبر القلب بـ ٥٠٠ مل من ٤ ٪ بارافورمالدهيد في ٠,١ مولار من محلول ملحي مخزّن بالفوسفات. تم قطع غطاء الجمجمة وفتح TG وإزالته بعناية. تم اختيار المقاطع وفقًا لأخذ العينات العشوائي المنهجي (SRS) وتم تلوينها بـ H&E. من كل قسم تم اختيار ١٠ حقول (٢٠٠ × ١٦٠ ميكرومتر) بشكل عشوائي وتم دراسة ملف تعريف الخلايا العصبية والأشكال العصبية و SGCs وعدد الملامح الشعرية.

النتائج: أظهر المظهر الجانبي المقاس للخلايا العصبية في TG للحيوانات الصغيرة (١٧,١١ ± ٤,٨) والحيوانات القديمة (١٥,٦٩ ± ٤,٤) مستوى ذي مغزى من الاختلافات (p <0.05). لم تظهر المقارنة بين عدد SGCs في الحيوانات الصغيرة (١٦ ± ٨,٨) والحيوانات القديمة (٢٠ ± ٧,١٢) أي فرق معنوي (p> 0.05). أظهر عدد الملامح الشعرية في TG للحيوانات الصغيرة (٧ ± ٢,٧٣) فرقًا ذا مغزى مع تلك الخاصة بالحيوانات القديمة (٣,٨ ± ٠,٨٣) (p <0.05).

الخلاصة: مع شيخوخة السكان العصبية الدبقية وسرير الأوعية الدموية من TG يخضعون لسلسلة من التغييرات الكمية والنوعية. ترتبط الشيخوخة بدقة بانخفاض حجم الخلايا العصبية وسرير الأوعية الدموية بينما يظل عدد الخلايا الجذعية السرطانية بدون تغيرات كبيرة. الشيخوخة تؤدي إلى تغييرات في توزيع الخلايا العصبية والتغيرات المورفولوجية المحتملة في مجموعات سكانية SGCs الفرعية