

Original Research Article

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DIFFERENTIAL SUSCEPTIBILITY OF MOTION SICKNESS BETWEEN TWO PHENOTYPICALLY DIFFERENT POPULATIONS OF ASANSOL, WEST BENGAL, INDIA.

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ABSTRACT: Motion sickness can occur when the sensory inputs about body position contradict what is expected. It can be provoked by abrupt changes in movement, such as occur during bumpy rides, turbulent flights, and rough seas. Signs of motion sickness include pallor, restlessness, and cold sweat. In later stages, nausea, excessive salivating, and vomiting occur. It involves both genetic and environmental cues. The precise etiology of motion sickness remains a mystery. The classic “sensory conflict” explanation suggests motion sickness is triggered when the brain interprets sensory messages regarding movement as inharmonious. The present paper aims to characterize the susceptibility frequency of motion sickness among the non-nepali and nepali population of Asansol. Nepali populations are more susceptible to motion sickness than the non-nepali population as observed in this study. This study emphasizes the important genetic role behind the motion sickness than the environmental cues.

KEYWORDS: Non-nepali, nepali, motion sickness, susceptibility.

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1. INTRODUCTION

Motion sickness can occur when the sensory inputs about body position contradict what is expected. It can be provoked by abrupt changes in movement, such as occur during bumpy rides, turbulent flights, and rough seas. It can also occur when one is exposed to moving visual scenes while the body is in a relatively fixed state. Early signs of motion sickness include pallor, restlessness, and cold sweat. In later stages, nausea, excessive salivating, and vomiting occur [1]. The degree of

symptoms that result from an acute exposure to provocative stimuli vary with the intensity of the stimulus and one's susceptibility to this condition. The matter of susceptibility involves both genetic and environmental cues. Although visually induced motion sickness (VIMS) has been reported in fixed-base flight and automobile simulation [2,3,4] and in a variety of non-vehicular virtual environments [5,6,7]. The precise etiology of motion sickness remains a mystery. The classic "sensory conflict" explanation, posed by Reason and Brand and supported by subsequent studies, suggests motion sickness is triggered when the brain interprets sensory messages regarding movement as inharmonious [8]. These messages are delivered by the parts of the body that detect motion, including the vestibular receptors, the eyes, and proprioceptors in the skin, muscles, and other tissues. Sometimes the incoming signals from these sensory sites conflict with each other; other times these signals conflict with the brain's "positional memory" [9]. The traditional sensory conflict theory does not explain motion sickness produced by all conditions. Additional sensory inputs other than those traditionally thought to trigger motion sickness may play some role. Mittelstaedt and others, for instance, recently introduced evidence suggesting inputs from visceral graviceptors may contribute to how the body determines its position [10,11]. Another theory for some cases of motion sickness is the postural instability theory, based on experiments in which motion sickness was preceded by statistically significant increase in postural sway. In these cases, motion sickness symptoms were not linked to sensory conflict, but rather to a decreased ability to actively control the body's postural motion [12,13]. It has been shown that circularvection induces symptoms of motion sickness in approximately 60% of healthy human subjects [14,15]. The vestibular system of the inner ear, which senses motion and body position and influences balance, signals 'moving' to the brain, while the eye signals 'stationary' because the car or boat appears stationary relative to the viewer. The vestibular system is also thought to serve as a sensor of disequilibrium-causing neurotoxins (i.e. a toxin detector) and is believed to trigger the emetic response in order to rid the body of toxins. Thus, motion sickness may be an aberrant trigger of the emetic response. However, gravity is sensed by the otoliths within our inner ears. These otoliths function as head fixed linear accelerometers [16, 17, 18, 19, 20], Evidence for the involvement of the vestibular system comes from the observation that individuals with complete loss of the vestibular apparatus, a component of the vestibular system, are immune to motion sickness [21].

2. MATERIALS AND METHODS

Candidates were chosen by means of random sampling from different pockets of Asansol during the time of study. Both the non-nepali and nepali peoples were asked regarding their sickness susceptibility during motion. Sickness susceptibility includes nausea, dizziness, headache, nausea, excessive salivating and vomiting during the time of motion, as moving through car or bus. From non-nepali population 2111 people were asked for motion sickness, out of which 991 were male and 1120 were female and from nepali population out of 1109 people 468 were male and 641 were

female.

3. RESULTS AND DISCUSSION

3.1: The data are expressed in the column chart and by pie chart. 27% of nepali population are susceptible to motion sickness, whereas 8.95% are from non-nepali population.

A. Column chart showing unaffected and affected percentage in non-nepali and nepali population.

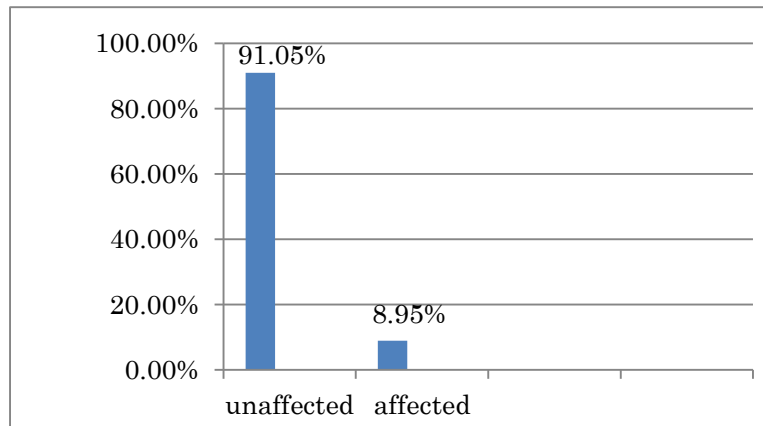


Fig 1 : Non-nepali population

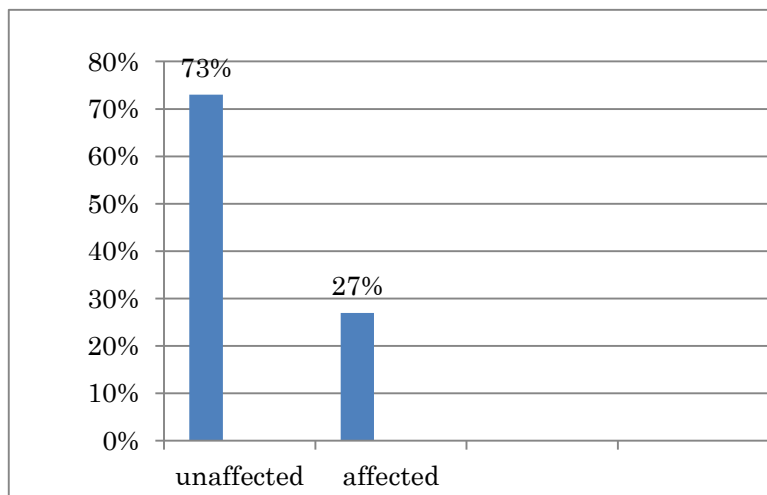


Fig 2: Nepali population

B. Pie chart showing unaffected and affected percentage in non-nepali and nepali population.

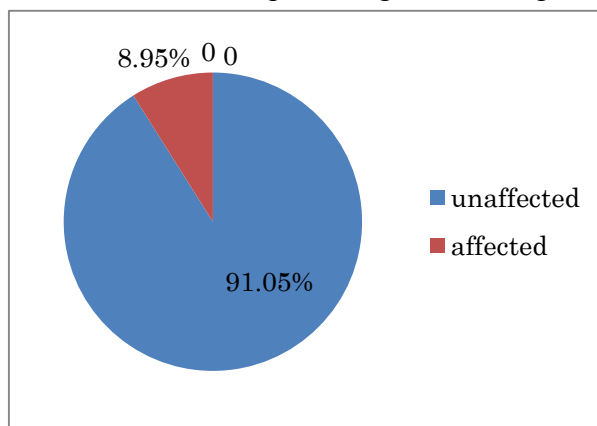


Fig 3: Non-nepali population

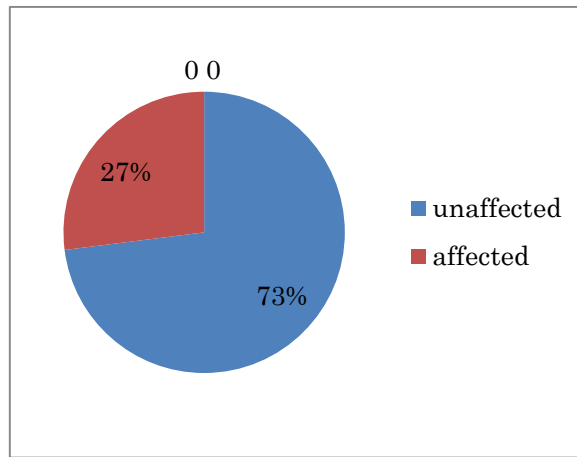


Fig 4: Nepali population

3.2:8.37% of males of non-nepali origin are prone to motion sickness and 6.19% males of nepali population are show the susceptibility.

A. Column chart showing unaffected and affected percentage among males of non-nepali and nepali population.

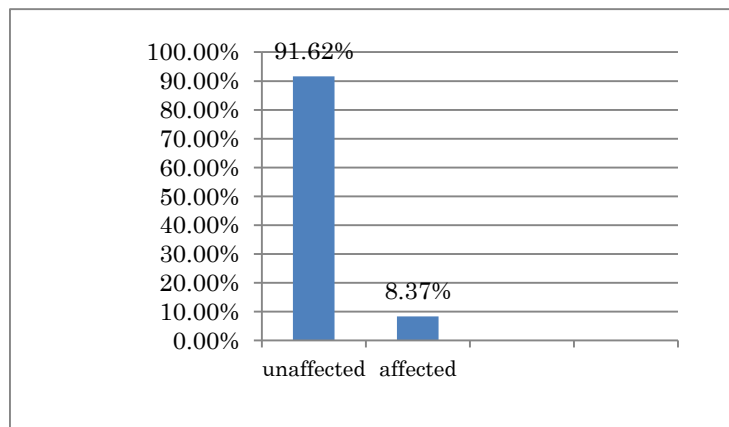


Fig 6: Nepali population

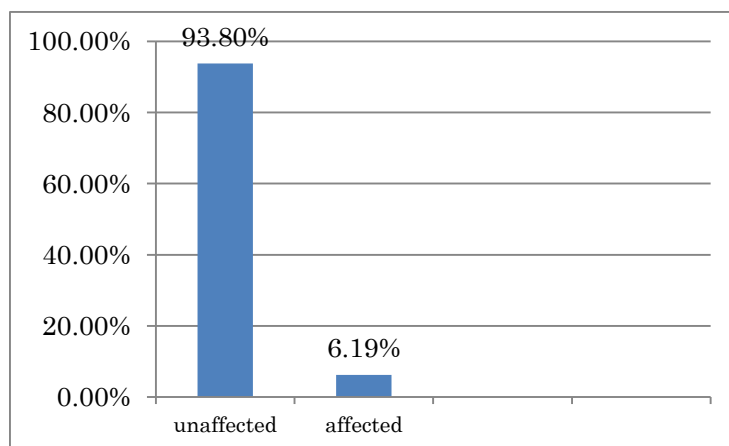


Fig 5: Non nepali population

B. Pie chart showing unaffected and affected percentage among males of non-nepali and nepali population.

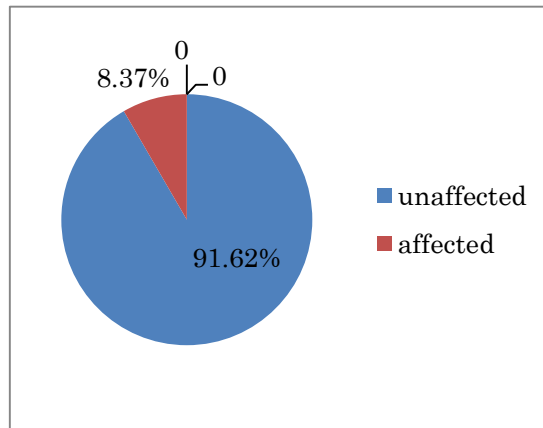


Fig: 7: Non-nepali population

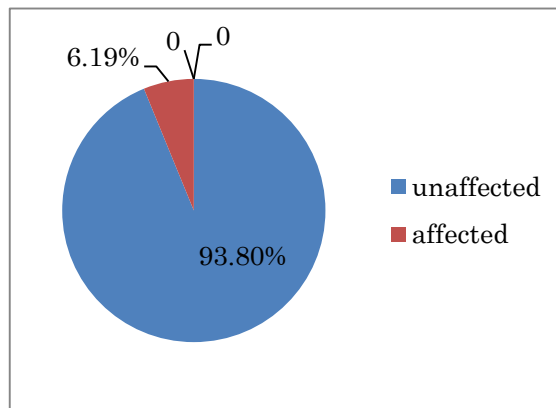


Fig 8: Nepali population

3.3: Nepali female population is highly susceptible for motion sickness and show 33.22% than the non-nepali female which show 9.46%.

A. Column chart showing unaffected and affected percentage among females of non-nepali and nepali population.

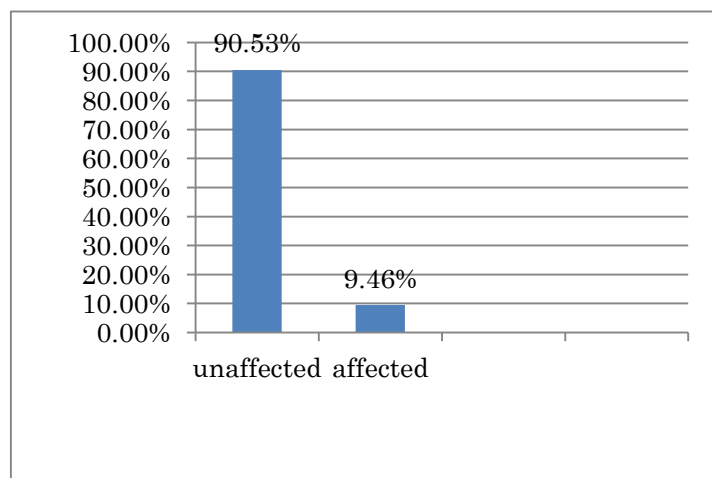


Fig 9: Non-nepali female population

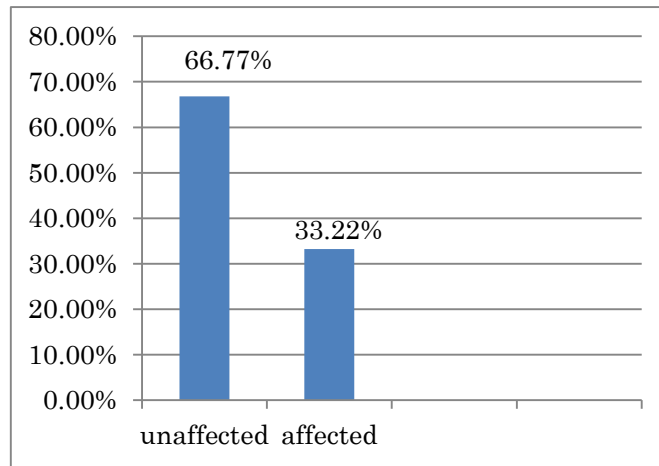


Fig 10: Nepali female population

B. Pie chart showing unaffected and affected percentage among females of non-nepali and nepali population.

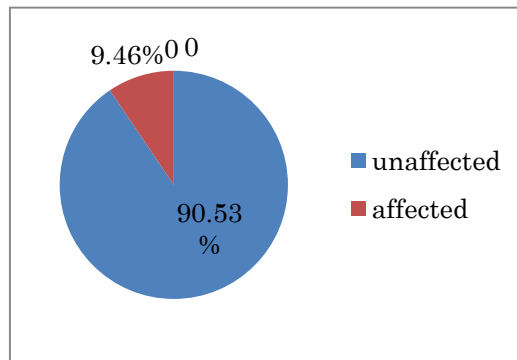


Fig 11: Non-nepali female population

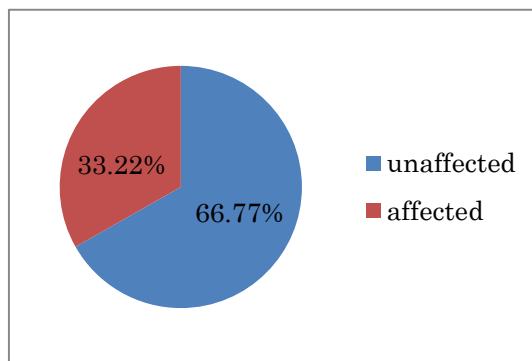


Fig 12: Nepali female population

DISCUSSION

Nepali and non-nepali people were identified by their phenotype, generally the nose of the nepali people is tapered than the non-nepali people, but this is not the sole identifying character. Final judgements were done by the names. From this study it is observed that nepali people are more prone to motion sickness than the people from non-nepali origin. Susceptibility of motion sickness is found more in males of non-nepali than nepali people, in case of female the scenario is just opposite. Interestingly the frequency of susceptibility in nepali female population is much higher

than the non-nepali female population and which is not true for the males. Genes in regions associated with motion sickness appear to play roles in eye and ear development, balance and other neurological processes and glucose homeostasis. Two of the genome-wide-significant regions contain hypoxia-inducible genes. Motion sickness tends to be more common in women and younger people than in male. It is heritable. It is just because of genetics, according to the first ever genome wide association study on motion sickness done by researchers at 23 and Me. 23 and Me researchers conducted their study on motion sickness and 35 variants were found that were significantly associated with the condition. Up to 70% of a person's risk for motion sickness is due to genetics. Due to conflicting signals in the inner ear, eyes and sensory receptors, motion sickness arises. This phenotypic association between motion sickness and its symptoms like vertigo, migraines helps provide many ideas about the causes and genetics of motion sickness [22]. Motion sickness is induced by a neurosensory mismatch that results in the symptoms of nausea and tachygastric and increased beta-endorphin [23], vasopressin [24;25] and epinephrine [26]. In this study the most important finding is nepali female are highly susceptible than the non-nepali female population and this is far more than the susceptibility between the males of the nepali and non-nepali. As the population is mixed and the nepali people are living at Asansol for a long time so the acting environment is more or less same for all. Thus the major role player for this susceptibility is the genes than the environment can be concluded from this study. Susceptibility to MS is affected by both ethnic origin and by gender in a rather complex fashion [27], as the persistence of motion sickness is found more in nepali population and females are more affected than male in the same. Thus the present study shows its consistency and supports the outcome of the previous worker.

4. CONCLUSION

Motion sickness, although the frequency of susceptibility is moderate and vary from gender to different ethnic group but very little focus is given in this milieu. Different medicines are found in the literature but their function may vary among individuals. Both the environmental and the genetic factors are responsible for the motion sickness. The inheritance pattern of this sickness is still lacking, although few SNP are discovered which are closely associated with the development of eye and ear. Thus this paper is a small step toward the journey of motion sickness.

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CONFLICT OF INTEREST

Author doesn't have any conflict of interest regarding this present study.

REFERENCES

1. Murray JB. Psychophysiological aspects of motion sickness. *Perp. and Motor Skills*.1997; 85:1163–1167.
2. Frank LH, Casali JG, Wierwille WW. Effects of visual display and motion system delays on operator performance and uneasiness in a driving simulator. *Human Fact*. 1988; 30:201–217.
3. Lee GCH, Yoo Y, Jones SA. Investigation of driving performance, vection, postural sway, and simulator sickness in a fixed-base driving simulator. *Proceedings of the Twentieth International Conference on Computers and Industrial Engineering*, October, Seoul, Korea.1996;677– 680.
4. Yoo Y, Lee GCH, Jones SA. Vection, compensatory sway, and simulator sickness. In: Dos, B.Karowski W, eds. *Advances in occupational ergonomics and safety 1997*. Amsterdam: IOS Press; 1997:589 –592.
5. DiZio P, Lackner JR. Spatial orientation, adaptation, and motion sickness in real and virtual environments. *Presence*. 1992; 1:319 –328.
6. Ellis SR. Nature and origins of virtual environments: A bibliographic essay. *Comput Syst Eng*. 1991; 2:321–347.
7. Stanney K, Salvendy G. Aftereffects and sense of presence in virtual environments: Development of an R&D agenda. *Int J Human Comp Int*. 1998; 10:135–187.
8. Reason JT, Brand JJ. *Motion sickness*.London:Academic Press, 1975.
9. Oman CM. Motion sickness: a synthesis and evaluation of the sensory conflict theory. *Can J Physiol Pharmacol* 1990; 68:294–303.
10. Mittelstaedt H. Somatic graviception. *Biological Psychology*. 1996; 42:53–74.
11. Kohl R, Homick JL. Motion sickness: a modulatory role of the central cholinergic nervous system. *Neuro & Biobehav Reviews*. 1983; 7:73–75.
12. Stoffregen TA, Hettinger LJ, Haas MW, Roe M & Smart LJ. Postural instability and motion sickness in a fixed-based flight simulator. *Human Factors*. 2000; 42:458–469.
13. Smart LJ, Smith DL. Postural dynamics, clinical and empirical implications. *J of Mani PhysioThera*. 2001; 24:340–349.
14. Stern RM, Koch KL, Leibowitz HW, Lindblad IM, Shupert CL, Stewart WR. Tachygastria and motion sickness. *Aviat Space Environ Med*. 1985; 56:1074-7.
15. Stern RM, Koch KL, Stewart WR, Lindblad IM. Spectral analysis of tachygastria recorded during motion sickness. *Gastroenterology*. 1987; 92:92-7.
16. Fernandez C, Goldberg JM. Physiology of peripheral neurons innervating otolith organs of the squirrel monkey. I. Response to static tilts and to long-duration centrifugal force. *J. Neurophysiol*. 1976; 39: 970–984.
17. Fernandez C, Goldberg JM. Physiology of peripheral neurons innervating otolith organs of the squirrel monkey. II Directional selectivity and force–response relations. *J. Neurophysiol*. 1976;

18. Fernandez C, Goldberg JM. Physiology of peripheral neurons innervating otolith organs of the squirrel monkey. III Response dynamics. *J. Neurophysiol.* 1976; 39: 996–1008.
19. Lindeman HH, Anatomy of the otolith organs. *Adv. Otorhinolaryngol.* 1973; 20: 405–433.
20. Mayne R. A systems concept of the vestibular organs, in: H.H. Kornhuber (Ed.), *Handbook of Sensory Physiology. IV-2 Vestibular System*, Springer Verlag, Berlin. 1974; pp. 493–580.
21. Covanis, A. Panayiotopoulos syndrome: A benign childhood autonomic epilepsy frequently imitating encephalitis, syncope, migraine, sleeps disorder, or gastroenteritis. *Pediatrics.* 2006; 118: e1237–e1243.
22. Bethann SH, Joyce YT, Amy KK, Chuong BD, David AH, Nicholas E. Genetic variants associated with motion sickness point to roles for inner ear development, neurological processes, and glucose homeostasis. *Hum Mol Gen.* 2015; 24: 2700–2708.
23. Koch KL, Stern RM, Vasey MW, Seaton JF, Demers LM, Harrison TS. Neuroendocrine and gastric myoelectrical responses to illusory self-motion in humans. *Am J Physiol.* 1990; 258:E304-310.
24. Koch KL, Summy-Long J, Bingaman S, Sperry N, Stern RM. Vasopressin and oxytocin responses to illusory self-motion and nausea in man. *J Clin Endocrinol Metab.* 1990; 71:1269-1275.
25. Li X, Jiang ZL, Wang GH, Fan JW. Plasma vasopressin, an etiologic factor of motion sickness in rat and human. *Neuroendocrinology.* 2005; 81:351-359.
26. Xu LH, Koch KL, Summy-Long J, Stern RM, Seaton JF, Harrison TS, Demers LM, Bingaman S. Hypothalamic and gastric myoelectrical responses during vection-induced nausea in healthy Chinese subjects. *Am J Physiol.* 1993;265:E578-584
27. Klosterhalfen S, Kellermann S, Pan F, Stockhorst U, Hall G, Enck P. Effects of ethnicity and gender on motion sickness susceptibility. *Aviat, Space, and Environ Med.* 2005; 76: 1051–1057.