

Motion Sickness: Manifestations and Prevention

G.P. Kumar*, K.R. Anilakumar, Mallesha, Y.C. Sekhar, and R.K. Sharma

DRDO-Defence Food Research Laboratory, Mysore - 570 011, India

**E-mail: phanikumar.g @dfrl.drdo.in*

ABSTRACT

Motion sickness is an ancient problem associated with transportation (ships and other vehicles), which is affecting humans since ages. Motion sickness is characteristically occurring during abnormal movements induced by the motion and when there is a conflict between various senses such as visual, vestibular and motor system. Depending on the type of motion, various kinds of sicknesses, such as air sickness, car sickness, train sickness, seasickness, etc. may occur. A very less per cent of individuals are highly susceptible to motion sickness and very less per cent of individuals are highly insusceptible for motion sickness. However, most of the population comes in between. The primary symptoms of motion sickness include nausea, vomiting, wanes, and cold sweating. Varieties of drugs are available to reduce susceptibility to motion sickness. However, nausea, pallor, sweating, headache, dizziness, malaise, increased salivation, apathy, drowsiness, belching, hyperventilation and stomach awareness are the other symptoms of motion sickness. Anti-cholinergics and anti-histamines are the most effective motion sickness prophylactics with apparent side effects such as dry mouth, drowsiness, and depression. There are theories and mechanisms which include intra-vestibular (Canal-Otolith) mismatch theory, sensory conflict theory, visual-vestibular mismatch theory, the poison theory, the postural instability theory, and the movement program theory. Benzodiazepines, anticholinergics, anti-histamines and monoamine antagonists have commonly used treatment regimes. The traditional way of tackling the problem is the consumption of ginger, peppermint, lemon, fennel, marjoram, rosemary, basil. This review summarizes prediction and evaluation, behavioural strategies to prevent or minimize symptoms of motion sickness and available countermeasures of motion sickness.

Keywords: Motion sickness; Sea sickness; Neurotransmitters; Vestibular disturbance; Neuronal mismatch

1. INTRODUCTION

Motion sickness (MS) is an ancient problem associated with transportation (ships and other vehicles), which is effecting humans since ages. MS is a physiological response to the conflicts between various senses like optical senses, vestibular system and muscular system. These conflicts can lead to discomfort and difficulties concentrating and in worst case lead to nausea and vomiting¹. MS can happen on practically anything that under motion. MS is also known as kinetosis and travel sickness. MS is a condition in which a disagreement of co-ordinations exists between visually perceived movement and the vestibular system's senses. The symptoms of MS are similar despite the consequences of the stimuli that cause it, however they are classified depending on where it occurs such as: sea sickness, car sickness, air sickness, space sickness, travel sickness and simulator sickness¹. About 5 to 10% of travellers are highly susceptible to MS, while others show moderate susceptibility². The susceptibility to motion sickness varies widely and does not affect all in road transportation³. The occurrence of MS, depending on population, Asians are more susceptible when compared to others⁴. The occurrence

of MS also depending on age and sex; women are experience more severe sickness and a higher occurrence of nausea than men⁵. Children less than 2 years old are more vulnerable to MS when compared to adults⁶; and incidents of MS peak between 3 and 12 years, and gradually decrease after that⁷. The general responses of autonomic nervous system during MS are gastrointestinal and other peripheral changes. It is clear that during the process of MS parasympathetic nervous system withdrawal and sympathetic nervous system activation plays a role. There are several possible relationships that were observed between motion sickness susceptibility and personality like some psycholological disorders such as anxiety, claustrophobia and migraine. They are at more risk to MS when compared to others⁸.

2. THEORIES, MECHANISMS AND MANIFESTATIONS

The human body can receive information about posture and movements by various systems such as sensory information comes from the inner ear, visual information receives from the eyes and prospective information receives from muscles (Fig. 1). Although the etiology and precise neurobiological mechanism of MS are still in ambiguous. However, several hypotheses have been proposed and varieties of countermeasures have

been developed to overcome the problems of MS since decades. Despite the fact that several hypotheses about the pathophysiological mechanisms, neurophysiological process and aetiological models of MS are by no means clear. Further, there was lack of scientific evidences for several models, pathways or the process of adaptation to provocative motion. Three main theories exist for motion sickness is as follows⁹:

2.1 The Sensory Conflict Theory

This is one of the oldest theory of MS and well accepted ones. Sensory conflict theory explains about the elicitation of MS to ‘conflicts’ between various sensory organs such as between signals from vestibular system and optical senses, or signals from canals and otoliths. Furthermore, this theory can be called as ‘Neural Mismatch Theory’. The converging sensory inputs from the otolith organs, semicircular canals, eyes and somatosensory receptors are mismatched with the expected sensory patterns in the neural store calibrated by past experience, and spatial orientation is disturbed, leading to motion sickness. It is unclear whether the brain can compare various types of sensory inputs directly, but it seems to be capable of creating expectations of motion based on earlier experience which is compared with the perceived motion¹⁰. When we are asleep these comparisons do not happen because the brain does not analyze the inputs, consequently the sensory conflict does not occur and we can accordingly not become motion sick¹¹. MS can also occur when an expected stimulus does not emerge or when unexpected stimuli emerge as per brain comparison¹. For example when a person reading in a car the vestibular system gives information about movement, but the visual system does not confirm this movement and these contradictory factors can cause MS¹². Norfleet¹³, *et al.* have experimented sensory conflict theory by rotating the gravity vector through 180° (inverted immersion), and created sensory conflict between the vestibular signals from semicircular canals and visual signals from eyes. They have confirmed sensory conflict by inverted immersion must have cause more severe motion sickness than upright immersion. Reason¹⁴ proposed neural mismatch hypothesis based on the reafference principle. According to his hypothesis, the situations that provoked motion sickness were characterised by a condition of (sensory) rearrangement. Further this theory is divided into two types:

2.1.1 Visual-Vestibular Mismatch Theory

This type of MS always happens on passive means of transportation. Mismatch between the senses from vestibular organs and the vision. For example, a person is travelling on a ship and observing the waves (visual information recognise a movement); however, in reality the body does not move.

2.1.2 Intra-vestibular (Canal-Otolith) Mismatch Theory

Sometimes Canal and otolith signals simultaneously give contradictory information. For example the vestibular system and the muscular system send senses of motion, but the visual information do not recognize this. Making head movements while rotating in opposite axis of motion or fast head movement may lead this type of MS.

2.2 The Poison Theory

Emetic signals received by the coordinated activity of both smooth and somatic muscles to induce certain changes in intra-abdominal and intra-thoracic pressures, and opening of the esophageal sphincters. The poison theory is based on genetic responses (old evolutionary programme), where brain misreads the neural signals like ‘poison response’ and lead to stomach emptying by reflux action to get the poison out of the body, as many poisons have the same effects on the body as MS does¹⁵. The toxin theory is based on the idea that the sensory conflicts and postural instability would give us an early sign of intake of neurotoxins¹⁰. The toxin system works as a backup to our other indicators of poisoning such as taste, smell or vomiting evoked by effects on the reflexive action on stomach or stimulation of the chemoreceptors after absorption. Some of the concepts of MS contradict this theory that the toxins that have already crossed the blood-brain barrier also have a very small biological advantage since the toxins have reached the brain cannot be removed by vomiting¹⁰. In addition, not all motion sickness leads to vomiting which opposes the explanation that the key purpose of motion sickness would be to remove toxins.

2.3 The Postural Instability Theory

The postural instability theory is not a modification of the sensory conflict theory but differs in fundamental ways. Loss of stability generally is coupled with a loss of control, such as falling down. Postural stability is defined as the condition in which uncontrolled movements of the perception and action systems are minimised¹⁶. Riccio and Stoffregen¹⁶ predicted that optical oscillations created by human locomotion should induce MS through unintended consequence of exposure to simulations of optical flow fields that are created by spontaneous postural sway¹⁷. Riccio and Stoffregen¹⁶ have experimented in animals and found that the animals experienced motion sickness due to postural instability. They have suggested that this instability, which occurred prior to the onset of the symptoms of motion sickness, was a necessary prerequisite of this response. Postural instability theory concludes that motion sickness results from perceptual-motor anomalies, but not from changes in sensory input. According to Riccio and Stoffregen¹⁶, MS is caused by instability in the control of the posture of the body and/or its segments. Therefore, it is defined as rearrangements in the relationship between movement and sensorimotor feedback. Smart¹⁸, *et al.*, reported that postural instability is being the onset of symptom after MS and the observations are compatible with other theories of motion sickness etiology.

2.4 The Movement Program Theory

The movement program theory is based on that a negative and uncomfortable experience of motion other than expected would discourage the development of movement programs adapted to situations where these vestibular conflicts occurs¹⁰. The phenomenon is explained as a negative reinforcement model, a sort of natural obedience training, to make us stop undesirable behaviour and elude the probability of injury. The obnoxious sensation of motion sickness is meant to make us stop the motion to reduce the feeling, and the vomiting is

meant to force us to stop what we are doing since vomiting is inconsistent with substantial movement. The effects of the negative experience can lead to early avoidance, reduction of movement and removal of oneself from the offending circumstances¹⁰.

Motion is initiated by the brain through different pathways of the nervous system including vestibular system, vision, muscular system, etc. The symptoms of motion sickness appear when the central nervous system receives conflicting messages from the vestibular system, the visual system, sometimes from smell and respiratory systems

3. PHYSIOLOGY OF MOTION SICKNESS

Motion sickness is linked to a pronounced activation of the glucocorticoid and sympatico-adrenergic stress response systems¹⁹. Number of pharmacological agents could potentially affect the occurrence of motion sickness through their actions in the vestibular nuclei. The cerebral cortex and limbic system, particularly the hippocampus, are major sites of spatial orientation information processing. A number of neurotransmitters influence the activity of vestibular nucleus neurons, including acetylcholine, glutamate, glycine, GABA, histamine, norepinephrine, dopamine, serotonin, substance P, somatostatin, adrenocorticotrophic hormone [ACTH] and enkephalin²⁰. The histaminergic neuron system in the brain has been supposed to play a significant role in the pathophysiology of motion sickness. Neural mismatch signals activates the histaminergic neuron system in the hypothalamus and stimulates H1-receptors of the brainstem, therefore H1-receptors triggers the development of the symptoms and signs of motion sickness, including emesis.

3.1 The Vestibular System

The vestibular system is exaggerated by vertical and horizontal vibrations and forces of acceleration. The vestibular system is maintaining the body balance; it records change in movement caused by motion and coordinate the position of the head through regulation of muscle tension which helps us keep our posture. The vestibular system consists of semicircular canals and otolith organs¹. Calcium carbonate crystals present on the otolith organs help us to detect linear acceleration and the position of the head. The otoliths are responsible for the opposite direction of visual signals when the head moves in a roll motion, which is known to be ocular counter-rolling²¹. The part that detects angular acceleration is hair cells, called cilia, attached to the inside walls of the semicircular canals. The vestibular nuclei receive MS signals from the semicircular canals and otolith organs but also visual, auditory, somatosensory, and proprioceptively related signals and a variety of other afferents including from the cerebellum. The indication of angular acceleration tells us that our head is moving¹. Individuals with a total loss of labyrinthine function are not susceptible to motion sickness.²²

3.2 The Visual System

The visual system is crucial part of the phenomenon of MS as it coordinates us to substantiate other organs such as vestibular, somatosensory and motor related signals tell us

to anticipate¹. Hence, an optical illusion of movement while standing still, or an illusion of non-movement while moving will create a conflict between signals to the brain from the optical-vestibular system. A conflict can also be created through an optical illusion of moving in an opposite direction than the actual.

4. SYMPTOMS OF MOTION SICKNESS

Individual's response towards MS depends on the comparative provocativeness of the stimulation, relative susceptibility and previous experience²³. Some people on receiving provocative stimulation will show a very brief response, and others will maintain discomfort for a prolonged period. One of the major symptoms of motion sickness is vomiting¹¹. Emesis can be triggered by the involvement of a variety of peripheral and central afferent mechanisms. The causes of vomiting are input from the vestibular apparatus, higher brain stem, cortex, gastrointestinal system or viscera²⁴. In severe motion sickness with multiple bouts of vomiting, alkalosis may develop because of hydrogen ion loss and lead to increased renal excretion of potassium bicarbonate resulting in potassium deficiency which can cause muscle weakness, constipation, and cardiac arrhythmias. The psychological part of motion sickness could also trigger vomiting on its own since MS signal input triggered from the cerebral cortex to the vomiting centre in the brain. The physiology of MS starts with provocative motion when travellers exposed initially, therefore, it is assumed that MS response would be 'physiological' in origin. Further, as the people have disagreeable experiences with motion then psychological components becomes more significant. Therefore, the degrees of MS arousal may vary from person to person due their past experiences with a variety of different provocative stimuli.

Physiological motion sickness manifestations are as follows: Cardiovascular system includes, Changes in pulse rate and/or blood pressure, peripheral circulation decreases especially in the skin of the head, tone of arterial portion of capillaries in the fingernail bed increases, muscle blood flow increases²⁵. During motion sickness respiratory system has some manifestation such as, alterations in respiration rate, sighing or yawning and air swallowing. In the gastrointestinal system the following changes may occur during motion sickness, inhibition of gastric intestinal tone and secretions, salivation, gas or belching, epigastric discomfort and vomiting. Changes in lactic dehydrogenase concentrations in the body fluids are one manifestation studied under motion sickness. Blood and urine of the patients have several changes like, increased haemoglobin concentration, decreased concentration of eosinophils and glucose utilisation, higher levels plasma protein and ADH. Both blood and urine have increased levels of 17-hydroxycorticosteroids and of catecholamines during motion sickness. Visual system has some adverse manifestations like ocular imbalance, dilated pupils during emesis, small pupils and nystagmus.

Serious symptoms of MS include: nausea, vomiting, pallor, sweating, drooling, short breath, dizziness, drowsiness; other common signs are: a general feeling of discomfort and not feeling well (malaise); mild symptoms are categorised as: headache, mild unease and yawning.

5. PREDICTION AND EVALUATION OF MS

It has been established that almost all travellers can obtain MS when exposed to appropriate provocative motion. MS incidence is depended on individual threshold to motion stimulation and varies under various vertical motion parameters such as acceleration magnitude, frequency, and duration²⁶. Some of the researchers have made some calculation to predict sea sickness named it as ‘sicken passengers ration’, which includes ship speed, loading condition, sea state, passenger behavior and habituation to moving environment²⁷. These calculations may improve the degree of comfort and the work ability on the sea. MS can be diagnosed according to the manifestations during motion exposure after excluding other pathological disorders. Heart rate variability (HRV) can be useful for assessing cardiac sympathovagal interactions and electrogastrogram (EGG) would be useful to find gastric motility during MS.

6. PREVENTING MOTION SICKNESS

Prevention of motion sickness can be complex. A small percentage of normal individuals are highly susceptible for

nearly all exposure situations, a small percentage are highly insusceptible, and most are in between. The best prevention for the highly susceptible is avoidance. For other individuals, spaced exposure of short duration can lead to a buildup of adaptation to the provocative situation. This is especially effective if only minor symptoms of motion sickness are allowed to develop before terminating each exposure period. Alternatively, incremental exposure to gradually increasing levels of provocative stimulation (e.g., making head movements during exposure to passive body rotation at higher and higher rotation velocities) can allow adaptation to be achieved without motion sickness being elicited even at stressor levels that if achieved in a single step would be intolerably provocative.

Some of the classes of drugs which have been used to treat motion sickness are given below. Medications are most effective when taken prophylactically before traveling, or as soon as possible after the onset of symptoms. Common side effects of these medications are dry eyes, dry mouth, sensitivity to bright light; Less common side effects are blurred vision, dizziness, headache, sedation. A list of pharmacological approaches have been given in Table 1.

Table 1. Pharmacological countermeasures for motion sickness

Category	Name of drug	Dosage & route of application	Usage	References
Antihistamines	Dimenhydrinate	p.o. (100 mg)	2 h before travel	45
		Chewing gum (3 X 20 mg)	Chewed for 30 min each during travel	28
	Cinnarizine	Oral (30 or 50 mg)	3 h before travel	29
	Cyclizine (Marezine)	p.o. (50 mg)	2 h before travel	44
		p.o. (25 or 50 mg)	2 h before travel	46
	Promethazine	i.m. (25 or 50 mg)	1–2 h before travel	30
		Suppository (25 or 50 mg)	1–2 h before travel	31
	Meclizine (Antivert)	p.o. (25 or 50 mg)	1–2 h before travel	47
	Chlorpheniramine,	p.o. (4 or 12 mg)	3–4 h before travel	32
Betahistine	p.o. (32 or 48 mg)	1–2 h before travel	33	
Dopamine Antagonists	Metoclopramide	i.v. (20 mg)	15 min after travel initiation	48
Anticholinergics	Scopolamine	p.o. (0.6 mg)	0.5–1 h before travel	43
		Transdermal therapeutic system (1.5 mg/patch)	6–8 h before travel	43
		Intranasal (0.4 mg)	0.5 h before travel	34
		p.o. (0.3 mg) + TTS	1 h before travel	35
Hormones	Dexamethasone	i.v. (0.5 mg)	Every 6 h for 48 h	36
5-HT1B/1D receptor agonist	Rizatriptan	p.o. (10 mg)	2 h before travel	37
Combination	Promethazine + d-amphetamine	p.o. (25 mg+10 mg)	2 h before travel	38
		Scopolamine + d-amphetamine	p.o. (0.4–1.2 mg+5 mg)	0.5–1 h before travel
	Scopolamine + ephedrine	p.o. (0.3 mg+25 mg)	0.5–1 h before travel or 3 times daily	40-41
		i.m. (0.2 mg+25 mg)	30 min before travel	
Chlorpheniramine + Ephedrine	p.o. (12 mg+50 mg)	3–4 h travel	42	

p.o : Per oral; i.v ; intravenous; i.m. intramuscular

6.1 Benzodiazepines

Benzodiazepines are occasionally administered for severe symptoms of motion sickness. The serotonin antagonist ondansetron (Zofran) is ineffective for the prevention and treatment of motion sickness.

6.2 Anticholinergics

Scopolamine, an anticholinergic, is a first-line option for preventing motion sickness in persons who wish to maintain wakefulness during travel. Transdermal patches are most effective, one patch applied to mastoid at least four hours before travel, then every 72 hours as needed. If the recommended dose of scopolamine does not adequately relieve symptoms, the dose may be doubled. Oral scopolamine treatment is moderately effective, 0.4 to 0.6 mg one hour before travel, then every eight hours as needed⁴³.

6.3 Antihistamines

First-generation antihistamines have been used to treat motion sickness since the 1940s. Antihistamines are H1-antagonists have been successfully demonstrated to be efficient in controlling motion sickness. As histaminergic neuron system is involved in the symptomatic mechanism of MS via H1-receptors, this group of medicines can reduce the severity of the symptoms and signs of motion sickness by blocking the emetic linkage. They are generally recommended for patients who can tolerate their sedative effects. Cyclizine (Marezine)⁴⁴, dimenhydrinate⁴⁵, promethazine⁴⁶, and meclizine⁴⁷ (Antivert) demonstrated effectiveness. Nonsedating antihistamines are not effective in preventing or treating motion sickness.

6.4 Monoamine Antagonists/Agonist

Dopamine D2 and D3 receptors are known to play a role in nausea and emesis. They can alter the amount of cAMP within neurons of the vomiting center via inhibiting the CTZ in the area postrema. Dopamine D2-receptor antagonists such as domperidone and metochlopramide are clinically used as anti-emetic drugs⁴⁸.

6.5 Traditional Medicinal Systems

Various Indian medicinal plants are also reported for their effective use in the treatment of emesis. Furthermore, several Ayurvedic system of medicine and traditional Chinese Medicine (TCM) consists of number of natural products used in the treatment of emesis. Asian herbs such as *Agastache rugosa*; leaves of *Mentha piperita*; flowers of *Eriobotrya japonica* and *Eugenia caryophyllata*; fruits of *Cocos nucifera* and *Amomum cardamomum*; roots of *Cyperus rotundus*; rhizome of *Zingiber officinale* are generally used to treat emesis⁴⁷⁻⁵⁰.

- **Ginger:** Ginger is one of the most effective home remedies for motion sickness. For nausea, ginger extract works very well. One can have this extract along with a tea spoon of honey or keeping a slice of ginger in mouth while travelling also proves effective to cope with travel sickness⁵¹.
- **Peppermint:** Peppermint also proves to be effective in dealing with motion sickness. Prepare and drink a peppermint tea before your travel⁵².

- **Lemon:** The smell of lemon gives relief when one feels nauseated. It is better to keep a lemon and keep inhaling the smell while travelling. Drinking a glass of lemon water also works well⁵³.
- **Fennel** (*Foeniculum vulgare*) is another herb that can be used to prevent and treat motion sickness⁵¹.
- **Marjoram, Rosemary and Basil** are other herbs that work for them against motion sickness⁵³.

7. BEHAVIORAL STRATEGIES TO PREVENT OR MINIMISE SYMPTOMS OF MOTION SICKNESS

The most effective remedial measure, at least in the long term, is adaptation to the provoking motion, and it is the ideal method of minimize motion sickness. Prevention of motion sickness is more effective than treating symptoms after they have occurred. Therefore, patients should learn to identify situations that may lead to motion sickness and be able to initiate behavioural strategies to prevent or minimise symptoms. With continuous exposure to motion, symptoms of motion sickness will usually subside in one to two days. Alternatively, slow, intermittent habituation to motion is an effective strategy to reduce symptoms. It is important to note that multiple and off-axis motions are worse than one-axis motions; low-frequency motions are worse than high-frequency motions; rotary motion is worse than linear motion and the vertical motions are worse than horizontal motions. Adaptation is one of the most effective techniques to overcome MS. A stimulus with a gradual onset generates fewer symptoms and allows for more rapid adaptation than a stimulus with an abrupt onset. Under the adaptation of MS the following two techniques may be useful such as, sensory adaptation-decreasing response following continuous stimulation of a receptor system, and protective adaptation-adaptation to a sensory mismatch signals. Golding⁵⁴ prepared a factor analysis of self-report questionnaires, which is designed to assess susceptibility MS, this questionnaire may help in getting information with respect to the existence of independent latent susceptibilities to different types of provocative environments, usually forming factors that might be termed transportation by land, air or sea (*Appendix 'A'*).

8. DIRECTIONS FOR PREVENTIVE MEASURES FOR MOTION SICKNESS

The important directions include sitting in a position so that the eyes can see the same motion that the body and inner ear feels, sitting in the front seat and looking at the distant scenery if in a car, going up on the deck and watching the motion of the horizon if on a boat, sitting by the window and look outside in an airplane, choosing a seat over the wings where the motion is minimised, avoiding reading while travelling, avoiding sitting in a seat facing backward, avoiding watching or talking to another traveller who is having motion sickness, avoiding strong odors and spicy or greasy foods immediately before and during travel, avoiding excessive alcohol and smoking and foods or liquids that make feel unusually full, adapting breathing techniques (as slow and deep breaths lead to comparatively fewer symptoms), chewing a gum to maintain balance between vision and balance, eating a bland food such

as pancakes or a bagel or eating banana, rice, apple and toast (BRAT) diet, avoiding acidic food and drinks, etc. Sleeping has a helpful on MS symptoms as it reduces the excitability of the vestibular system and thus reduces the sensory conflict.

9. CONCLUSION

This paper reviews the signs and symptoms, stimuli and response characteristics, anatomical structures, susceptibility factors, and theories of motion sickness. It is evident from this review that motion sickness is both polysymptomatic and polygenic and traditional way of tackling by dietary therapy might also play a vital role. More systematic research using animal model, the toxicological studies of the herbal formulations and the structured human trials are warranted to provide an insight into the problem of seasickness.

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CONTRIBUTORS

Dr G. Phani Kumar, received MSc, PhD. Presently he is working as Scientist 'E' in DRDO-Defence Food Research Laboratory, Mysore. His expertise is in herbal formulation. He is involved in the studies of development and evaluation of functional foods and nutraceuticals to support hepato-protective, neuro-protective, anti-fatigue, anti-anxiety, anti-depression and anti-sea sickness properties in experimental animals and cell line models. He has published more than 50 research papers in peer reviewed journals.

In the current study, he has written the manuscript with the collection of review.

Dr. K R Anilakumar did Masters and PhD in food science. Presently working as Scientist 'F' and heading the Food Quality Assurance Division, DRDO-Defence Food Research Laboratory, Mysore. He is involved in the studies of development and evaluation of functional foods and nutraceuticals to support hepato-protective, neuro-protective, anti-ulcer, anti-fatigue, anti-anxiety, anti-depression and anti-sea sickness properties in experimental animals. He has published 82 research papers in peer reviewed journals, 21 review papers/ book chapters and filed/granted 14 patents.

In the current study, he has provided the information about the herbal antidotes against MS.

Dr Mallesha, received MSc, PhD. Presently he is working in Food Quality Assurance Division, DRDO-Defence Food Research Laboratory, Mysore. He has developed microbial testing kit for the presumptive identification of coliforms and *Salmonella* in food samples and also worked on the microbial production of gamma linolenic acid, bacteriocins and biopolymers by using food wastes. Presently, working on the development of simple field test kits for the detection of pesticides. He has published more than 30 research papers in journals, presented more than 32 research papers in conferences and filed/granted 3 patents.

In the current study, he has collected the data for pharmacology of motion sickness.

Dr Chandrasekhar Yadavalli received PhD (Life Sciences) from Bharathiar University, in 2019, presently working in the Department of Biology at Indian Institute of Science Education and Research, Tirupati. He has contributed his work on anti-stress properties of *Terminalia* species.

In the current study, he has collected the data regarding molecular mechanism of MS and human trail relevant studies.

Prof. Rakesh Kumar Sharma holds his PhD in Chemistry from the University of Delhi. D.Sc. (HonorisCausa) has been conferred on him by Dr. K.N. Modi University, Newai, Tonk, Rajasthan. He was superannuated as Director from DRDO-Defence Food Research Laboratory, Mysore.

In the current study, he has provided overall guidance.

Appendix 'A'

Table 2. Symptom questionnaire for motion sickness including illusory feelings of motion (Golding, 1998)

Do you have any of the following symptoms right now ? (tick boxes)					
S. No.		None	Slight	Moderate	Severe
1	General discomfort				
2	Fatigue				
3	Headache				
4	Eye strain				
5	Difficulty focusing				
6	Increased salivation				
7	Sweating				
8	Nausea				
9	Difficulty concentrating				
10	Fullness of head				
11	Blurred vision				
12	Dizziness (eyes open)				
13	Dizziness (eyes closed)*				
14	Vertigo				
15	Stomach awareness				
16	Burping				