Pre-exposure Training as a Means to Reduce Vection Induced Symptoms of Motion Sickness

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Pre-exposure training as a means to reduce vection induced symptoms of motion sickness

By

Nicholas James Stapleton

A Thesis to be Submitted to the College of Arts and Sciences
in Partial Fulfillment of the Requirements for the Degree of
Master of Science in Human Factors & Systems

Embry-Riddle Aeronautical University
Daytona Beach, Florida
May 2013
Pre-exposure training as a means to reduce vection induced symptoms of motion sickness

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This thesis was prepared under the direction of the candidate’s Thesis Committee Chair, Dr. Jon French, Professor, Daytona Beach Campus, and Thesis Committee Members Dr. Albert Boquet, Professor, Daytona Beach Campus, Dr. Mustapha Mouloua, Professor, University of Central Florida, and Dr. Robert S. Kennedy, Professor, University of Central Florida. It was submitted to the College of Arts and Sciences in partial fulfillment of the requirements for the degree of Master of Science in Human Factors & Systems.

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Thank you to all the members of my committee for all the time and effort you have put into helping me complete this study. Your input and insight has helped make this study more comprehensive than I could have created on my own.

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Thank you for all of your assistance Lauren. This study could not have happened without you.
The focus of this study was designed to empirically examine the effect of inducing mild motion sickness as an ameliorative to a more severe motion sickness exposure. Twenty-seven participants were selected for this study based upon their susceptibility to motion sickness, that is, only people who were determined to be motion susceptible were tested. All participants were exposed to a motion sickness-inducing environment. Eighteen participants were trained to adapt to motion sickness by exposure to a milder motion sickness-inducing environment, ending either 6 hours or 24 hours prior to the more severe motion test environment.

Participants during the pre-exposure experimental conditions were trained to perform a mild motion sickness procedure, the Coriolis illusion. Following this training period, all participants were exposed to the motion sickness testing environment; the optokinetic drum. Tests results were measured subjectively through a conventional motion sickness questionnaire and objectively through the use of a cognitive test, balance tests, and salivary markers for cortisol and amylase. A series of statistical analyses were conducted to compare the no motion pre-exposure condition to the other mild pre-exposure conditions in their measures from the motion sickness testing environment using both parametric and non-parametric tests. Based upon prior research, it was hypothesized that the subjective responses, cognitive performance,
and biomarkers for motion sickness would decrease and balance would increase for the 6 and 12 hour exposures relative to the no pre-exposure condition.

The results indicated that the SSQ, Cortisol levels, SSS, and Finger to Nose Test were the only measures that captured the onset of motion sickness, and the SSQ was the only measure that identified any difference between training and non training groups. The SSQ indicated that recovery from motion sickness occurred at a faster rate following OKD exposure if the participant had training prior to exposure. No differences in symptoms were shown between the 6 hour and 24 hour training groups. The results may help to identify a more effective countermeasure to improve perceptual training in motion sickness inducing environments. Theoretical and practical implications are also presented.
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<td>Automated Neuropsychological Assessment Metrics</td>
</tr>
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<td>ANOVA</td>
<td>Analysis of Variance</td>
</tr>
<tr>
<td>A.R.E.</td>
<td>asymptotic relative efficiency</td>
</tr>
<tr>
<td>CTZ</td>
<td>Chemoreceptor trigger zone</td>
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<tr>
<td>EPSE</td>
<td>Extrapyramidal side effects</td>
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<td>FTNT</td>
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<td>NASA</td>
<td>National Aeronautics and Space Administration</td>
</tr>
<tr>
<td>OK</td>
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</tr>
<tr>
<td>OKD</td>
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<td>STS</td>
<td>Space Transportation System</td>
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<td>USB</td>
<td>Universal Serial Bus</td>
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<td>Abbreviation</td>
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<tr>
<td>VOR</td>
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<td>VSM</td>
<td>Velocity storage mechanism</td>
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Introduction

The human desire to explore has always pushed the limits of human ingenuity and physical ability. The great journeys across the Atlantic Ocean in the 16th century and the arctic explorations by ship of the 19th century, for example, tried human endurance using the latest technology. During the last half century humans have begun to explore space. In space exploration, human adaptation to microgravity has proven challenging. Space Adaptation Syndrome (SAS) is one of these adaptations and manifests as motion sickness in the microgravity environment (Smither, Mouloua, & Kennedy, 2008). Currently, pharmaceutical countermeasures against motion sickness only slightly dilute this problem. They usually interfere with cognitive ability, and can even have adverse effects since the impact of microgravity on absorption, distribution, or elimination of these drugs is not well known (Gandia, Saivin, & Houin, 2005).

This study evaluates the use of a behavioral countermeasure to motion disruption, that of pre-exposure to a less intense motion disruption than the anticipated motion sickness inducing event. This study could have implications for other human endeavors where motion sickness (microgravity, simulators, boats, airplanes) is restrictive. First a thorough background in motion sickness physiology is described then the most effective current countermeasures for motion disruption are discussed, and finally the hypotheses to be tested are stated.

Significance of the Study

This study was used to further analyze the impact of pre-exposure training on symptoms in a motion sickness inducing environment. The results of this study have implications in a wide variety of fields from shipping and cruise lines to aircraft operations to videogame and
simulators to space operations as motion sickness is an obstacle for safe operations in all environments where motion sickness can occur. Proper research into mitigating motion sickness symptoms can significantly improve comfort and safety in these environments. This study examines one way to mitigate motion sickness symptoms without the side effects that come with various pharmacological countermeasures. This study will also identify which measures have the sensitivity to identify differences between different groups.

**Statement of the Problem**

There are numerous motion sickness inducing environments which any given person may come across, including rough seas, simulators, and perhaps outer space in the near future. Motion sickness is a serious challenge to space exploration in the near future due to the impacts it has on safety, and the recent expansion of commercial space companies could mean more people flying into space. On Earth, the reliance on simulators for reduced training costs can lead to a form of motion sickness known as simulator sickness (Kennedy, Fowlkes, & Lilienthal, 1993). Ocean cruise lines have long been familiar with illness related to motion disruption as well, and the advent of space tourism will surely face the same challenges as sea travel, increasing our experiences of motion sickness (Stevens & Parsons, 2002). Research into mitigating motion sickness symptoms could significantly improve the comfort and safety in each of these environments.

**Purpose Statement**

The purpose of this study was to identify whether pre-exposure training had a significant impact on the symptoms of motion sickness. Additionally, different methods of measuring
motion sickness symptoms were evaluated for their sensitivity to changes between groups. The evaluations were conducted using an Optokinetic Drum (OKD) to induce motion sickness in a controlled laboratory environment.

**Hypotheses and statistical design**

This study used a between-groups design consisting of 3 groups: a control condition with no training, an experimental group receiving pre-exposure training ending 6 hours prior to OKD exposure, and another experimental group receiving pre-exposure training ending 24 hours prior to OKD exposure. Participants were each tested in the OKD once. The experimental groups of participants received a mild pre-exposure to an event designed to induce motion sickness known as the Coriolis illusion. The length of time following exposure of 24 hours was necessary to be compatible with Mouloua, Smither, Kennedy, Kennedy, Compton, & Drexler (2005), which found 24 hours to be effective in improving motion sickness symptoms in both the OKD and in virtual reality environments.

It was hypothesized that both pre-training groups (6 hours or 24 hours prior to OKD exposure) would experience a reduction in motion sickness symptoms associated with OKD exposure. It was additionally hypothesized that the impact of the countermeasures would be the more effective in the 6 hour after pre-training, because would be the exposure closest to the end of training. Similarly, the least effective pre-exposure training would be the 24 hour after training group, as the exposure would be the longest time after training. It was further hypothesized that cortisol and amylase measurements would be positively correlated with the severity of motion sickness symptoms.


Delimitations

The subjective measures were evaluated using a Kruskal-Wallis and Mann-Whitney non-parametric tests because of the ordinal nature of the data. All other objective measures were evaluated using an analysis of variance.

Power Analysis. The power analyses conducted in this study were performed using an adjusted power estimator for t-tests. According to Lehmann (2006), so long as homogeneity of variance is not violated, a power estimator for the t-test can be used to estimate power for the corresponding nonparametric test, the Mann-Whitney test. This adjustment takes the form of a difference in sample size. The correction on the sample size for the Mann-Whitney test (N_u) is equal to the sample size used in the corresponding t-test (N_t) divided by the asymptotic relative efficiency (A.R.E.), or Pitman efficiency, of the Mann-Whitney test, resulting in the formula of N_u = N_t / A.R.E. (Lehmann, 2006). According to Lehmann (2006), the A.R.E. of the Mann-Whitney test is equal to .955. The calculated N_u is then used in the standard power analysis for t-tests in place of the sample size. This study was using Mann-Whitney tests to analyze SSQ Total scores and the Nonparametric Lavine test indicated that homogeneity of variance was not violated. The difference between the resulting power values calculated from the parametric test was less than .1% higher than the nonparametric results.

Limitations and Assumptions

My limitations and assumptions are due to budgetary constraints, the size of the participant groups, and time constraints, measures such as melatonin were cut from the original study. Due to the limited budget, a single salivary sample was collected immediately prior to
OKD exposure rather than a resting day baseline. This meant that a baseline was only taken immediately prior to OKD exposure, and could possibly impact the results of the salivary cortisol and amylase levels. Additionally, participants were not tested beyond 30 minutes following exposure to the motion sickness inducing environment, except with the Simulator Sickness Questionnaire (SSQ) to determine if a participant was fit to leave, when applicable.

**Definition of Terms**

Asymptotic relative efficiency: The assumed adjustment needed when converting the sample size of a nonparametric test to its corresponding parametric test when calculating the result of a power estimator (Lehman, 2006).

Behavioral Countermeasure: Countermeasures of motion sickness that do not rely on the use of pharmaceutical means (Yen Pik Sang, Billar, Golding and Gresty, 2003).

Imagineer: A person who devises and implements a new or highly imaginative concept or technology, in particular one who devises the attractions in Walt Disney theme parks (OED Online, 2013).

Pharmaceutical Countermeasure: Countermeasures of motion sickness that involve the use of substances created or used to mitigate the symptoms or susceptibility to motion sickness (Takeda et al., 1993).

Pittman efficiency: See asymptotic relative efficiency.

Simulator Sickness: A type of motion sickness that occurs due to the effects of simulators on the human body (Kennedy, Fowlkes, & Lilienthal, 1993).
Vection: A specific type of simulator sickness where apparent motion causes the symptoms (Smither, Mouloua, & Kennedy, 2008).
Review of the Relevant Literature

Motion Sickness

Motion sickness has affected humanity for as long as humans have used modes of transportation other than walking. The term motion sickness does not adequately describe the condition, since the symptoms are not associated with a true sickness, but a normal response to a disturbance of proprioceptive feedback. Symptoms can range from a mild inconvenience of a headache or nausea to temporary incapacitation. General symptoms include nausea, eyestrain, blurred vision, headache, vertigo, and stomach concerns (Kennedy, Lane, Berbaum, & Lilienthal, 1993). Less familiar symptoms, such as Sopite Syndrome (malaise), oscillopsia and retinal slip (which are related to difficulty focusing the eyes), and postural instability (commonly called the leans) can also occur (Kennedy, Massey, & Lilienthal, 1995). Any unusual or illusory motion can also cause motion sickness. Symptom severity and susceptibility vary greatly from person to person, and even within an individual experiencing different causes of motion sickness (Stevens & Parsons, 2002).

Causes. Not everyone susceptible to motion sickness succumbs to symptoms while experiencing every type of motion sickness each time. Most of the causes are fairly well known. Sea sickness has been around as long as humans have taken to the sea. The word nausea stems from naus, the Greek word for ship, literally translating to “the sickness of ships” (Merriam-Webster, 2008). The loping camel is another ancient source of motion sickness for riders without experience on the sea (Stevens & Parsons, 2002). As time passed, humans continued to suffer from sea sickness as well as new forms of motion sickness from the technology that we created.
Besides sea sickness, the most common cause of motion sickness over the past 100 years has come from passengers in ships, automobiles and aircraft.

Modern technology has provided many new ways for people to get motion sick. The advancement of virtual reality and computer simulated environments has been impacted by motion sensitivity. Simulator sickness has become a very common term in the modern world and has been a challenge ranging from programmers building virtual environments to Imagineers at Walt Disney World making live-action simulator rides. The apparent motion that causes symptoms in the case of simulator sickness is known as vection. As technology pushes the boundary of what motion experiences we encounter, considerations should be made to ensure that those exposed are not impaired by the cognitive and physical debilitations of motion sickness.

When astronauts first arrive in space, they experience something that they have never felt for an extended time, the lack of a significant gravity. There are many physiological issues associated with extended exposure to a microgravity environment, including muscle atrophy, compromised immune systems and disorientation. Presumably, disruption of the proprioception system is ultimately the cause of motion sickness by the occasionally intense mismatch of vestibular, ocular and kinesthetic sensory information. In space, the sensory confusion can lead to motion sickness known as Space Adaptation Syndrome (SAS), which can last from 2-4 days, preventing astronauts from performing potentially mission essential tasks (for example, emergency egress). Approximately 70% of astronauts suffer symptoms of SAS, but eventually, all astronauts and their proprioception system adapts to the new microgravity environment of space and they stop reporting symptoms (Lackner & DiZio, 2006).
It is prohibitively expensive to simulate the conditions that generate SAS on the earth but perhaps parallels exist in laboratory techniques for inducing motion sickness. This would allow humans to test SAS countermeasures without the costs and dangers of space travel. An Optokinetic Drum (OKD) is able to induce a similar type of motion sickness through vection in a safe, laboratory environment producing a similar pattern of symptoms at SAS. Because they are both motion sickness, it is likely that if pretreatment improves the symptoms caused by the OKD, then pretreatment will also improve symptoms of SAS.

One particularly strong example of the hazards of SAS became clear on STS-51-D, when United States Senator Jake Garn flew on Space Shuttle Discovery as a congressional observer and payload specialist. During Senator Garn’s flight, he suffered from the strongest symptoms of SAS NASA has on record. Astronauts within NASA jokingly use “one Garn” as a measuring stick for the worst SAS symptoms can get, where most astronauts will reach a tenth of a Garn in their symptoms (Butler & Stevenson, 1999).

**Special Symptoms.** Some side effects of motion sickness are less known than the general symptoms listed above, though they are no less consequential. These include Sopite Syndrome, oscillopsia, retinal slip, and postural instability. These symptoms can be troubling for both earthbound forms of motion sickness, as well as SAS.

Sometimes the only symptom of motion sickness experienced is a condition known as Sopite Syndrome. This condition is characterized by drowsiness, fatigue, and in long-term cases, mental depression. A study by Graybiel & Knepton (1976) found significant evidence to show that fatigue and drowsiness manifest in acute, or short-term, motion sickness, and mental depression may manifest as well during long term, or chronic, exposure to motion sickness.
stimuli. This symptom of motion disruption is not easily recognized as originating from a motion experience (Graybiel & Knepton, 1976).

Motion of the eyes also is a commonly overlooked symptom of motion sickness. One measure of retinal slip is the amount of motion the eye sees during movement. Normally, the eye can compensate for motion to keep acuity when retinal slip, or the movement of the image across the eye, is within 4 degrees per second. Exceeding this, visual acuity decreases and causes blurring or other illusions. While experiencing motion sickness, the ability of the eye to compensate for motion can decrease, meaning motion is more likely to exceed the current limits of the eye to compensate for retinal slip. A second symptom of motion sickness relating to the eye is known as Oscillopsia, or the illusion of movement when remaining stationary. This is common of constant motion, occurring when spinning for a period of time long enough for the vestibular system to adapt, then stopping. The stabilized vestibular system would now be indicating motion despite being stationary and the room may appear to be spinning, which is oscillopsia (Leigh, Dell’Osso, Yaniglos, & Thurston, 1988). Both excess retinal slip and oscillopsia can be distracting and disorienting symptoms of motion sickness.

As astronauts adjust to the microgravity environment of low-Earth orbit, they are setting up for another period of adaption that will be required upon landing. According to both Black, Paloski, Doxey-Gasway and Reschke (1995) and Black et al. (1999), evidence indicates that all returning astronauts exhibit a period of postural instability upon landing, generally ranging from two to five days. During this time period, astronauts would face clumsiness with movements, difficulty walking in a straight line, persisting sensation aftereffects (the feeling of being unbalanced), vertigo while walking, vertigo while standing, nausea, difficulty concentrating, and vomiting (Bacal, Billica, & Bishop, 2003). Black et al. (1998) found that irregular motion, such
as eccentric pitch rotation, disrupted adaptation and could prolong or exacerbate symptoms. Generally, both studies found a recovery within 2-4 days, consistent with the expected 2-4 days during space adaptation, though extreme cases have lasted as long as 5 days (Black et al., 1998).

**Proprioception**

To understand why motion sickness occurs, it is important to understand the physiological processes that detect motion in the body. Proprioception is the body’s ability to sense where different body parts are located relative to each other. The proprioception system can be broken up into the vestibular system, which is designed to detect movement of the head, the kinesthetic system, which discerns position, and a visual aspect, the visual cues to orientation. These signals from both systems are sent through the visuo-vestibular pathways known as the velocity storage mechanism (VSM), where the information is processed and compared to provide the interpretation of body position and movement (Ventre-Dominey, Luyat, Denise, & Darlot, 2008). In accordance with the Neural Mismatch Theory, a leading theory of motion sickness, the discrepancies between the vestibular and kinesthetic inputs are detected by the VSM that cause motion sickness symptoms. These discrepancies stimulate the emesis center and the chemoreceptor trigger zone (CTZ), and can cause the nausea and vomiting generally associated with motion sickness.

**Vestibular Systems.** The vestibular system is designed to detect the position of the head. It is located entirely inside the inner ear and consists of the semicircular canals and the otoliths and controls the vestibulo-ocular reflex. Signals from the vestibular system are sent to the vestibular nuclei of the brain stem, where they are combined with signals from the visual system
and the rest of the proprioception system to establish postural and positional data for the body (Lackner, 2004).

**Semicircular canals.** The three semicircular canals are responsible for discerning rotational cues for the brain. Each of the canals is oriented in a different direction to best gather rotational information. The horizontal canal, also known as the lateral canal, is located along the lateral axis, sensing the rotation of the head left and right. The superior semicircular canal, or the anterior canal, is located to sense motion around the rostral-caudal axis, or the tilt of the head, as in a nod or leaning the head backwards. The final canal, known as the posterior canal, is positioned to detect rotations along the sagittal plane, or the rotation of the face clockwise or counterclockwise, as would be done when cocking the head. The canals are shown in Figure 1.

The canals are full of fluid known as endolymph which, under a stable condition, is unmoving in the ear. As soon as motion of the head occurs, the canals and the bony labyrinth move, and the endolymph lags behind due to inertia. The difference between the speed of head movement and the inertial lag of the endolymph varies with the speed of rotation in the head as well as the direction of rotation. As the fluid moves, it passes the ampullae, a location in the canal containing the cupula. The cupula in each semicircular canal is held in a neutral location until movement adjusts it.

Hair cells around the cupula sense the movement and transfer the information to the brain via the vestibulocochlear nerve (Lackner, 2004). These hair cells are mechanoreceptors that fire constantly, regardless of motion. The rate of fire varies depending on the direction and magnitude of the motion of the cupula, as seen in Figure 2, increasing the rate of fire when moved in one direction, decreasing rate of fire in the other (Buytaert, 2011). Between each of
the three semicircular canals in each inner ear, the vestibular system is able to identify any rotational motion of the head or body.

Figure 1. The Vestibular System. Note. From Rutka (2004)

Figure 2. Hair cell rate of fire (Buytaert, 2011).
**Otoliths.** Like the semicircular canals, the saccule and utricle sense the motion of the head, but instead of measuring the rotational motion, they are responsible for measuring the linear acceleration, including gravity. The saccule and utricle are oriented to be sensitive to different directions of movement. The saccule provides cues to vertical acceleration, like standing from a squat, where the utricle is oriented to register horizontal movement (Beule & Allum, 2006). Both are also filled with endolymph, just as the semicircular canals are, and also contain hair cells. The hair cells in the otoliths are topped with hexagonally shaped calcium carbonate crystals known as otoconia. As motion occurs, the inertia of the otoconia causes the hair cells to bend which changes the signals being sent to the VSM (Buyaert, 2011).

**Vestibulo-ocular reflex.** When the position of the head is adjusted while the eyes are focused on an object, the eyes would lose focus if not for an automatic response from the body. This response is known as the vestibulo-ocular reflex (VOR). This results from the nerves connecting the vestibular system and the brain in communication with nerves that relay information to the ocular muscles around the eyes. This provides near-instantaneous compensation to the eye, and allows for tracking despite any motion that the head may undertake. This is where the primary impact of the VOR on motion sickness stems from. Since the human eye is able to continue to track during motion, a person can make out the details that will be important in identifying visual cues for motion (Raphan & Cohen, 2002).

**Visual Cues.** Apparent or illusory motion can cause motion sickness as readily as actual motion in many people. This is due to the body’s reliance on visual cues to interpret motion as well as the vestibular and kinesthetic inputs. These cues are objects in motion, especially relative
to the perceived horizon for motion around 2 planes; the sagittal plane and the rostral-caudal plane (Duh, Parker, Phillips, & Fumess 2004). While motion cues, can lead to motion sickness, it is not a necessity, as Graybiel (1970) found that visually-impaired persons can still experience motion sickness symptoms.

**Kinesthetic system.** When well-coordinated adults climb stairs, they have no need to watch their feet making each step, yet are still able to climb the stairs quickly without much thought. This is because of kinesthesia, or the body’s natural ability to judge how it is oriented based upon muscle, tendon, and joint positions. Nerves connecting every muscle, tendon and joint in the body send information about these positions to the brain where it is organized into a mental picture of how the body is positioned. Subconsciously using the kinesthetic sense, one can perform tasks out of sight, while blindfolded, or without thought due to this sense. This sense is also used to differentiate between head motion and body motion; for example, it is used to differentiate between tilting your head to the left and your whole body being tilted left. Part of this sense is based upon gravity’s pull upon different parts of the body, such as more weight being present on one foot due to the whole body tilting in the previous example. Together with the vestibular system, this makes up all of the proprioception system; how the body determines its position in the environment.

Organs that make up the kinesthetic system include Golgi tendon organs, muscle spindles, and mechanoreceptors located in the skin and joint tissue. Golgi tendon organs are a stretch- and compression-sensitive organs located within muscles throughout the body. These organs react to compressions that occur when a muscle contracts or tightens, and the stretching that occurs when a muscle is returned to it lengthened state, sending signals through the afferent nerves to the brain, which notes the new position (Prochazka, Gillard, & Bennett, 1997). Muscle
spindles are similar to Golgi tendon organs in that they are located within muscles and respond to stretching and contracting forces, however they are constantly providing information on their position. They are located within the muscle tissue and are the entire length of the muscle. When the muscle contracts or returns to a resting state, the muscle stretches or contracts respectively, and alters the signal being sent along a separate afferent neuron to the brain. Due to the muscle spindles being a specific length, they can also be used to determine joint position, since the brain can interpret which muscles should be stretched or loosened for a joint to be in a particular position (Vallbo & Al-Falahe, 1990). The primary mechanoreceptors used in kinesthesia are Ruffini and Pacinian corpuscles. Ruffini corpuscles measure stretching of the skin and tissue around joints, and are relatively slow to adapt to the new position, sending signals of their altered state for long after the initial change has occurred. Conversely, Pacinian corpuscles are rapidly adapting and are sensitive to touch and vibration. Due to their location throughout the skin, these mechanoreceptors are used in kinesthesia to identify where a body part is located based upon what other tissue it is touching (Hagert, Forsgren, & Ljung, 2005). For example, a brain is able to interpret an arm’s position due to the fact that it is touching the side of the chest to the bottom of the rib cage.

These organs are naturally calibrated to working in a 1g gravity environment, and as such, when introduced into a microgravity environment, the inputs are weaker, causing a weaker or even incorrect signal being sent to the brain. Stevens & Parsons (2002) indicated that the kinesthetic system alone is unable to induce motion sickness, though they also state that it is likely that inputs from this system, when in conflict with vestibular or visual signals, can lead to or strengthen motion sickness symptoms.
**Emesis Center and CTZ.** The emesis center is responsible for nausea and vomiting. It is located in the reticular formation and collects data from the CTZ, a chemoreceptor site located in the medulla, in the fourth ventricle. The CTZ receives inputs from the blood in order to collect neurotransmitters to excite or inhibit the emesis center. The neurotransmitters that excite the emesis center, or agonists, include Histamine, Muscarinic cholinergic, Dopamine, Serotonin (5-HT$_3$), and Substance P. Substances that counter the agonists, known as antagonists, include Promethazine, Atropine, Droperidol, Serotonin (5-HT$_3$) receptor antagonists, and NK-1 receptor antagonists. When the CTZ is excited, it sends signals down the neuronal pathways to the emesis center, which can cause the emesis center to reach its threshold, which results in nausea and vomiting. The receptors on the CTZ can be seen in Figure 3. The emesis center can also be excited directly through higher centers, such as vision and taste, the pharynx, and GI tract distension (Watcha & White, 1992).

![Figure 3. The Chemoreceptor Trigger Zone and the Emetic Center (Watcha & White, 1992).](image-url)
Motion Sickness Theories

Though we have an understanding of how the systems important to motion sickness work, the differences and variations between people and the near-unpredictable variability of symptoms and severity make finding a comprehensive theory that adequately explains the many aspects of motion sickness quite elusive. Therefore, there are several theories that attempt to explain how motion affects the human body and causes the symptoms associated with motion sickness. Three of the most prevalent theories are Neural Mismatch, Postural Instability, and Poison Response.

Neural Mismatch Theory. The most prevalent theory is the Neural Mismatch Theory. It also is known as the Sensory Rearrangement theory and the Sensory Conflict theory. This theory states that motion sickness is caused by a mismatch in signals from the different internal systems responsible for identifying the position of the body, proprioception. These signals give the competing information about body orientation in a space, which conflict with one another and with signals interpreted during past experiences. The past experiences, also known as exposure-history, form the pattern which the body typically uses, namely the natural motion of the body on stable land.

All motion sickness symptoms can be divided into two categories of conflict under the Neural Mismatch Theory. These categories are visual-inertial conflict and canal-otolith conflict. Visual-inertial conflict occurs when discrepancies exist between the visual system and the proprioception systems. Canal-otolith conflict occurs completely within the vestibular system, specifically between the semicircular canals determining rotation and the otoliths determining head position though gravitational pull (Cobb, Nichols, Ramsey, & Wilson, 1999).
Also covered under the Neural Mismatch Theory, three types of sensory conflict can occur, known as Type I, II, and III conflicts. A Type I conflict is where two receptors are sending information to the brain that either contradicts or fails to correlate with one another. Type II conflicts occur when one signal is being received by the brain while another signal that also identifies the type of motion occurring with respect to the first signal is missing, in the case of motion not occurring but the visual indications of movement, or visual indications but no proprioceptive sensation of movement. Type III conflicts occur when the opposite of a Type II conflict occurs, or when the physical sensations are present, but the visual indications are telling the brain that movement is not occurring (Cobb, Nichols, Ramsey, & Wilson, 1999).

A key point in the Neural Mismatch Theory is the ability to acquire adaptation to the disruption these mixed signals cause. As Reason (1978) states, the theory bases this assumption on the ability for seasick sailors to adapt over time, to get their sea-legs back. This is common to SAS as well, as susceptible astronauts generally adapt to their environment within the first two to four days of the mission.

**Postural Instability Theory.** A competing theory on the causes of motion sickness relates to Postural Instability. This theory stems from the basis that the human body in action has its own natural sway. The body suffers symptoms when the movement of a ship or camel has a different sway pattern, or in the case of simulation, virtual reality, and space, a lack of sway. In other words, any motion that is contrary to a person’s natural frequency and magnitude of movement will cause motion sickness symptoms. One of the main hypotheses of this theory states that the intensity of the motion sickness symptoms is directly proportional to the intensity
Poison Response Theory. A third explanation for the causes of motion sickness is the Poison Response Theory. Unlike the other two, this theory states that the signals being sent to the brain are not the cause of motion sickness, but rather the use of the body’s reflex response to poisoning that cause the symptoms. The assumption is that the brain interprets the different, confusing sensory inputs motion disruption causes as indicators that they body is being poisoned and takes action. Evidence has shown that the vestibular system may act as a secondary mechanism to the chemoreceptors, sending a signal to the CTZ for the body’s poison response to occur (Hershkovitz, Asna, Shupak, Kaminski, & Bar, 2009). The Poison Response theory relies on two primary components: stomach emptying and stress response. Alkalosis, or the heightened pH of blood, can be caused by a loss of hydrogen ions, and can cause vomiting and stress symptoms such as those associated with motion sickness. This hydrogen ion loss can be associated to dissolving carbon dioxide into the fluids of the stomach during rapid churning, causing carbonic acid, which attracts hydrogen ions. Sodium loss through perspiration and the release of antidiuretic hormones can also cause similar symptoms. This theory provides explanation that fills some of the holes in the Neural Mismatch and the Postural Instability theories (Lackner, 2004).

Each of these three theories explains a different aspect of what causes motion sickness, though none of these theories are comprehensive. Warwick-Evans et al. (1998) performed a study designed to evaluate the Postural Instability theory and compare it with the Neural Mismatch theory and found more evidence supporting the Neural Mismatch Theory.
Specifically, they found that a reduction in the impacts of postural instability does not directly relate to the reduction of motion sickness symptoms, thus presenting a strong case that the Neural Mismatch Theory better describes the theory behind motion sickness. Critics of the Poison Response theory state that subjects lacking a functioning vestibular system do not experience motion sickness, but proponents of the theory state that experimental animals are less sensitive to drugs that induce vomiting following the removal of the entire vestibular system (Lackner, 2004). There is therefore no theory that adequately covers every aspect of motion sickness, and this leads to a difficulty in the predictability of motion sickness symptoms. In the future, a comprehensive theory may be established and would certainly include many aspects of the individual theories just discussed.

**Measurements**

Though the exact theory of motion sickness is unclear, how the many symptoms are measured, including disorientation and nausea is well-documented. To gauge the effectiveness of countermeasures, researchers quantify symptoms. This section covers a portion of the techniques used to quantify these symptoms, concentrating on ones which will be used in this study. These techniques can be classified as subjective measures, measures of fatigue, cognitive, and biochemical measures.

**Subjective.** Subjective measurements are among the most commonly used when researching. They are relatively easy to acquire and can put numbers to aspects that are difficult to evaluate otherwise. For some aspects, subjective measurements can be the only source of data. One of the drawbacks of subjective measurements though, stems from the lack of quantifiable
data from the measurements. For example, if a subjective measure is asking for nausea on a scale of one to ten, one being low feeling, ten being high, each participant will have a different belief of what each number in the range represents, leading one participant to mark “6” while a different participant might mark “4” for the same level of the symptom. Another common drawback of subjective measures stems from pride in participants, especially when studying fatigue. Many participants, in an attempt to show how tough they are, will refuse to admit they are tired, sore, or sick. This is usually combated by testing individually, in an environment where the participant doesn’t feel the need to show off for anyone. A third drawback was illustrated by Young, Adelstien, and Ellis (2007), when they found that administering a pre-test questionnaire made it significantly more likely for participants to suffer from symptoms. Three subjective measures that will be utilized in this study are the Motion Sickness Susceptibility Questionnaire (MSSQ), the Simulator Sickness Questionnaire (SSQ), and the Stanford Sleepiness Scale (SSS).

The MSSQ was designed in 1991 in an attempt to predict the susceptibility of a participant to motion sickness symptoms. Participants using this questionnaire self-rate themselves in 12 situation based upon past experiences with motion sickness, then 2 questions on how they feel they experience symptoms compared to others, and finally 2 questions about any past or present health concerns. Results are analyzed and compared to the results of participants from previous studies using the test (Griffin & Howarth, 2000).

In order to get an accurate representation of motion sickness symptoms, many questionnaires have been developed. The Pensacola Motion Sickness Questionnaire (MSQ) was a popular prior to the early 1990’s, though some concerns led to it becoming less commonly used. One major concern of the MSQ was the sensitivity of the tests used to develop it. These tests consisted of stimuli that would likely produce vomiting, or come close to it. Kennedy et al.
(1993) found low sensitivity to early symptoms of motion sickness, requiring more severe stimuli that scientists were using to identify motion sickness was occurring.

One such questionnaire that has become common in recent studies has been the Simulator Sickness Questionnaire (SSQ) developed by Kennedy et al. (1993). The SSQ was derived from the MSQ, with the full SSQ asking for the same 30 symptoms the original MSQ asked from participants, though only 16 are scored. This selfanswered questionnaire is presented to participants in motion sickness studies and allows for a four point (0-3) scale, 0 being no symptoms and 3 being severe. The 16 scored symptoms of the Simulator Sickness Questionnaire were divided into three symptom clusters based upon a factor analysis: Oculomotor, Disorientation, and Nausea. In these clusters of Kennedy et al. (1993) include eyestrain, difficulty focusing, blurred vision, and headache in the oculomotor cluster, dizziness with eyes open or closed, and vertigo in the disorientation cluster, and increased salivation, stomach awareness, and burping fall into the nausea cluster. General discomfort, fatigue, sweating, nausea, fullness of head, and difficulty concentrating were identified as symptoms, but did not fit into any of the three clusters though are no less important. Other potential symptoms were considered and reviewed, but were ultimately ruled out due to the lack of frequency or occurrences with other identified symptoms (Kennedy et al., 1993).

The SSQ has been identified as an accurate method of classifying motion sickness symptoms well beyond just simulator sickness, including many other forms of motion sickness. Both Palmisano, Bonato, Bubka, and Folder (2007) and Bubka, Bonato, Urmey and Myceqicz (2006) showed the SSQ was successful in capturing motion sickness symptoms when participants were exposed to vection. The SSQ was expected to fulfill the needs of this study.
Fatigue. As discussed earlier, Sopite Syndrome will occasionally be the only symptom someone sensitive to motion sickness will suffer from. To ensure that all participants suffering from the effects of motion sickness are identified, it is important to measure this induced fatigue as well. The SSS is a simple subjective measure for identifying fatigue, and was used in this study. It used a simple 7 point self-testing scale with definitions specific to each level to measure sleepiness. The specific definitions for each level help to eliminate some of the variability between participant responses at the same level of alertness (Hoddes, Zarcone, Smythe, Phillips, & Dement, 1973). Participants selected the level that best applies to them. This data was used to identify fatigue and to check for Sopite.

Cognitive Test. Yet another measure that was used in this study was the Switching Test. This test, which was made up of one section of the much larger Automated Neuropsychological Assessment Metrics (ANAM) test, evaluates mental flexibility and shifting. The three parts of this dual test section were the Manikin test and the Mathematical Processing Task, with a third task requiring attention to a cue that tells the participant when to move between tests. The Manikin test shows images of a human suspended in different directions, forward, backwards, upside-down, with an object in either outstretched hand. The participant must identify which of the human’s hands a particular object is in. This is usually considered a test of spatial reasoning. The Mathematical Processing Task presents a series of simple mathematical equations in the format of (X+Y-Z=?). In this task, each of X, Y, Z, and the result are numbers between 1 and 9, though the answer is never 5, where each number is equally probable in any position except 5. The Switching test can be administered in multiple ways, including both Manikin and Mathematical Processing on the same screen with an arrow pointing to one another to identify
which question to answer or only one on the screen at a time alternating randomly between Manikin and Mathematical Processing (Reeves, Winter, Bleiberg, & Kane, 2007). The results of this test indicate and predict any cognitive impairment as a result of motion sickness.

**Balance.** Balance is often cited as a symptom of motion sickness, therefore two balance tests were given as part of this study. The first was a heel-to-toe test, in which participants walked a straight line for 7 steps, heel-to-toe, turned on the balls of their feet, and then returned along the line an additional 7 steps. Both completion time and missed steps were recorded.

The second balance test was an adapted Finger-to-nose test (FTNT), where the participant closed their eyes, stand with one foot approximately twelve inches above the ground, and extend both arms horizontally. Then the participant was instructed to bring their middle finger tip to their nose with one hand, return it to the original extended position, and repeat with the other hand. The number of times the participant either lowered their raised foot or needed assistance in balancing was recorded.

**Biochemical.** Endocrine measures can provide strong insight into motion sickness as well. Cortisol is a hormone released by the adrenal gland that can be easily collected in saliva samples. It has been identified as an indicator of stress levels. Chouker et al. (2010) found a significant correlation between the increasing intensity of motion sickness and increasing cortisol levels. Comparing cortisol levels in participants following vection induced motion sickness to levels prior should provide another objective indication of motion sickness.

Norepinephrine (NE) is another biochemical indicator of that is useful in objectively identifying symptoms associated with motion sickness. Specifically, NE is an indicant of stress
and has evidence that it may be even more sensitive an indicator than blood pressure or heart rate to stress (Aragaki, Etoh, Hojo, Takaj, & Nishikawa, 2003; Chatterton, Vogelsong, Ellman, Lu, & Hudgens, 1996). It is possible that, combined with indications from other biochemical markers, NE would provide an objective indication of motion sickness. NE can be tracked through salivary amylase (Skosnik, Chatterton, Swisher & Park, 2000).

**Countermeasures**

Countermeasures for motion sickness have been around since the early days of boat and camel travel. Many countermeasures have scientific backing, while others are pure fable. Countermeasures can be divided between two major categories: Pharmacological countermeasures and behavioral countermeasures. Both methods have their benefits and drawbacks, but both can be applied to motion sickness symptoms and provide varying results and side effects in combating SAS.

**Pharmacological.** Many different pharmacological countermeasures have been developed for quelling motion sickness symptoms. Many have successful application on Earth that would not be suitable for use in space due to unwanted side effects. As seen in Table 1, many pharmacological countermeasures have one common side effect, drowsiness. This can be a dangerous symptom when paired with fatigue stemming from motion sickness already, especially in an emergency situation. Extrapyramidal side effects (EPSE), such as the inability to move, akinesia, or the inability to remain stationary, akathisia, can be just as deadly. If an astronaut were suffering for symptoms like drowsiness, lack of concentration, vertigo, or EPSE, he or she might not be able to adequately complete checklist items, follow through with emergency procedures, or even identify that an emergency is occurring which requires his or her
attention. Until a pharmacological countermeasure that does not subject users to side effects such as these are identified, another solution to motion sickness symptoms must be found.

Table 1. Common pharmacological countermeasures to motion sickness, their success, and side effects.

<table>
<thead>
<tr>
<th>Pharmaceutical</th>
<th>Type</th>
<th>Effectiveness</th>
<th>Side Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dramamine</td>
<td>H1 Antihistamine/Muscarinic antagonist</td>
<td>Some²</td>
<td>Drowsiness³</td>
</tr>
<tr>
<td>Chlorpheniramine</td>
<td>H1 Antihistamine</td>
<td>Effective³</td>
<td>Drowsiness, Lack of Concentration, indigestion²</td>
</tr>
<tr>
<td>Scopolamine (Hyoscyamine)</td>
<td>Anti-cholinergic</td>
<td>“great success,”¹</td>
<td>Dry mouth, drowsiness, mydriasis (pupil dilation)¹</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Effective in adaptation²</td>
<td></td>
</tr>
<tr>
<td>Promethazine</td>
<td>Anti-cholinergic</td>
<td>“great success,”¹</td>
<td>Drowsiness, vertigo, constipation, Dry mouth, impaired alertness¹</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Subjectively considered ineffective²,⁴</td>
<td></td>
</tr>
<tr>
<td>Ondansetron/Ramosetron</td>
<td>5HT₃ Receptor Antagonist</td>
<td>Subjectively considered ineffective²,⁴</td>
<td>Headache, thirst, constipation, diarrhea, fever, EPSE (akinesia and muscle spasms)⁵</td>
</tr>
</tbody>
</table>

Note. From ¹(Lackner & DiZio, 2006), ²(Takeda et al., 1993), ³(Weinstein & Stern, 1997), ⁴(Hershkovitz et al., 2009), ⁵(Shi et al., 2007)

Behavioral. Behavioral countermeasures have been in use nearly as long as motion sickness has been an issue. They have the general benefit of always being available, despite mixed effectiveness between people. Behavioral countermeasures can be into between two different types: in situ and pre-exposure training.

In situ. In situ countermeasures, or countermeasure performed while motion sickness is occurring, have varying effectiveness on Earth. In space, many of these same countermeasures are much less effective than on the ground. Yen Pik Sang, Billar, Golding and Gresty (2003)
found that controlled breathing and listening to calming music delayed the onset of motion sickness, though did nothing to prevent or lessen symptoms of motion sickness when sickness eventually occurred. This follows similar research that indicates that most behavioral countermeasures used on Earth do little to nothing for astronauts facing SAS. The theory behind this result is on Earth, these countermeasures can end the sensory conflict, whereas in space, the conflict will continue to persist until the environment is left. Adaption time and symptom severity have been shown to relate to the behavioral activities of astronauts experiencing the symptoms. Lackner and DiZio (2006) found that excessive head motion or orientation different to that of other astronauts on the space station would increase SAS symptoms and extend adaptation time of astronauts during the first days of their mission. Therefore, extrapolating from those results, avoiding rapid head movements or abnormal orientation in comparison to other astronauts should be effective in decreasing the length of time SAS impacts an astronaut.

**Pre-Exposure Training.** While little can be done in the environment to decrease the symptom severity and adaptation time of an astronaut currently experiencing motion sickness, studies have examined the possibility of increasing adaptability prior to reaching the microgravity environment. One such method of decreasing the symptoms associated with motion sickness that has shown to be effective is pre-exposure training. The usefulness of pre-exposure training stems from the learning aspect of the Neural Mismatch Theory which states that it is possible to acquire resistance to the type of motion sickness being presented. Continuing to build off the aspects of this theory, motion sicknesses of the same type should react the same way to pre-exposure.
Smither et al. (2008) found positive results for participants receiving training via self-propelled rotation simulation on symptoms caused by OKD and a virtual reality device 24 hours after the last training session. Similar results were found by Clement et al. (2001) when comparing cosmonaut vestibular training to rotating chair motion sickness exposure.

Pre-exposure training has never been adequately tested prior to an actual space launch. As stated by Clement et al. (2001), the expense of testing the hypothesis of the training effects on SAS, however the earthbound results are promising, and the in situ experiments are not significantly effective. Therefore, it would seem that the most likely countermeasure for SAS in the future would be pre-exposure training.
Method

Participants

Participants were recruited through advertising in the college newspaper and flyers posted around campus. All participants were compensated monetarily for their time. There were a total of 27 university students who participated in this study ranging from 18-24, all of whom were tested to ensure susceptibility to motion sickness through the use of the 75% threshold of the MSSQ (Appendix A). The participants were randomly divided into 3 groups. Group 1 and 2 received the self-induced Coriolis pre-exposure training. Group 1 was exposed to the OKD 6 hours after the completion of the last training session, while group 2 was exposed to the OKD 24 hours after the completion of the last training session. Group 3 received no training prior to OKD exposure.

Materials and Apparatus

The study was conducted in two different rooms in accordance with experimenter (single) blind procedures. Pre-exposure training occurred in one room administered by one person, while the OKD exposure occurred in a second room administered by a second person. Standard office conditions were used for both rooms.

Optokinetic exposures occurred in the OKD constructed at Embry-Riddle Aeronautical University. This drum has been used to successfully induce motion sickness in the past. The device consists of a 1.6 meter tall drum with a radius of 2.1 meters, which can be rotated with a motor attached to the center of the device. A stationary chair set at a viewing distance of 60 centimeters from the drum allowed participants to be seated during the experiment for both
safety and control. The drum is covered in a curtain that has a pattern that provides an indication of motion when the drum is spinning. The signal mismatch between visually perceived motion due to the drum spinning and lack of physically perceived motion due to remaining stationary provides a sensory conflict, inducing motion sickness. An example of a participant seated in an OKD can be seen in Figure 4, though the pictured drum is much smaller than the one being used in the present study. This study will be making use of a sandpaper-like pattern, which can be seen in Figure 5.

![Figure 4. An Optokinetic Drum (OKD) in operation.](image)

*Note.* The actual drum is not transparent.

Measurements were conducted in a single test battery one prior to and twice following OKD exposure. The tests that will be administered were the SSQ, the SSS, the switching test, the balance tests, and the biochemical amylase collection.
The SSQ was given verbally, with the researcher recording the results on paper. The SSS was given in paper form. Results were compared to the participants’ initial responses to determine the effect of the OKD, then compared with the other participants to discern the effect of the primary hypothesis. Multiple Friedman and Mann-Whitney U tests were conducted to compare the results.

The Switching test was administered on USB drives to participants. A copy of the switching test was given to participants one week prior to OKD exposure, and participants were expected to consistently reach a score over 90% prior to testing. Data collected from this test were accuracy and mean reaction time, as well as a correlation between the two. The results were statistically analyzed for significance.

The saliva to be tested for cortisol and alpha-amylase were collected during the test battery prior to and following OKD exposure. The collected saliva were sent for biochemical
assay to a commercial facility, Salimetrics, LLC., to analyze for cortisol. These samples were collected by the Salimetrics Passive Drool tool and frozen until shipping.

Two methods of testing a participant’s balance were used. A heel-to-toe line walk measured the length of time required to complete 14 steps and a turn, and an adapted FTNT was used to identify how many times a participant was required to use exterior support while attempting a moving balance test.

**Experimental Design**

The experiment was conducted to study three aspects of motion sickness. The study was separated into two between-groups designs and one within-subject experimental design.

**Study 1.** Analyze the effect of pre-exposure training on motion sickness symptoms

**Study 2.** Analyze the effect of a shorter time delay between pre-exposure training and a stronger stimulus has on motion sickness symptom strengths rather than a longer delay.

**Study 3.** Analyze the efficacy of measuring melatonin, cortisol and alpha-amylase to ascertain the occurrence of motion sickness.

**Independent variables.** There were two independent variables for this study. The first independent variable was experiencing pre-exposure training or not. The second independent variable was the length of time between the last training session and exposure to the OKD.

**Dependent measures.** The dependent measures for this study were: (1) salivary Cortisol level, (2) salivary alpha-amylase level, (3) missed balance steps, (4) heel-to-toe balance time, (5) FTNT score, (6) motion sickness symptoms, (7) fatigue level, and (8) cognitive performance.
Procedure

After signing an informed consent form (Appendix B) and completing a demographics form (Appendix C), participants took a Motion Sickness Susceptibility Questionnaire (MSSQ). Using the MSSQ, the researcher ensured that all participants are sensitive to motion at the 75% threshold based upon past data (Griffin & Howarth, 2000). Groups 1 and 2 were given the pre-exposure training, while for group 3 did not. The pre-training consisted of self-induced Coriolis training for 30 seconds at a time, five times over 2 hours each of 4 days. Participants were asked to raise their left arm straight up, grab their left ear with their right arm, and bend 90 degrees at their waist in order to put their proprioceptive senses in an abnormal position while spinning. Participants rotated themselves 10 times in 30 seconds, rotating at 20 revolutions per minute. The SSQ were administered following each day of training. Following the last day of training each week for Groups 1 and 2, or in the first session for Group 3, participants were exposed tovection induced motion sickness in the OKD. During OKD exposure, the SSQ was administered every 5 minutes by a recorded message, with the researcher marking the verbal results for the participant on paper.

Following OKD exposure, a series of tests occurred. This test battery began with the Simulator Sickness Questionnaire (SSQ) (Appendix D) and the SSS (Appendix E). The SSQ has been developed and proven effective at quantifying the symptoms of motion sickness (Kennedy et al., 1993). Tiredness of participants was measured through the Stanford Sleepiness Scale (SSS). Following the SSQ and SSS, a 5-minute cognitive test, known as a switching test, was given to assess cognitive effects of the OKD exposure. Instructions given to participants are located in Appendix F. Neuroendocrine measures identified stress and motion sickness through saliva samples, one taken for a baseline prior to OKD exposure, one immediately after each
cognitive test following OKD exposure. This test battery was administered immediately following OKD exposure and a second time 30 minutes following OKD exposure.

Handling of motion sickness symptoms in the laboratory. Throughout the study, the researcher kept air sickness bags, cleaning products including bleach, gloves, and light snacks readily available. No participants vomited during this study. For their safety, all participants were required to remain in the laboratory until they no longer show motion sickness symptoms on the SSQ, and no less than 30 minutes following the end of their OKD exposure.
Results

The present study was designed to identify the ability of the OKD to cause motion sickness in participants and to determine if the measures used were sensitive enough to capture differences between groups. Twenty-seven participants were selected to participate in this study, 16 female and 11 male. Only participants who exceeded the 75th percentile of the MSSQ were selected to participants. Participants were randomly divided into 3 groups; Group 1 (PT06) received the mild pre-exposure training and were exposed to the more severe motion sickness inducing event (the OKD) 6 hours after their last training session, Group 2 (PT24) received the mild pre-exposure training and were exposed to the OKD event 24 hours after their last training session, and Group 3 (NT) did not receive pre-exposure training and were only exposed to the OKD.

Effect of OKD Exposure

SSQ Total Score. A Friedman test was conducted to compare the effect of OKD exposure on SSQ Total (SSQT) score. A significant effect was found on SSQT score on the combined groups, \( \chi^2(3, 27) = 69.172, p < 0.001 \). A Wilcoxon Signed Ranks test was conducted to further investigate the effect of OKD exposure on SSQ Total score. The results of this test indicated that within all groups, higher SSQT scores were collected 10 minutes into OKD exposure than the baseline pre-test, \( Z = 4.542, p < 0.001 \). The results of this Wilcoxon Signed Ranks test also indicated that within all groups, higher SSQT scores were collected 20 minutes into OKD exposure than the baseline pre-test, \( Z = 4.543, p < 0.001 \). The results of this Wilcoxon Signed Ranks test also indicated that within all groups, higher SSQT scores were collected 30 minutes into OKD exposure than the baseline pre-test, \( Z = 4.543, p < 0.001 \).
Wilcoxon Signed Ranks test also indicates that within all groups, higher SSQT scores were collected 20 minutes into OKD exposure than 10 minutes into OKD exposure, \( Z = 3.930, p < 0.001 \). The results of this Wilcoxon Signed Ranks test also indicated that within all groups, higher SSQT scores were collected 30 minutes into OKD exposure than 10 minutes into OKD exposure, \( Z = 3.844, p < 0.001 \). The results of this Wilcoxon Signed Ranks test also indicated that within all groups, higher SSQT scores were collected 30 minutes into OKD exposure than 20 minutes into OKD exposure, \( Z = 3.229, p < 0.001 \). These results indicated that exposure to the OKD can induce motion sickness symptoms. They also indicated that the longer the time spent in the OKD, the more intense the OKD symptoms. This trend can be seen in figure 6.

Figure 6. SSQ Total Scores between groups, with SSQ Total score on the vertical axis and time relative to initial OKD exposure on the horizontal axis. The time = 0 point represents the pre-test. A difference exists between the NT group and both PT groups at the 35 and 65 minute marks. Asterisks indicate the presence of a significant difference, and are in the color corresponding to the group that is different with. Error bars indicate quartiles.
**Cortisol.** A repeated measures analysis of variance (ANOVA) was conducted to compare the effect of OKD exposure on salivary cortisol level. The results of the test indicated that there was a difference in cortisol levels due to OKD exposure, $F(2, 72) = 6.957, p < .01$. A Tukey HSD post hoc test was conducted between the different test intervals to further analyze the effect of OKD exposure on salivary cortisol levels. The results of the test indicated a higher cortisol levels during 30 minutes following exposure ($M = .433, SD = .38$) than the levels prior to exposure ($M = .1597, SD = .13$), and higher levels in 30 minutes following exposure than immediately following exposure ($M = .2739, SD = .22$), but showed no difference between cortisol levels sampled immediately following exposure and levels sampled prior to OKD exposure. These results indicated that salivary cortisol may take longer to indicate symptoms of motion sickness that other measures used in this study, but does identify the occurrence of motion sickness symptoms due to exposure to the OKD. The increasing trend can be seen in Figure 7.

**Stanford Sleepiness Scale (SSS).** A Friedman test was conducted to compare the effect of OKD exposure on SSS score. The results of the test indicated that a difference was found in SSS scores due to OKD exposure, $\chi^2(2, 27) = 29.370, p < .01$. Wilcoxon Signed Ranks tests were conducted to further analyze this difference. The results of the test indicate that SSS score were higher immediately following OKD exposure than those collected prior to OKD exposure, $Z(26) = -4.173, p < .01$, the SSS score were higher 30 minutes following OKD exposure than those prior to OKD exposure, $Z(26) = -2.784, p < .01$, and higher immediately following OKD exposure than 30 minutes following OKD exposure, $Z(26) = -3.753, p < .01$. This indicates that
Figure 7. Salivary Cortisol levels between groups, with cortisol level in ml/dl on the vertical axis and time relative to OKD exposure on the horizontal axis. The time = 0 point represents the pre-test. A difference exists between the 0 and 35 minute time intervals and the 0 and 65 minute time intervals, but not between the 35 and 65 minute time intervals.

motion sickness symptoms related to disorientation were identified by participants following OKD exposure and partial recovery occurred within 30 minutes, as can be seen on Figure 8.
**Figure 8.** SSS Scores between groups, with SSS Score on the vertical axis and time relative to OKD exposure on the horizontal axis. The time = 0 point represents the pre-test. A difference exists between the 0 and 35 minute time intervals, the 0 and 65 minute time intervals, and the 35 and 65 minute time intervals. Error bars are quartiles for each group.

**FTNT test score.** An ANOVA was conducted to compare the effect of OKD exposure on FTNT test score. The results of the test indicated a difference between FTNT test scores as a result of OKD exposure, $F(2, 72) = 3.527, p = .035$. A Tukey HSD post hoc test was conducted to further analyze the results. The results indicated higher FTNT test scores immediately following OKD exposure ($M = .6296, SD = .96$) than prior to OKD exposure ($M = .2222, SD = .42$). The result of the tests also showed no difference between FTNT scores immediately following OKD exposure and those 30 minutes following OKD exposure ($M = .3333, SD = .55$), or 30 minutes following OKD exposure and prior to OKD exposure. This indicates that the FTNT test may capture the onset of motion sickness symptoms accurately, though symptoms of motion sickness that cause a decrease in performance on the FTNT test may take longer than 30 minutes to recover from. This trend can be seen in Figure 9.
**Figure 9.** FTNT test score between groups with FTNT score on the vertical axis and time relative to OKD exposure on the horizontal axis. The time = 0 point represents the pre-test. A difference exists between the 0 and 35 minute time intervals, and the 0 and 65 minute time intervals, but not the 35 and 65 minute time intervals. Error bars are in standard deviation for each group.

**Alpha Amylase.** An ANOVA was conducted to compare the effect of OKD exposure on salivary amylase level. The results of the test showed no difference between salivary amylase levels exists, *F*(2, 72) = 2.861, *p* = .064. This indicates that salivary amylase did not indicate the occurrence of motion sickness symptoms during this study. It should be noted that participant amylase level varied greatly, which can be seen with the standard deviation bars in Figure 10.
Figure 10. Salivary amylase levels between groups, with amylase level in U/minute on the vertical axis and time relative to OKD exposure on the horizontal axis. The time = 0 point represents the pre-test. No differences between times were shown.

**Heel-to-Toe test time.** An ANOVA was conducted to compare the effect of OKD exposure on heel-to-toe balance test time. The results of this test showed no difference existed on heel-to-toe balance test time due to OKD exposure, $F (2, 72) = 1.465, p = .238$. The results indicate that heel-to-toe balance times were not likely affected by OKD exposure.
**Switching test accuracy.** An ANOVA was conducted to compare the effects of OKD exposure on ANAM Switching test accuracy. The results of the test showed no difference existed in Switching test accuracy due to OKD exposure, \( F(2, 72) = 1.559, p = .217 \). This indicates switching test accuracy was not impacted by the onset of motion sickness.

**Switching test time.** An ANOVA was conducted to compare the effect of OKD exposure on Switching test response time. The results of the test showed no difference between Switching test response time due to OKD exposure, \( F(2, 72) = 2.162, p = .122 \). These results indicate that the ANAM Switching test time did not identify the onset of motion sickness symptoms.

**Effect of Pre-Exposure Training**

The median scores for each of the SSQ tests are reported for the combined groups in Tables 2. This table also shows median SSQ scores are much higher for the immediately post OKD as compared to the scores 30 minutes following OKD. Medians for SSQ Nausea and Oculomotor score immediately following OKD exposure are not shown in the Table 2 as no significant difference was found.

**SSQ Total score.** A Kruskal-Wallis test was conducted to compare the effect of pre-exposure training on SSQ Total score during OKD exposure. The results of the test showed that there was no difference between groups during the pretest, \( \chi^2(2, 27) = 1.064, p = 0.59 \), no difference between groups 10 minutes into OKD exposure, \( \chi^2(2, 27) = 0.519, p = 0.77 \), no
Table 2. *Descriptive Statistics for identifying the effect of pre-exposure training on SSQ tests*

<table>
<thead>
<tr>
<th>Measure</th>
<th>N</th>
<th>Median Immediately Following OKD</th>
<th>Median 30 Minutes Following OKD</th>
</tr>
</thead>
<tbody>
<tr>
<td>SSQ Total Score</td>
<td>27</td>
<td>115.9</td>
<td>37.4</td>
</tr>
<tr>
<td>SSQ Nausea Score</td>
<td>27</td>
<td>28.6</td>
<td></td>
</tr>
<tr>
<td>SSQ Oculomotor Score</td>
<td>27</td>
<td>75.8</td>
<td></td>
</tr>
<tr>
<td>SSQ Disorientation</td>
<td>27</td>
<td>153.12</td>
<td>41.76</td>
</tr>
</tbody>
</table>

*Only significant results shown.

difference was shown between groups 20 minutes into OKD exposure, $\chi^2(2, 27) = 1.253, p = 0.53$, and no difference was shown between groups 30 minutes into OKD exposure, $\chi^2(2, 27) = 2.749, p = 0.253$. These results indicate that there was no difference in the onset of symptoms in the OKD regardless of the presence or absence of pre-exposure training.

A Kruskal-Wallis test was conducted to compare the effect of pre-exposure training on SSQ total score following exposure on all groups. A significant effect was found on SSQ total score taken from participants immediately after exposure, $\chi^2(3) = 6.409, p = 0.041$, and 30 minutes after exposure, $\chi^2(3) = 9.092, p = 0.011$. A Mann-Whitney test was conducted to test the hypothesis that pre-exposure training that concluded 6 hours prior to OKD exposure would decrease self-reported motion sickness symptoms immediately following OKD exposure as compared to participants who did not receive training. The results of the test indicated that, immediately following exposure, the 6 hours after training group had less self-rated motion sickness symptoms ($Mdn = 86.02$) than the no training group ($Mdn = 134.64$), $U = 11.5, p = 0.008$. A power of .73 was calculated for this comparison. A nonparametric effect size estimator indicated that a 14.2% chance exists that a participant randomly selected from the 6 hour after
training group would have higher motion sickness symptoms than a participant randomly selected from the no training group. A Mann-Whitney test was conducted to test the hypothesis that pre-exposure training that concluded 6 hours prior to OKD exposure would decrease self-reported motion sickness symptoms 30 minutes following OKD exposure as compared to participants who did not receive training. The results of the test indicated that 30 minutes following exposure, the 6 hours after training group had less self-rated motion sickness symptoms ($Mdn = 26.18$) than the no training group ($Mdn = 82.28$), $U = 11.0, p < 0.01$. A power of .77 was calculated for this comparison. A nonparametric effect size estimator indicated that a 13.6% chance exists that a participant randomly selected from the 6 hour after training group would have higher motion sickness symptoms than a participant randomly selected from the no training group.

A Mann-Whitney test was conducted to test the hypothesis that pre-exposure training that concluded 24 hours prior to OKD exposure would decrease self-reported motion sickness symptoms immediately following OKD exposure as compared to participants who did not receive training. The results of the test indicated that the 24 hour after training group did not have less self-rated motion sickness symptoms ($Mdn = 104.72$) than the no training group ($Mdn = 134.64$) immediately after training, $U = 22.50, p = 0.113$. A power of .45 was calculated for this comparison. A nonparametric effect size estimator indicated that a 27.8% chance exists that a participant randomly selected from the 24 hour after training group would have higher motion sickness symptoms than a participant randomly selected from the no training group. A Mann-Whitney test was conducted to test the hypothesis that pre-exposure training that concluded 24 hours prior to OKD exposure would decrease self-reported motion sickness symptoms 30 minutes following OKD exposure as compared to participants who did not receive training. The
results of the indicated that the 24 hour after training group had less self-rated motion sickness
symptoms ($Mdn = 26.18$) than the no training group ($Mdn = 82.28$) 30 minutes following
exposure, $U = 11.5, p = 0.008$. A power of .35 was calculated for this comparison. A
nonparametric effect size estimator indicated that a 14.2% chance exists that a participant
randomly selected from the 24 hour after training group would have higher motion sickness
symptoms than a participant randomly selected from the no training group.

Comparing the 6 hour after training group with the 24 hour after training group, a Mann-
Whitney test was conducted to test the hypothesis that pre-exposure training that concluded 6
hours prior to OKD exposure would decrease self-reported motion sickness symptoms
immediately following OKD exposure as compared to participants who concluded pre-exposure
training 24 hours prior to OKD exposure. The results of the test indicated that self-rated motion
sickness symptoms for the 6 hour after training group ($Mdn = 86.02$) were no different than the
self-rated symptoms of the 24 hour after training group ($Mdn = 104.72$) immediately after
exposure, $U = 33.5, p = 0.546$. A nonparametric effect size estimator indicated that a 41.4%
chance exists that a participant randomly selected from the 6 hour after training group would
have higher motion sickness symptoms than a participant randomly selected from the 24 hour
after training group. A Mann-Whitney test was conducted to test the hypothesis that pre-
exposure training that concluded 6 hours prior to OKD exposure would decrease self-reported
motion sickness symptoms 30 minutes following OKD exposure as compared to participants
who concluded pre-exposure training 24 hours prior to OKD exposure. The results of the test
indicated that self-rated motion sickness symptoms for the 6 hour after training group ($Mdn =
26.18$) were no different than the self-rated symptoms of the 24 hour after training group ($Mdn =
26.18$) 30 minutes after exposure, $U = 38.5, p = 0.863$. A nonparametric effect size estimator
indicated that a 47.5\% chance exists that a participant randomly selected from the 6 hour after training group would have higher motion sickness symptoms than a participant randomly selected from the 24 hour after training group.

These results indicated that pre-exposure training can decrease the time required to recover from symptoms of motion sickness, and that exposure 6 hours prior to the more extreme motion sickness inducing event may decrease the peak symptoms of motion sickness. The results indicated self-rated symptom recovery benefitted from pre-exposure training regardless of the time between the pre-exposure training and the more extreme motion sickness environment. This trend can be seen above in Figure 6.

**SSQ Nausea score.** A Kruskal-Wallis was conducted to compare the effect of pre-exposure training on SSQ Nausea score following OKD exposure. The results of the test revealed difference in SSQ Nausea scores immediately after exposure, $\chi^2(3) = 4.467, p = 0.107$.

A Kruskal-Wallis test was conducted to compare the effect of pre-exposure training on SSQ Nausea score. The results of the test indicated a difference in SSQ Nausea score 30 minutes after exposure, $(\chi^2(3) = 9.835, p < 0.01)$. A Mann-Whitney test was conducted to further analyze the difference indicated by the previous test. The results of this test indicated that participants in the 6 hour after training group had less self-rated motion sickness symptoms related to nausea ($Mdn = 9.54$) and the no training group ($Mdn = 57.24$) 30 minutes following exposure $U = 7.0, p < 0.01$. A nonparametric effect size estimator indicated that a 8.6\% chance exists that a participant randomly selected from the 6 hour after training group would have higher nausea-related motion sickness symptoms than a participant randomly selected from the no training group.
A Mann-Whitney test was conducted to further analyze the difference found in the Kruskal-Wallis test. The results of this test indicated that participants in the 24 hour after training group suffered from less self-rated, nausea-related symptoms of motion sickness ($Mdn = 19.08$) than participants in the no training group ($Mdn = 57.24$) 30 minutes following exposure, $U = 15.0$, $p = 0.024$. A nonparametric effect size estimator indicated that a 18.5% chance exists that a participant randomly selected from the 24 hour after training group would have higher nausea-related motion sickness symptoms than a participant randomly selected from the no training group.

A Mann-Whitney test was conducted to further analyze the difference found in the Kruskal-Wallis test. The results of this test showed no difference in self-rated, nausea-related symptoms of motion sickness between participants in the 6 hour after training group ($Mdn = 9.54$) and 24 hour after training group ($Mdn = 19.08$) 30 minutes after exposure, $U = 33.0$, $p = 0.546$. A nonparametric effect size estimator indicated that a 40.7% chance exists that a participant randomly selected from the 6 hour after training group would have higher nausea-related motion sickness symptoms than a participant randomly selected from the 24 hours after training group.

These results indicates that pre-exposure training may not have an impact on the peak nausea symptoms of motion sickness, but exposure to a mild motion sickness inducing event 6 hours or 24 hours prior to the more extreme motion sickness inducing event may decrease the time required to recover from nausea concerns associated with motion sickness symptoms. The length of time between training and exposure would not appear to impact recovery from nausea symptoms of motion sickness. This trend can be seen in Figure 11.
Figure 11. SSQ Nausea Score between Groups with SSQ Nausea score on the vertical axis and time relative to initial OKD exposure on the horizontal axis. The time = 0 point represents the pre-test. A difference exists between the NT group and both PT groups at 65 minute mark. Asterisks indicate the presence of a significant difference, and are in the color corresponding to the group that is different with. Error bars are quartiles for each group.

SSQ Oculomotor Score. A Kruskal-Wallis was conducted to compare the effect of pre-exposure training on SSQ Oculomotor score following OKD exposure. The results of the test revealed no significant effect of pre-exposure training on SSQ Oculomotor score immediately after exposure ($\chi^2(3) = 3.828, p = 0.147$).

A Kruskal-Wallis was conducted to compare the effect of pre-exposure training on SSQ Oculomotor score following OKD exposure. The results of the test indicated a significant effect of pre-exposure training on SSQ Oculomotor score 30 minutes after exposure ($\chi^2(3) = 8.856, p = 0.012$).

A Mann-Whitney test was conducted to further analyze the difference found in the previous Kruskal-Wallis test. The results of the test indicated that self-rated oculomotor
symptoms of motion sickness were lower in participants within the 6 hour after training group ($Mdn = 22.74$) than participants in the no training group ($Mdn = 15.16$) 30 minutes following exposure, $U = 12.0, p = 0.011$. A nonparametric effect size estimator indicated that a 14.8% chance exists that a participant randomly selected from the 6 hour after training group would have higher oculomotor motion sickness symptoms than a participant randomly selected from the no training group.

A Mann-Whitney test was conducted to further analyze the difference found in the previous Kruskal-Wallis test. The results of the test indicated that self-rated oculomotor symptoms of motion sickness were lower in participants within the 24 hour after training group ($Mdn = 22.74$) than participants in the no training group ($Mdn = 60.64$) 30 minutes following exposure, $U = 12.5, p = 0.011$. A nonparametric effect size estimator indicated that a 15.4% chance exists that a participant randomly selected from the 24 hour after training group would have higher oculomotor motion sickness symptoms than a participant randomly selected from the no training group.

A Mann-Whitney test was conducted to further analyze the difference found in the previous Kruskal-Wallis test. The results of the test indicated that self-rated oculomotor symptoms of motion sickness were no different between participants in the 6 hour after training group ($Mdn = 22.74$) and participants in the 24 hour after training group ($Mdn = 15.16$) 30 minutes after exposure, $U = 30.5, p = 0.387$. A nonparametric effect size estimator indicated that a 37.7% chance exists that a participant randomly selected from the 6 hour after training group would have higher oculomotor motion sickness symptoms than a participant randomly selected from the 24 hour after training group.
This results indicates that pre-exposure training may not have an impact on the peak oculomotor symptoms of motion sickness, but exposure to a mild motion sickness inducing event 6 hours or 24 hours prior to the more extreme motion sickness inducing event may decrease the time required to recover from oculomotor concerns associated with motion sickness symptoms. The length of time between training and exposure would not appear to impact recovery from oculomotor symptoms of motion sickness. This trend can be seen in Figure 12.

**Figure 12.** SSQ Oculomotor Score between Groups with SSQ Oculomotor score on the vertical axis and time relative to the beginning of OKD exposure on the horizontal axis. The time = 0 point represents the pre-test. A difference exists between the NT group and both PT groups at 65 minute mark. Asterisks indicate the presence of a significant difference, and are in the color corresponding to the group that is different with. Error bars are quartiles for each group.

**SSQ Disorientation Score.** A Kruskal-Wallis test was conducted to compare the effect of pre-exposure training on SSQ Disorientation score following OKD exposure. The results of
the test revealed a significant effect of pre-exposure training on SSQ Disorientation score immediately after exposure, $\chi^2(3) = 7.790, p = 0.02$, and 30 minutes after exposure ($\chi^2(3) = 6.510, p = 0.039$). A Mann-Whitney test was conducted to further analyze the difference found in the previous Kruskal-Wallis test. The results of the test indicated that self-rated disorientation symptoms of motion sickness were lower in participants within the 6 hour after training group ($Mdn = 97.44$) than participants in the no training group ($Mdn = 194.88$) immediately following exposure, $U = 9.0, p < 0.01$. A nonparametric effect size estimator indicated that a 11.1% chance exists that a participant randomly selected from the 6 hour after training group would have higher disorientation motion sickness symptoms than a participant randomly selected from the no training group.

A Mann-Whitney test was conducted to further analyze the difference found in the previous Kruskal-Wallis test. The results of the test indicated that self-rated disorientation symptoms of motion sickness were lower in participants within the 6 hour after training group ($Mdn = 27.84$) than participants in the no training group ($Mdn = 83.52$) 30 minutes following exposure, $U = 13.50, p = .014$. A nonparametric effect size estimator indicated that a 16.7% chance exists that a participant randomly selected from the 6 hour after training group would have higher disorientation motion sickness symptoms than a participant randomly selected from the no training group.

A Mann-Whitney test was conducted to further analyze the difference found in the previous Kruskal-Wallis test. The results of the test indicated that self-rated disorientation symptoms of motion sickness were lower in participants within the 24 hour after training group ($Mdn = 27.84$) than participants in the no training group ($Mdn = 83.52$) 30 minutes following exposure, $U = 18.5, p = 0.05$. A nonparametric effect size estimator indicated that a 22.8%
A Mann-Whitney test was conducted to further analyze the difference found in the previous Kruskal-Wallis test. The results of the test indicated that self-rated disorientation symptoms of motion sickness were not lower in participants within the 24 hour after training group ($Mdn = 111.36$) than participants in the no training group ($Mdn = 194.88$) immediately following exposure, $U = 20.0, p = 0.077$. A nonparametric effect size estimator indicated that a 24.7% chance exists that a participant randomly selected from the 24 hour after training group would have higher disorientation motion sickness symptoms than a participant randomly selected from the no training group.

A Mann-Whitney test was conducted to further analyze the difference found in the previous Kruskal-Wallis test. The results of the test indicated that self-rated disorientation symptoms of motion sickness were no different between participants in the 6 hour after training group ($Mdn = 97.44$) and 24 hour after training group ($Mdn = 111.36$) immediately after exposure, $U = 33.0, p = .546$. A nonparametric effect size estimator indicated that a 40.7% chance exists that a participant randomly selected from the 6 hour after training group would have higher disorientation motion sickness symptoms than a participant randomly selected from the 24 hour after training group.

A Mann-Whitney test was conducted to further analyze the difference found in the previous Kruskal-Wallis test. The results of the test indicated that self-rated disorientation symptoms of motion sickness were no different between participants in the 6 hour after training group ($Mdn = 27.84$) and the 24 hour after training group ($Mdn = 27.84$) 30 minutes after
exposure, \( U = 39.5, p = 0.931 \). A nonparametric effect size estimator indicated that a 48.8% chance exists that a participant randomly selected from the 6 hour after training group would have higher disorientation motion sickness symptoms than a participant randomly selected from the 24 hour after training group.

The results indicate that pre-exposure training can decrease the peak disorientation symptoms of motion sickness, and exposure to a mild motion sickness inducing event 6 hours to the more extreme motion sickness inducing event may decrease the time required to recover from disorientation associated with motion sickness symptoms. The length of time between training and exposure would not appear to impact peak disorientation symptoms of motion sickness. This trend can be seen in Figure 13.

![SSQ Disorientation](image)

*Figure 13.* SSQ Disorientation Score between Groups with SSQ Disorientation score on the vertical axis and time relative to the beginning of OKD exposure on the horizontal axis. The time = 0 point represents the pre-test. A difference exists between the NT group and both PT groups at the 35 minute and 65 minute marks. Asterisks indicate the presence of a significant difference,
and are in the color corresponding to the group that is different with. Error bars are quartiles for each group.

**Cortisol level.** A One-way analysis of the variance was conducted on the change in cortisol level, significance was not found in the first post-test ($F = .738, p = .488$) or the second post-test ($F = .815, p = .605$). This indicated that the cortisol level is not sensitive enough to identify differences between training and non-training groups.

**Amylase level.** A One-way analysis of the variance was conducted on the change in amylase level, significance was not found in the first post-test ($F = 3.271, p = .055$) or the second post-test ($F = .144, p = .867$). This indicated that the amylase level is not sensitive enough to identify differences between training and non-training groups.

**SSS Score.** A Kruskal-Wallis test revealed no significant effect of training on SSS score immediately following exposure ($\chi^2(3) = 2.197, p = 0.333$) or 30 minutes following exposure ($\chi^2(3) = 2.062, p = 0.357$). This indicated that the SSS is not sensitive enough to identify differences between training and non-training groups.

**ANAM Switching test response time.** A One-way analysis of the variance was conducted on ANAM Switching test response time, significance was not found in the first post-test ($F = .110, p = .896$) or the second post-test ($F = 1.289, p=.294$). This indicates that the ANAM Switching test response time is not sensitive enough to identify differences between training and non-training groups.
**ANAM Switching test score.** A One-way analysis of the variance was conducted on ANAM Switching test score, significance was not found in either the first post-test ($F = 1.018, p = .376$) or the second post-test ($F = .605, p = .554$). This indicated that the ANAM Switching test score is not sensitive enough to identify differences between training and non-training groups.

**FTNT score.** A One-way analysis of the variance was conducted on FTNT score, significance was not found either in the first post-test ($F = 2.294, p = .123$) or the second post-test ($F = .410, p = .668$). This indicated that the FTNT score is not sensitive enough to identify differences between training and non-training groups.

**Heel-to-toe balance time.** A One-way analysis of the variance was conducted on heel-to-toe balance test times, significance was not found in the first post-test ($F = 3.039, p = .067$) or the second post-test ($F = .787, p = .467$). This indicated that the heel-to-toe balance time is not sensitive enough to identify differences between training and non-training groups.
Discussion and Conclusions

The findings indicate 4 of the 8 measures used in this study were able to capture the occurrence of motion sickness symptoms; SSQ scores, SSS scores, cortisol levels, and the FTNT. The amylase levels, ANAM test accuracy and response time, and heel-to-toe balance test each did not capture the onset of motion sickness. Future studies should have participants perform the heel-to-toe and FTNT tests a number of times prior to experimental exposure in order to achieve a standard baseline for the study. The results also confirm the ability of the sandpaper pattern used in this OKD as being able to induce motion sickness symptoms, as predicted by Rodriguez-Jimenez (2012).

In addition, the SSQ identified differences at each time step during OKD exposure. Specifically, a graded response was noted, where symptoms after 10 minutes of OKD exposure were greater than those immediately prior to OKD exposure, symptoms after 20 minutes of OKD exposure were greater than those after 10 minutes of OKD exposure, and symptoms 30 minutes after OKD exposure were greater than those after 20 minutes of OKD exposure. Additionally, each time step was different from each other time step. This indicates that researchers can have a great control over motion sickness symptoms using the OKD, where motion sickness symptoms increase as time exposed to the OKD increased.

The SSQ total and disorientation scores identified differences between the training groups and non-training group both immediately following OKD exposure and 30 minutes following OKD exposure. This indicates that the self-induced motion sickness training was likely successful in diminishing some motion sickness symptoms typically experienced in the OKD, and concurs with the results reported in both Smither et al. (2008) and Mouloua et al. (2005). SSQ Oculomotor and Nausea scores also indicate a decrease in symptoms due to
exposure to a milder motion sickness stimulus prior to a more extreme motion sickness stimulus during recovery 30 minutes following the exposure to the more extreme motion sickness inducing event. It is important to note that there is very little difference between SSQ scores taken while participants were experiencing the motion sickness inducing environment of the OKD. This indicates that pre-exposure training does not decrease the onset of motion sickness symptoms associated with the OKD, but rather the symptom recovery following OKD exposure.

Nonparametric effect size estimates indicated low $\hat{p}_{ab}$ values for each of the statistically different measures. This indicates that the effect size for these measures are high, corresponding to between 51% and 91% of the difference identified between the groups was due to the pre-exposure training. Effect size was largest between 6 hour after training group and the no training group on almost every significant measure. This indicates that 6 hours after training may be closer than 24 hours after training to the optimum length of time between the completion of pre-exposure training and experiencing a more severe motion sickness inducing event for the greatest reduction in motion sickness symptoms. A potential topic for future research includes further evaluating the length of time between the last pre-exposure training session and the more severe exposure to a motion sickness inducing environment. This study only evaluated the difference between 6 hours and 24 hours. Other times, such as 3 hours, 12 hours or 48 hours should be explored to further capture the impact training has on motion sickness symptoms and determine an optimal wait time between pre-exposure training and exposure to a more severe motion sickness inducing event.

Additionally, research should be conducted to ascertain if the reduced motion sickness symptoms due to pre-exposure training last for more than one session. If motion sickness symptoms are reduced for multiple days following exposure, it is possible that the symptoms
could be reduced until maximum adaptation occurs. If future results indicate the effects of pre-exposure training lasting until adaptation occurs, than cruise line, commercial space tourism operators, and other operations sensitive to motion sickness can recommend similar pre-exposure training for motion sickness sensitive passengers prior to cruise departure, spacecraft launch, or motion sickness event dates.

The power estimator conducted on the SSQ Total scores indicates that the Mann-Whitney tests between the 6 hour after training group and the no training group had the highest power. To achieve similar power between the 24 hour, between 11 and 19 more participants per group would have been needed, which was beyond the scope of this study. The statistical power was expected to be lower than optimal for the nonparametric measures, as nonparametric analyses are known have less power (Mumby, 2002). Power estimates were only conducted on the SSQ Total score due to a high correlation between SSQ Total scores and SSQ scores for each of the Nausea, Oculomotor, and Disorientation categories. Future studies should consider increasing the number of participants to increase the power of self-reporting measures to levels comparable to calculations that can be analyzed parametrically.

Measures other than the SSQ did not indicate differences between groups, such as SSS score, cortisol levels, alpha amylase levels, cognitive performance, and the balance tests. It was deemed that based upon the relatively high standard deviations associated with these measurements, these measurements were not sensitive enough to accurately depict the changes expected as a result of pre-exposure training. Future studies using the ANAM Switching test should consider setting the initial testing benchmark at a higher value than the 90% benchmark that was used in this study; a score over 95% or even high would possibly lead to a greater impact from motion and reduce the variability seen in this study. A concern for doing this,
though is while this may increase the variability between participants, it may create a ceiling effect, where participants are unable to get results due to a lack of variability on the higher end. Additionally, future studies conducting biochemical assays should also consider measuring melatonin level, as melatonin may be useful as a non-subjective measure of fatigue and sopite.

A possible explanation considered by the researcher for the lack of discriminating results from the cortisol and amylase levels relates to the motion sickness symptom curves indicated by these measures. Chouker et al. (2010) has shown that salivary biochemical indicators have a delayed appearance in test samples; in their study, salivary cortisol levels increased to peak during post-test collection, rather than immediately following the final exposure to their motion sickness inducing environment. As the curves associated with salivary cortisol and amylase only rise in this study, it is possible the biomarkers had just or never reached their peak, and the differences between groups would not have been displayed until further beyond the times which samples were collected as part of this study. These can be seen in Figures 7 and 8. It is possible that a difference between groups could have been noted if samples from participants were also collected 60 minutes after OKD exposure. Future research using saliva samples with motion sickness should consider collecting samples beyond 30 minutes following exposure to better map the cortisol and amylase curves associated with motion sickness as further testing was beyond the scope of this study.

No results indicated a difference between the 6 hour and the 24 hour pre-training groups. This indicates that the positive impacts of training are present regardless of whether exposure occurs 6 or 24 hours following the pre-exposure training.

When selecting measures for future studies measuring motion sickness symptoms, consideration should be made to ensure measures maintain a balance between sensitivity, the
ability to diagnose motion sickness, cost, effort, and the training requirement on both researchers and participants. This can change between testing environments and may not be the same as they were in this study. For this study, it was found that the SSQ was the most balanced for this testing environment.

One consideration that was not taken into account was the nature of the OKD to cause claustrophobia, which could present similar symptoms to those expected from motion sickness. The OKD is approximately 6 feet in diameter and participants were seated in the OKD for 30 minutes during this experiment. Future research in the OKD should provide an indication that claustrophobia was not impacting the results by having an additional control group who sit in the OKD without it spinning for the length of the experiment.

The results of this study indicate the presence of adaptation to motion sickness. The presence of adaptation indicates support for the Neural Mismatch theory of motion sickness. One point against that theory is that the adaptation was only observed as a decrease in subjective symptoms during the recovery from OKD exposure. Perhaps with longer or different training, this effect could be seen earlier in the recovery or even during the exposure. The results of this study confirm the results of Smither et al. (2008) indicating that pre-exposure training does have an impact on subjective motion sickness symptoms following OKD exposure. Additionally, it is possibly that the lack of a difference shown by the salivary amylase levels in this study indicates that norepinephrine is not a strong indicator of stress as it relates to motion sickness. Rather, it is possible that the stress identified by prior studies using salivary amylase could stem from the stress of the environment meant to induce motion sickness rather than the stress associated with the sickness itself.
While research into pre-exposure training is not complete, the results in this study may lead to practical application of pre-exposure training in the future. Pre-exposure training could increase safety during transportation in both public and private industries. If pre-exposure training is found to be effective in mitigating symptoms in the real world, the training could be initiated for military personnel prior to participating in helicopter or fixed-wing air transport operations to ensure that passengers being dropped off are still combat ready. Additionally, in the private sector, airline passengers that get air sick could possibly benefit from pre-exposure training to recover from symptoms following their flight. If the pre-exposure training performed in this study is found to be applicable to other forms of motion sickness, motion sickness symptom recovery could be increased in real world situations. Theme park operators could recommend a simple pre-exposure training the week prior to attendance so that visitors who are typically very sensitive to motion sickness could recover faster and enjoy more of the park. Since this study found no decrease in the onset or maximum symptoms, this pre-exposure training does not appear to be suitable for reducing motion sickness experienced during significantly longer motion sickness inducing environments, such as passengers on a long cruise. Future studies should be conducted to identify whether this holds true or not.

**Further Research**

There are many areas related to motion sickness which could benefit from further research that were beyond the scope of this study. Further research should also consider keeping and evaluating participants for a full hour or more after exposure to a more severe motion sickness inducing event. This could identify further impacts pre-exposure training has on the recovery from motion sickness symptoms and could provide better insight into the biochemical
measures associated with motion sickness. Another factor that was beyond the scope of the study was evaluating the impact of the pre-exposure training on other motion sickness inducing environments. A fourth area requiring further research is finding the optimum training length for the most efficient reduction of motion sickness symptoms. This could be accomplished by varying the number of days of training prior to exposure to a motion sickness inducing environment or varying the length of time of the training sessions.

Another possible area of further study could be with the method of conducting the pre-training. The method used in this study was the method used by Mouloua et al. (2005), but it may not be the only or the best method of performing pre-exposure training with participants susceptible to motion sickness symptoms. Further research should be conducted to explore these possibilities.

Another area of research that should be considered is comparing the effectiveness of pre-exposure training against the effectiveness of pharmacological countermeasures such as Dramamine, Chlorpheniramine, Scopolamine (Hyocine), Promethazine, Ondansetron and Ramosetron. These countermeasures have had mixed reports of success and can have serious drawbacks, but testing these pharmacological countermeasures against behavioral countermeasures such as pre-exposure training could reveal that pharmacological countermeasures are more effective or less effective that behavioral countermeasures. Side by side testing may also provide an indication that motion sickness symptoms may be causing the side effects experienced by taking pharmacological countermeasures to motion sickness. An example could be that the fatigue associated with these countermeasures could be the manifestation of sopite syndrome caused by the motion sickness inducing event rather than a side effect of the countermeasures. Another potential outcome of such side-by-side research could be
findings that indicate combining pharmacological countermeasures with behavior countermeasures further decreasing the impact of motion sickness symptoms.

An additional method to test the impacts of pre-exposure training on motion sickness symptoms is to expose motion sickness sensitive participants to a non-laboratory motion sickness inducing environment after the pre-exposure training has been conducted. This could be accomplished through the use of a small boat being sent out on rough seas following four days of the mild pre-exposure training used in this study. This experiment would confirm that the pre-exposure training is effective for mitigating motion sickness symptoms when participants are exposed to motion sickness inducing environments beyond just the OKD.

Unfortunately, predicting rough seas the five days needed for training prior to sending participants out on a boat can be difficult, and cause participants to be trained without a sufficient motion sickness environment to test in. An alternative that could resolve this issue is the use of parabolic flight rather than a boat on rough seas. Parabolic flight in an aircraft, though more expensive, can be scheduled for most days and would be especially handy in areas of the country where storms are rare. Using parabolic flight at the motion sickness inducing environment would provide a better analog to the environment experienced as part of SAS than any environment simulated on the ground. The results such an experiment could provide considerable data on the usefulness of pre-exposure training on reducing symptoms associated with SAS and could show OKD exposure as a sufficient analog to other motion sickness inducing environments.
Summary

In summary, this study found pre-exposure training to be effective in reducing motion sickness symptoms induced by an OKD. This study also showed no difference between completing the pre-exposure training 6 hours or 24 hours prior to exposure. Further, it has been concluded that the SSQ is the most sensitive measure for identifying changes between groups exposed to the same motion sickness inducing environment. The results of this study could provide a valuable tool for mitigating motion sickness symptoms in environments such as microgravity, simulators, boats, and airplanes, where motion sickness symptoms could negatively impact performance and safety.
References


Kennedy, R.S., Fowlkes, J.E., & Lilienthal, M.G. (1993). Postural and performance changes following exposures to flight simulators. *Aviation, Space, and Environmental Medicine, 64*, 912-920. [Kennedy et al., 1993a]


Rodriguez-Jimenez, W. (2012). The effects of different optokinetic drum rotation speeds on motion sickness, cognitive performance, and sleep amount. (Master’s Thesis, Embry-Riddle Aeronautical University, Daytona Beach, Fl.)


Appendix A

Motion Sickness Susceptibility Questionnaire

MOTION SICKNESS SUSCEPTIBILITY QUESTIONNAIRE

INSTRUCTIONS

This questionnaire is primarily concerned with: (1) your susceptibility to motion sickness, and (2) what types of motion are the most effective in causing this sickness.

Please read the questions carefully and answer them ALL by FILLING IN with an “X” in the boxes which most closely correspond to you as an individual.

All the information you give is CONFIDENTIAL and will be used for research purposes only.

Thank you very much for your cooperation.

NAME  APPROXIMATE WEIGHT  HEIGHT

EMAIL ADDRESS  TELEPHONE NUMBER

GENDER  AGE

1. In the past YEAR, how many times have you TRAVELED AS A PASSENGER in the following types of transport?

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2. In the past **YEAR**, how many times have you **FELT ILL**, whilst traveling as a **PASSENGER** in the following types of transport?

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3. In the past **YEAR**, how many times have you **VOMITED**, whilst traveling as a **PASSENGER** in the following types of transport?

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4. Do you **EVER** feel **HOT** or **SWEAT** whilst traveling as a **PASSENGER** in the following types of transport?

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5. Do you **EVER** suffer from HEADACHES whilst traveling **AS A PASSENGER** in the following types of transport?

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6. Do you **EVER** suffer from LOSS/CHANGE OF SKIN COLOR (go pale) whilst traveling **AS A PASSENGER** in the following types of transport?

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7. Do you **EVER** suffer from MOUTH WATERING whilst traveling **AS A PASSENGER** in the following types of transport?

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8. Do you **EVER** feel DROWSY whilst traveling AS A PASSENGER in the following types of transport?

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9. Do you **EVER** feel DIZZY whilst traveling AS A PASSENGER in the following types of transport?

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10. Do you **EVER** suffer from NAUSEA (stomach discomfort, feeling sick) whilst traveling AS A PASSENGER in the following types of transport?

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11. Have you **EVER VOMITED** whilst traveling **AS A PASSENGER** in the following types of transport?

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12. Would you **AVOID** any of the following types of transport because of motion sickness?

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<tr>
<td>TRAINS</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

13. Which of the following best describes you **SUSCEPTIBILITY** to motion sickness?

<table>
<thead>
<tr>
<th>SUSCEPTIBILITY</th>
</tr>
</thead>
<tbody>
<tr>
<td>MUCH LESS THAN AVERAGE</td>
</tr>
<tr>
<td>LESS THAN AVERAGE</td>
</tr>
<tr>
<td>AVERAGE</td>
</tr>
<tr>
<td>MORE THAN AVERAGE</td>
</tr>
<tr>
<td>MUCH MORE THAN AVERAGE</td>
</tr>
</tbody>
</table>

14. Have you ever suffered from any serious illness or injury

<table>
<thead>
<tr>
<th>YES</th>
<th>NO</th>
</tr>
</thead>
</table>
15. Are you under medical treatment or suffering a disability affecting daily life?

<table>
<thead>
<tr>
<th>YES</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>NO</td>
<td></td>
</tr>
</tbody>
</table>

Thank you for your time!

(Adapted from Griffin & Howarth, 2000)
Appendix B

Demographic Form

(Begins on next page)
Demographic Form

Please fill out all answers if applicable. All answers will be kept anonymous.

ID # _______     Gender _______     Age _______     Weight_______

Do you wear glasses? (Please circle)     Yes    No
If yes:     All the time    Sometimes    Just for Distance    Just for Reading

Medications & Medical Conditions

______________________________________________________________________________
______________________________________________________________________________
______________________________________________________________________________

Are you currently taking any anti-depressant medications? If so, which?

______________________________________________________________________________
______________________________________________________________________________
______________________________________________________________________________

Do you have a history of inner ear disorders or regularly have ear infections? If yes, explain.

______________________________________________________________________________
______________________________________________________________________________
______________________________________________________________________________

Ethnicity (Choose one)

___ American Indian or Native Alaskan
___ Asian or Asian American
___ Black or African American
___ Hawaiian or Other Pacific Islander
___ Hispanic or Latino
___ Non-Hispanic White
___ Other (specify): ___________

Comments:

______________________________________________________________________________
______________________________________________________________________________
Appendix C

Informed Consent Form

(Begins on next page)
Embry-Riddle Aeronautical University

Nicholas Stapleton, B.S., Principal Investigator
Jonathan French, Ph. D., Advisor

Thesis Research: “Pre-exposure training as a means to reduce vection induced symptoms of motion sickness”

This Informed Consent Form has two parts:
- Information sheet (to share information about the study with you)
- Certificate of Consent (for signatures if you choose to participate)

You will be given a copy of the full Informed Consent Form once signed

Part I: Information Sheet

Introduction
The study I am conducting as part of my Master’s thesis is the evaluation of motion sickness symptoms induced by the Coriolis illusion (which will be demonstrated for you) and the optokinetic (OK) drum. The Coriolis illusion will involve several repetitions over 4 days prior to the OK drum exposure, involving about 2 hours each day. The experiment will last 5 weeks, only 3 of which you will be participating in. Two weeks will be considered off weeks to allow you to fully recover from any training effects prior to the start of the next experimental week. The OK drum is a device that is designed to induce symptoms of motion sickness by having the participant stare at a pattern rotating around their head. For the OK drum, you will be seated through the 30 minutes of exposure.

You must first read, understand and sign this consent document before proceeding. If you agree to participate, you will first complete a Motion Sickness Susceptibility Questionnaire (MSSQ) to determine if you are eligible for this study.

Purpose of the Research
The induction of perceived motion through the use of visual stimuli is known as vection; this is the type of experience you will have in the Optokinetic drum. The corilis illusion uses the vestibular (balance) sense. We are interested in determining the degree of symptoms produced by each.

Type of Research Intervention
This research will require you to answer a series of questionnaires, providing saliva samples periodically (for hormone changes), and taking a cognitive test (to determine if cognitive ability is impaired by exposure). The saliva samples will be processed to determine the content of the carbohydrate enzyme amylase and the hormones melatonin and cortisol. These samples are only natural biochemical agents in your body. Your DNA will not be examined during the processing of your saliva samples. No other agents or markers will be examined from your samples. These data from your saliva samples will hopefully lead to a more useful and rational means to develop motion sickness drugs as countermeasures.
**Participant Selection**
You must complete an MSSQ (Motion Sickness Susceptibility Questionnaire) in order for us to determine how susceptible you are for motion stimuli. In other words, this test will determine whether you will get motion sickness symptoms. We only have a limited number of participants due to the number of chemical assays and their expense.

**Voluntary Participation**
Participation in this experiment is completely voluntary. You will be paid $200 for your participation in this study. At any time, you may decide you do not wish to participate in this research. You will be paid whatever amount you have earned up to that point. However we encourage you to remain and learn something about your susceptibility to motion symptoms.

**Duration & Procedures**
This experiment requires your participation for one week. Depending on the group you are participating in, this means either 2 hours or 10 hours for that week. The procedures require you to answer some symptom questionnaires, take cognitive tests and provide saliva samples. The OK drum is a fixed structure. Entering and exiting the drum will require you to climb under the drum on your hands and knees. The researcher will assist you if you request or require it.

**Risks**
As the experiment being conducted is testing for motion sickness, it is likely that you will experience motion sickness symptoms while participating in this test. These should not be severe and we will do all we can to keep them minimal. General symptoms may include nausea, eyestrain, blurred vision, headache, vertigo, and stomach concerns depending on your susceptibility. During every stage of the experiment, you will be closely monitored by a researcher to ensure your comfort. While in the OK drum, you will be observed by camera. This camera is not recording, only providing your current status to the researcher controlling the OK drum. We expect these symptoms to be mild in most cases similar to those experienced on a rotating carnival ride. Our experience has shown that the effects are quite short, and typically pass within an hour of onset. You will not be able to leave the facility until your symptoms have subsided as determined by the questionnaire and the researcher.

Additionally, the OK drum is a small space, roughly 6 feet in diameter. It is possible for participants sensitive to small spaces to feel claustrophobic. If the feeling of claustrophobia is beyond comfortable levels, report it to the researcher, who will stop the drum if it is rotating and assist you in leaving.

**Benefits**
You will be helping us to discover the effects of motion sickness behavioral countermeasures and to understand the neurohormonal consequences of motion sickness for the development of pharmaceutical countermeasures. In other words, the data you provide through your saliva samples helps us understand how a person reacts to motion sickness on a biological level.
Reimbursement
Your participation in this study will amount to approximately $10 per hour of participation.

Confidentiality
Your results will be kept by the PI in a locked file cabinet and your name will not be associated with your results. We will use a coding system to keep the results separate from your name.

Saliva Protocol
Your saliva sample will be kept anonymous for the duration of its existence. Samples will be sent to Salimetrics Inc., a reputable saliva processing facility. Samples will be stored in a secure biological storage refrigerator prior to shipping to Salimetrics, Inc. Your names will not be sent with your samples. Only the researcher will have information to match your samples to the remainder of your records. Samples will be stored at Salimetrics for 30 days following the processing. After 30 days, samples will be disposed of into a residual waste collection and removed with other waste from the Salimetrics facility.

Sharing the Results
The results will not be shared and will only be reported in the aggregate (averages and medians).

Right to Refuse or Withdraw
As mentioned previously, you have the right to withdraw without penalty from the experiment at any time.

Who to Contact
Should you have any questions, you can ask them now or any time in the future. To ask questions later, contact either of the following:

Mr. Nicholas Stapleton, Principal Investigator
   E-mail: staple3f@my.erau.edu
   Call/Text: (315) 317-5837

Dr. Jonathan French, Advisor
   Phone: (386) 226-6284
   E-mail: Frenc70f@erau.edu

IRB Approval
The Institutional Review Board of Embry-Riddle Aeronautical University has reviewed and approved this study to ensure the experiment is not a risk to your health and wellbeing.
Part II: Certificate of Consent

I have read the information provided to me in Part I of the Informed Consent Form and have had opportunity to ask questions about it and the study. These questions have been answered to my satisfaction. I voluntarily consent to be a participant in this study.

Print Participant Name: _____________________________

Participant Signature: _____________________________

Date: __ / __ / ___

Statement by the researcher/person taking consent

I have read out all the information accurately to the potential participant and have, to the best of my ability, ensured that the participant understands the information provided to them on this form. I confirm that the participant was allowed the opportunity to ask questions about the study, and all questions were answered to the best of my ability. I also confirm that the potential participant has not been coerced into giving consent and the consent has been given freely and voluntarily.

A copy of this consent form has been provided to the participant.

Print Name of Researcher/Person taking consent: _____________________________

Signature of Researcher/Person taking consent: _____________________________

Date: __ / __ / ___
Appendix D

Simulator Sickness Questionnaire

Please complete all lines of this questionnaire. Each line is a separate symptom. Circle the strength of each symptom you feel.

<table>
<thead>
<tr>
<th></th>
<th>General discomfort</th>
<th>None</th>
<th>Slight</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>Fatigue</td>
<td>None</td>
<td>Slight</td>
<td>Moderate</td>
<td>Severe</td>
</tr>
<tr>
<td>3</td>
<td>Headache</td>
<td>None</td>
<td>Slight</td>
<td>Moderate</td>
<td>Severe</td>
</tr>
<tr>
<td>4</td>
<td>Eyestrain</td>
<td>None</td>
<td>Slight</td>
<td>Moderate</td>
<td>Severe</td>
</tr>
<tr>
<td>5</td>
<td>Difficulty focusing</td>
<td>None</td>
<td>Slight</td>
<td>Moderate</td>
<td>Severe</td>
</tr>
<tr>
<td>6</td>
<td>Salivation increase</td>
<td>None</td>
<td>Slight</td>
<td>Moderate</td>
<td>Severe</td>
</tr>
<tr>
<td>7</td>
<td>Sweating</td>
<td>None</td>
<td>Slight</td>
<td>Moderate</td>
<td>Severe</td>
</tr>
<tr>
<td>8</td>
<td>Nausea</td>
<td>None</td>
<td>Slight</td>
<td>Moderate</td>
<td>Severe</td>
</tr>
<tr>
<td>9</td>
<td>Difficulty concentrating</td>
<td>None</td>
<td>Slight</td>
<td>Moderate</td>
<td>Severe</td>
</tr>
<tr>
<td>10</td>
<td>Fullness of the head</td>
<td>None</td>
<td>Slight</td>
<td>Moderate</td>
<td>Severe</td>
</tr>
<tr>
<td>11</td>
<td>Blurred vision</td>
<td>None</td>
<td>Slight</td>
<td>Moderate</td>
<td>Severe</td>
</tr>
<tr>
<td>12</td>
<td>Dizziness eyes open</td>
<td>None</td>
<td>Slight</td>
<td>Moderate</td>
<td>Severe</td>
</tr>
<tr>
<td>13</td>
<td>Dizziness eyes close</td>
<td>None</td>
<td>Slight</td>
<td>Moderate</td>
<td>Severe</td>
</tr>
<tr>
<td>14</td>
<td>Vertigo</td>
<td>None</td>
<td>Slight</td>
<td>Moderate</td>
<td>Severe</td>
</tr>
<tr>
<td>15</td>
<td>Stomach awareness</td>
<td>None</td>
<td>Slight</td>
<td>Moderate</td>
<td>Severe</td>
</tr>
<tr>
<td>16</td>
<td>Burping</td>
<td>None</td>
<td>Slight</td>
<td>Moderate</td>
<td>Severe</td>
</tr>
</tbody>
</table>

(Kennedy et al., 1993)
## The Stanford Sleepiness Scale (SSS)

<table>
<thead>
<tr>
<th>Degree of Sleepiness</th>
<th>Scale Rating</th>
</tr>
</thead>
<tbody>
<tr>
<td>Feeling active, vital, alert, or wide awake</td>
<td>1</td>
</tr>
<tr>
<td>Functioning at high levels, but not at peak; able to concentrate</td>
<td>2</td>
</tr>
<tr>
<td>Awake, but relaxed; responsive but not fully alert</td>
<td>3</td>
</tr>
<tr>
<td>Somewhat foggy, let down</td>
<td>4</td>
</tr>
<tr>
<td>Foggy; losing interest in remaining awake; slowed down</td>
<td>5</td>
</tr>
<tr>
<td>Sleepy, woozy, fighting sleep; prefer to lie down</td>
<td>6</td>
</tr>
<tr>
<td>No longer fighting sleep, sleep onset soon; having dream-like thoughts</td>
<td>7</td>
</tr>
<tr>
<td>Asleep</td>
<td>X</td>
</tr>
</tbody>
</table>
Appendix F

Switching Test Instructions

The Switching Test Instructions

The Switching test is our cognitive assessment tool. It is a test that is entirely contained on a small USB computer plug-in device which you will be assigned upon entering the study. The Switching test is a challenging task but we are confident you will learn to do it quickly and accurately within the first 7 days of the study.

Once started, the Switching test takes about **10 minutes**.

**Steps**

1. The “Battery Selection” window will be loaded. Add your ID # _____. Click “Next”

![Battery Selection Window](image)

2. The first time you use the software and your ID # an “Information” window will appear. Click “Yes”.
3. Verify you ID # on the “Confirmation” window. Click “Yes” if correct.

4. The “Test Settings” window will appear. Click “Next”

5. A description about the Switching test will appear. Please take a moment to read them. Press the space bar on your keyboard to continue.
6. The next window will display actual **Instructions** for the test. Take a moment to read them. Make sure you understand them before pressing your keyboard’s space bar.

When the Manikin test is indicated:

Decide which of the man’s hands holds the object at the bottom of the screen.

If the object is in the man’s Left hand, press keyboard key W.
If the object is in the man’s Right hand, press keyboard key D.

When the Mathematical Processing test is indicated:

If the answer is Less Than 5, press keyboard key J.
If the answer is Greater Than 5, press keyboard key I.

Be fast AND accurate!

7. The next window will display the name of the test, “Switching”. Press space bar to start the test.
8. A red arrow will indicate the task that needs to be answered. The red arrow will switch back and forth between the tasks so your job is to pay attention to when the switch occurs and answer the correct task. Be sure to answer quickly because the program only gives you a few seconds before it moves onto the next question.

9. After finishing the test, a window will display the percent of correct answers (see a sample below). The researcher will record your results. Please DO NOT press the space bar.
If you would like more information about the Switching test or any task in this study, please feel free to contact Nicholas Stapleton:

E-mail: staple3f@my.erau.edu
Text: (315) 317-5837

Thank you for your interest and participation!

(Adapted from Rodriguez-Jiménez, 2012)