



Review

Motion sickness: A negative reinforcement model

Brad Bowins*,¹

University of Toronto Student Services, 214 College Street, Main Floor, Toronto, Ontario, Canada M5T 2Z9

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ABSTRACT

Theories pertaining to the “why” of motion sickness are in short supply relative to those detailing the “how.” Considering the profoundly disturbing and dysfunctional symptoms of motion sickness, it is difficult to conceive of why this condition is so strongly biologically based in humans and most other mammalian and primate species. It is posited that motion sickness evolved as a potent negative reinforcement system designed to terminate motion involving sensory conflict or postural instability. During our evolution and that of many other species, motion of this type would have impaired evolutionary fitness via injury and/or signaling weakness and vulnerability to predators. The symptoms of motion sickness strongly motivate the individual to terminate the offending motion by early avoidance, cessation of movement, or removal of oneself from the source. The motion sickness negative reinforcement mechanism functions much like pain to strongly motivate evolutionary fitness preserving behavior. Alternative why theories focusing on the elimination of neurotoxins and the discouragement of motion programs yielding vestibular conflict suffer from several problems, foremost that neither can account for the rarity of motion sickness in infants and toddlers. The negative reinforcement model proposed here readily accounts for the absence of motion sickness in infants and toddlers, in that providing strong motivation to terminate aberrant motion does not make sense until a child is old enough to act on this motivation.

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1. Introduction

The “why” of motion sickness has received very little attention compared to the “how” of this very disturbing condition. There are numerous articles pertaining to variations of sensory conflict theory and postural instability hypothesis, currently the two most popular perspectives regarding the “how” of motion sickness. In contrast there are only two theories, with little attention paid to them, focusing on why such a seemingly non-adaptive phys-

iological event occurs. Based on its presence in so many species and virtually every human possessing an intact vestibular system, motion sickness has a very strong biological basis [11,8]. This reality suggests that there must be an evolutionary fitness enhancing function to the condition.

The two “why” theories examining the evolutionary fitness advantage of motion sickness will be referred to as the toxin and movement program theories. Both theories have significant shortcomings and neither is able to account for why motion sickness is rare or absent in infants and toddlers. An alternative highly parsimonious explanation is posited here that accounts for the lack of motion sickness in the very early years of life. Essentially, motion sickness evolved as a form of negative reinforcement providing potent motivation for the cessation of any motion producing sensory conflict or postural instability. Aberrant motion of this form

* Tel.: +1 416 322 7935/978 8070; fax: +1 416 322 7951/978 7341.

E-mail address: brad.bowins@bellnet.ca.¹ Private Practice, 2200 Yonge Street, Suite 1700, Toronto, Ontario, Canada M4S 2C6.

would have greatly increased the risk of injury or signaled weakness and vulnerability to predators, thereby reducing evolutionary fitness.

2. Alternative “why” theories

Motion sickness has been described as an evolutionary anomaly, given that such a powerful mechanism seems to have evolved in so many species when there does not appear to be any survival value in the occurrence [4,2]. Yates et al. [12] suggest that there might not be an evolutionary aspect to motion sickness. According to these researchers motion sickness results from aberrant activation of neural pathways that serve to maintain a stable internal environment, with conflicting signals regarding body position in space producing atypical activation of brainstem neurons normally serving to maintain homeostasis, resulting in emesis. Instead of offering a non-evolutionary why theory of motion sickness, their explanation provides a possible how mechanism based on sensory conflict. There is no elaboration of why aberrant activation occurs in the first place—the domain of why theories. We are left guessing why this would occur, but assuming it is non-evolutionary the only reasonable explanation is a disease process either metabolic or infectious to explain the aberrant activation.

Beyond the reductionist nature of such an explanation when applied to phenomena of universal prevalence, motion sickness does not fit a disease model. Short of rare time limited pandemic infections, disease occurs in a subset of the larger population and arises from an interaction of genetic (diathesis) and environmental influences (stress). For example, with Type II diabetes there is a genetic vulnerability to disordered glucose metabolism and the stress of insulin resistance related to aging and excess body weight. Not everyone is able to develop diabetes and other diseases. With motion sickness present over recorded history in everyone with an intact vestibular system, and multiple and diverse animal types, a disease model does not fit at all. The inter-species and intra-species commonality supports an evolutionary basis.

Proposing an evolutionary advantage for inherently positive behavior is much easier than for what clearly appears to be maladaptive behavior, likely accounting for so few theories regarding the “why” of motion sickness. After all, how could it possibly be adaptive to feel violently ill and become dysfunctional during challenging circumstances? Such an occurrence would seem to represent an instance where biologically based behavior is maladaptive. Hence, the toxin and movement program theories have an uphill battle from the start. Treisman [11] proposed that movement control mechanisms provide an early warning system for the detection of neurotoxins. Working from a sensory conflict perspective regarding the “how” of motion sickness, Treisman [11] indicates that neurotoxins will produce mismatch between sensory (vestibular) and eye coordination systems given the continual action and high degree of susceptibility to disruption of these processes. The toxin mechanism serves as a backup to taste and emesis evoked by effects on the gastrointestinal lining or stimulation of chemoreceptors after absorption.

While intriguing there are several major problems with the toxin theory beyond it representing a non-parsimonious and highly complex mechanism. As Treisman [11] indicates, evolution has already provided mechanisms for dealing with toxins in the form of taste and the response of the gastrointestinal systems before and after absorption. In addition, the liver has evolved as an organ largely responsible for ridding the body of toxins. To evoke an additional mechanism, and then only for toxins capable of crossing the blood–brain barrier, might be considered somewhat redundant. Furthermore, not all motion sickness leads to vomiting, and significantly less so than physical disgust reactions. If toxin removal

constitutes the key purpose for motion sickness vomiting would always occur. Of course, for neurotoxins to induce the motion sickness response they must already be present in the brain where they cannot be removed by vomiting.

To produce the desired effect the toxin mechanism relies on direct sensory conflict between the vestibular and eye coordination systems. Some versions of sensory conflict theory emphasize direct conflict between the senses as implied by the name, but it is doubtful that different types of sensory input can actually be compared directly [10]. A more valid version of sensory conflict theory takes the form of a “neural mismatch” hypothesis whereby perceived motion is at variance with expected motion [7,6]. Whereas the brain might not be able to directly compare different types of sensory input, it does seem to be capable of forming expectations of motion based on experience. For example, no one perceives walking as unusual, whereas most people perceive flipping upside-down to be odd. Another major challenge to the toxin theory is that infants and toddlers with rapidly developing brains most sensitive to toxins do not experience motion sickness [8,9]. It simply does not follow that fully developed brains less vulnerable to most neurotoxins would have a pronounced toxin ejection mechanism, and the brains of those highly sensitive to most neurotoxins would lack the mechanism.

Regarding support for the toxin theory there has only been one instance of evidence since the theory was proposed [12,5]. Money and Cheung [5] observed that labyrinthectomy in 7 dogs increased the latency and threshold for vomiting in response to some emetic drugs. Their results fail to support the theory for several reasons. First, the substances tested are not toxins per se but emetic agents. Second, while the emetic response was reduced for some of these drugs (lobeline, levodopa, nicotine) it was not for others (apomorphine, pilocarpine). If the toxin theory is valid there should not be a selective effect for only some “toxins” that cross the blood–brain barrier. The mechanism is designed as a final backup, and thus has to act on all toxins that enter the brain. Third, as Yates et al. [12] indicate the removal of vestibular input due to labyrinthectomy results in disfacilitation of central emetic circuitry, providing a more plausible mechanism for the results of Money and Cheung [5]. Fourth, dogs are distinct from many other species in so far as drug effects on motion sickness are concerned, and consequently the dog model of motion sickness has largely been abandoned [12]. Hence, generalization of the Money and Cheung results for labyrinthectomized dogs to any other species is dubious at the best. Fifth, there is the possibility that the results cannot be generalized at all given some inexplicable findings of the study. The emetic response to apomorphine, lobeline, levodopa, and nicotine act on the area postrema, while that of pilocarpine depends on forebrain structures [1]. Money and Cheung found that the emetic response of apomorphine and pilocarpine were unaffected but that of lobeline, levodopa, and nicotine was reduced, a finding that seemingly lacks a neurobiological basis given that the results for apomorphine and pilocarpine should logically diverge. Therefore, for a variety of solid reasons the very limited “support” for the toxin theory provided by the Money and Cheung study cannot be viewed as valid.

The second “why” theory of motion sickness suggests that the innate displeasure generated by movement programs yielding vestibular conflict discourage the development of these programs [3]. The displeasure resulting from vestibular conflict trains and conditions the spatial orientation system to develop perceptual-motor programs that are efficient in the operating environment of the individual. Once again we see the emphasis on conflict between sensory inputs that might not be directly comparable. However, three other considerations are more damaging to this theory. First, positive reinforcement provides a powerful training mechanism for spatial orientation programs making another system largely redundant. As an example, a young child obtains

rewards by successfully maintaining balance and walking to a desired object. Second, training of the spatial orientation system to develop efficient perceptual-motor programs is highly active in the very early years of life when motion sickness is rare. Third, the training suggested constitutes punishment consisting of “displeasure” in response to movement programs that yield vestibular conflict. Punishment is not a reliable motivational framework typically producing inconsistent results. For example, punishing a child for a certain behavior often just results in the child being more secretive about the behavior.

3. A negative reinforcement model

Motion significantly different than expected or that producing postural instability would have reduced evolutionary fitness due to risk of injury and/or signaling weakness and vulnerability to predators. During our evolution and that of most other species, circumstances where motion was beyond that expected or involving postural instability meant uncontrolled states where the risk of injury was high. For example, while descending a slope a person slips, spinning and twisting to the bottom. Major fractures were likely in this scenario and any observant predator would detect a state of weakness and vulnerability. Many species would suffer in terms of evolutionary fitness from motion of this type. The relatively few species that do not experience motion sickness such as rabbits appear to have evolved motion patterns different than the expected to deceive predators. They are also likely to have well-developed capacities to avoid fitness reducing injuries arising from unstable motion.

If we assume that motion beyond the expected or that producing postural instability reduced evolutionary fitness, having a natural mechanism to avoid and terminate such motion would make sense. It is posited that motion sickness evolved as a potent negative reinforcement system, much like pain, designed to avoid and terminate aberrant motion. Any motion that produces a “neural mismatch” with that expected on the basis of the individual’s experience either triggers a signal or amplifies an existing signal activating this negative reinforcement system. Likewise, instances of postural instability can activate the defense. Reinforcement increases the frequency of a behavior as opposed to punishment diminishing a behavior. Positive reinforcement does so by positive outcomes, while negative reinforcement does so via the lessening, elimination, or removal of a noxious experience.

No sensation, other than perhaps pain, is as noxious as motion sickness. With the slightest motion sickness sensation people naturally reduce or cease activity to lessen or eliminate the horrible sensation. At the first instance of a neural mismatch between experienced and expected motion and/or significant postural instability, nausea and autonomic-based sensations arise to provide negative reinforcement for behavior designed to stop the offending motion. If the motion is not terminated the sensation intensifies to the point of producing an urge to vomit. Vomiting, due to motion sickness at least, is inconsistent with substantial movement, ensuring that this most extreme of motion sickness sensations terminates or lessens motion. Behavior negatively reinforced by the motion sickness sensation can include early avoidance, reduction or cessation of motion, and removal of the self from the offending circumstance. For instance, the person in our example sliding down the slope will be highly motivated to stop the motion by grabbing at whatever might arrest the fall. If the ground itself is sliding such as due to a landslide the person is motivated to grab hold of a tree branch or jump to secure ground.

With the right intensity of provocation, motion sickness arises immediately. For example, people often experience immediate and intense symptoms on an amusement park ride that severely

challenges them in terms of expected motion and/or postural instability. No long buildup of symptoms is required in this circumstance. In many instances, perhaps more relevant outside of a natural context, challenges to expected motion and/or postural instability develop gradually. As an example, the motion of a boat heading out into open water, or the vibrations of a car on a rough road. In response to these lesser provocations mild nausea and autonomic symptoms can intensify gradually as the challenge builds, such as a boat moving into choppy water. With more intense provocation and no cessation of the motion, a strong urge to vomit takes hold to ensure the cessation of motion. From an adaptive perspective inducing vomiting with the associated dysfunction too early in the motion sickness response might be disadvantageous. Induction of emesis transpires when there is failure to respond defensively to the initial motion sickness sensations.

During evolution, when confinement to motion sickness inducing circumstances was rare, it is likely the case that provocations were more immediate with rapid and intense activation of the negative reinforcement system, and a more definitive response prior to full-blown manifestations such as vomiting. The natural environment wields a sharp knife and organisms persisting in behavior with a substantial probability of injury or predation run the risk of being quickly eliminated. Natural processes typically display a range of expressions as opposed to all or none. Motion sickness as a natural evolved process shows a range of expression with some individuals profoundly sensitive and others not so much. It is feasible that those on the extreme high end of the vulnerability spectrum, such that they experience an intense urge to vomit with the mildest provocation, represent a disease variant based on excessive genetic loading. For these rare individuals motion sickness will diminish the defensive response to aberrant motion, given that they will be dysfunctional from the outset. Those on the low vulnerability end of the spectrum might feel fortunate that motion sickness is rarely experienced and only builds gradually even with significant provocation. However, these very individuals might miss out on the adaptive benefit of rapid avoidance, escape, reduction, or elimination of motion entailing a substantial risk of injury or predation. Of course in our modern day environment of safe aberrant motion aboard sources of transportation and amusement, and virtual reality worlds, the advantage appears to have shifted more to those less vulnerable to motion sickness. Since these sources of aberrant motion lack reproductive consequences the negative reinforcement system is not diminished.

For early *Homo sapiens* and most other species it was feasible to act in a fashion that might avoid, reduce, eliminate, or escape the offending motion, thereby minimizing the risk of injury or attack by a predator. Unfortunately, in our modern day environment most motion sickness is experienced while essentially trapped in a moving object such as a boat or fun fair ride. The offending motion is externally induced and hence ceasing one’s own motion has limited impact. Furthermore, short-range departure from the offending agent is also not feasible. As the adverse sensation persists in its most intense form and the motivation paradigm is not properly responded to suffering increases. People trapped in such circumstances typically demonstrate behavior designed to reduce the experience of motion such as lying still and watching the horizon. With enough exposure over days or weeks the experienced motion shifts into the expected category, eliminating the neural mismatch that typically activates the motion sickness negative reinforcement system.

The motion sickness negative reinforcement paradigm is very similar to how pain enhances evolutionary fitness. At first glance pain like motion sickness does not seem to make any sense given that it so severely impairs functioning. During evolution physical damage often significantly impeded fitness preserving or enhancing behavior, and would frequently have signaled weakness and

vulnerability to predators. Pain represents a potent negative reinforcement system strongly motivating the organism to terminate the pain-inducing state by avoidance, reduction, cessation, or removal of the damaging agent. Likewise, motion sickness provides potent negative reinforcement for rapidly and effectively dealing with offending motion circumstances. Of course the proposed motion sickness mechanism only makes sense if the individual has the physical capacity to avoid, reduce, terminate, or remove itself from the offending motion. A human infant or toddler lacks the capacity to do so, providing a very coherent reason why people of very young age are largely or completely immune to motion sickness. It is only when a child acquires the physical capacity to effectively deal with motion sickness circumstances that the negative reinforcement system begins to operate.

One interesting source of neural mismatch and/or postural instability potentially very relevant for the proposed negative reinforcement model is inner ear infections, referred to as acute labyrinthitis (of infectious origin). Some symptoms of labyrinthitis such as tinnitus and reduced hearing are not the same as those of motion sickness, but the core symptom of vertigo (perceived rotational movement of the surround or self) can activate the motion sickness response. Vertigo can readily trigger a mismatch between expected and experienced motion and induce postural instability. In our evolutionary context and even some modern day circumstances, labyrinthitis could be fitness reducing due to uncoordinated motions such as an unsteady wobbling gait. Motion of this type could result in significant injury or the appearance of weakness and vulnerability to ever-vigilant predators. By activating the motion sickness negative reinforcement system the fitness impairing behavior would be terminated or minimized, thereby preserving fitness.

It might be argued that labyrinthitis was of such significance during evolution that it drove the development of the motion sickness response in part or full. Labyrinthitis while not uncommon is not so common in modern day humans that this is likely, but we cannot say for sure that the same frequency of occurrence applies to early *H. sapiens*. It is more feasible that the vertigo symptom of labyrinthitis resulted in uncoordinated behavior exposing the individual to risk of injury and or predation. Activation of the negative reinforcement motion sickness response curtailed activity. In support of this proposed sequence, motion sickness rarely involves vertigo. If labyrinthitis played a major role in the evolution of the motion sickness response we would expect it to be a very common or universal symptom of motion sickness. Furthermore, it is very improbable that all the species experiencing motion sickness would suffer from very high rates of labyrinthitis during their evolution. A preliminary test would be to evaluate the prevalence of acute labyrinthitis of infectious origin in animal species affected by motions sickness compared to animals not affected by this condition. Another test would be to evaluate the prevalence of acute labyrinthitis in humans high and low in motion sickness propensity. Based on the low frequency of occurrence in humans it is very unlikely that acute labyrinthitis of infectious origin would be so much more common in the specified groups that it could drive the evolution of motion sickness.

A general critique of “why” theories of motion sickness is that they can never be proven or disproved. While this criticism appears to apply to the toxin and movement program hypotheses, the negative reinforcement model proposed here is testable. Implicit is the notion that those with a more pronounced motion sickness response will more effectively avoid, reduce, eliminate, or escape motion circumstances where there is a substantial risk of injury and/or predation. Both animal and human subject models can be designed to test this prediction. The central experimental procedure must be such that it satisfies the following criteria: first, readily elicit the motion sickness response but not in such an

intense fashion that it prevents subjects from acting defensively. Second, have a natural equivalent capable of resulting in significant injury, with the risk neutralized in the controlled setting. For example, a rotating and swaying platform that subjects can fall from but land in a safe medium or be restrained, although the restraining apparatus cannot reduce realism too greatly. Third, provide options for early avoidance, reduction, elimination, or escape from the motion paradigm. For example, when a subject steps from a stable platform onto the rotating and swaying platform there is the option of early avoidance by stepping right back onto the stable one. Motion could be reduced or eliminated by grabbing hold of a support bar or activating an appropriate control lever. Escape once significant motion occurs could be achieved by jumping to another more stable platform.

Subjects for animal studies would be divided into two groups either high and low on motion sickness propensity established by earlier trials, or intact vestibular system versus labyrinthectomized. Human studies would compare subjects high and low in motion sickness propensity, or bilateral vestibular dysfunction and intact vestibular function. Any subject who experiences vomiting at the slightest provocation should be excluded given that such an extreme response precludes testing of the hypothesis. Assessment would measure the frequency of early avoidance, reduction or elimination of aberrant motion, and escape responses, as well as the reaction time for these parameters in the two groups. It is predicted that subjects high in motion sickness vulnerability, and those with an intact vestibular system will demonstrate a higher frequency of motion diminishing responses, and/or faster reaction times for such responses. If the theory is not accurate, then motion sickness propensity should, if anything, decrease the frequency and increase the latency of “safe” responses via interference effects. The negative reinforcement model proposed here represents a significant advantage over other motion sickness “why” theories by providing testable hypotheses.

4. Conclusion

The negative reinforcement model for motion sickness posited here represents a logical and parsimonious explanation, with a solid precedent in the form of pain. Unlike the toxin and movement program perspectives this negative reinforcement model readily accounts for the puzzling issue of motion sickness being rare in infants and toddlers. It also accounts for other motion sickness occurrences such as the escalation of symptoms, the extremely adverse and seemingly dysfunctional induced state, and the tremendous relief experienced upon termination of the offending motion. Furthermore, it explains the presence of anxiety symptoms in motion sickness, in that circumstances evoking the sensation represented threat throughout our evolution.

The ‘why’ of motion sickness has been neglected relative to the ‘how’. This occurrence is understandable given that the seemingly detrimental nature of motion sickness contrasts so sharply with its clear evolution and biological basis in many species. Having a solid “why” in the form of a negative reinforcement model should assist with intervention strategies. Based on the negative reinforcement paradigm motion sickness will intensify with motion significantly different from that expected by the individual or that inducing postural instability, and persist for a substantial period of time if the offending motion is not terminated. Behavioral strategies will then be most effective for gradually increasing the range of expected motion, a procedure commonly used in training astronauts and military personnel. Pharmaceutical efforts might best be directed at the neural and autonomic mechanisms underlying negative reinforcement systems. Beyond the practical implications there is satisfaction in placing such a seemingly inexplicable entity as motion sickness in a solid evolutionary-based context.

Conflict of interest

None.

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