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The impact of vehicle motion during transport on animal welfare

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ABSTRACT

Motion sickness is a common response in humans and some species of farm livestock during transport, but research on the impact of motion has been primarily focused on the use of animal models for humans. During livestock transportation, animals seek to minimise uncontrolled movements to reduce energy consumption and maintain posture. Road and sea transport of livestock can produce motion sickness and stress responses. Clinical signs are the result of autonomous nervous system activation. Studies conducted on road transportation effects in domestic animals showed several motion sickness behaviours including vomiting and, in ruminants, a reduction in rumination. However, there is a lack of knowledge on the impact of sea transport motion. Despite the paucity of data on livestock, there is sufficient evidence to believe that motion might affect animal welfare when animals are transported by road or sea.

1. Introduction

There has been limited research into the impact that motion of a vehicle or vessel during transport has on the welfare of livestock. Most farm livestock are transported infrequently (Weeks, 2007). One of the most common and important consequences that both non-human animals (hereafter animals) and humans experience during transport is motion sickness. This term has been used mainly in humans to refer to discomfort associated with atypical patterns of passive motion (not initiated by the individual) during sea transport (sea sickness) (Aranda et al., 2005; Shupak and Gordon, 2006), road transport (train or truck sickness) (Lackner, 2009) and space transport (space sickness) (Muth, 2006), as well as more recent phenomena in which there is no vehicle involved, such as cybersickness and simulator sickness (Bonnet et al., 2006).

Motion sickness is a physiological reaction to motion patterns (Caillet et al., 2006), which integrates multiple responses from different physiological systems (Doweck et al., 1997) and affects most humans at least once in their lives (Fukutake and Hattori, 2000), in particular females (Lawther and Griffin, 1986). Motion sickness has been investigated in animal models mainly for human benefit (Chen et al., 2010), such as the use of fish as an experimental model to study space motion sickness, a research area of particular interest to astronauts (Anken and Hilbig, 2004).

Motion sickness has been demonstrated in a range of species, including squirrel monkeys (Brizzee et al., 1980), rats (Cai et al., 2010), dogs (Cannas et al., 2010; Doring-Schatzl and Erhard, 2004), cats (Crampton and Lucot, 1991; Lang et al., 1999) and the house musk shrew (*Suncus murinus*), which is an insectivore that has been used as an animal model for motion-induced emesis (du Sert et al., 2010; Uchino et al., 2001). Other species for which motion sickness has been described are fish (Anken and Hilbig, 2004), guinea pigs (Ossenkopp and Ossenkopp, 1990), pigs (Randall and Bradshaw, 1998), horses (Lee et al., 2001), sheep (Hall et al., 1998), seals and birds (Money, 1970). Some species of lower vertebrates, such as amphibians, are believed to be incapable of experiencing motion sickness because of the absence of the relevant brain structures, such as a vomiting centre (Lychakov, 2012).

This review includes human research literature on motion sickness because of its relevance for other species and knowledge obtained in this field, as well as the research on animals for the purposes of investigating animal transport. Implications for livestock welfare are the primary focus of the review. Both ship and road transport are considered where relevant information is available; no information is available for air travel. This topic is increasing in importance as the number of food animals exported annually has increased substantially over the last 50 years, e.g. from 2.6 to 36.5

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million pigs, 6.5–15.2 million sheep, 4.9–10.4 million cattle, and 0.8–1.4 million chickens (FAOSTAT, 2014).

2. Causes of motion sickness

There are two main theories for the causes of motion sickness. The first, broadly known and accepted, is the sensory conflict theory, also known as the sensory rearrangement theory (SRT) (Oman, 1982; Reason and Brand, 1975; Warwick-Evans et al., 1998). This states that "*all situations which provoke motion sickness are characterised by a condition of sensory rearrangement in which the motion signals transmitted by the eyes, the vestibular system and the non-vestibular proprioceptors are at variance either with one another or with what is expected based upon previous experience*" (Reason and Brand, 1975). Some authors partially agree with SRT, but still emphasise that subjective vertical experience by individuals is the major component (Bles et al., 1998; de Graaf et al., 1998). Others have rejected SRT because of its low predictive validity (Draper et al., 2001; Riccio and Stoffregen, 1991).

The second motion sickness theory emphasises control of body orientation (Bles et al., 1998) and is known as the postural instability theory. Environments that generate a prolonged postural instability will produce motion sickness, and individual behaviour responses are a key aspect of the aetiology of motion sickness (Owen et al., 1998; Riccio and Stoffregen, 1991; Stoffregen et al., 2010). Although this theory does not predict the environments that will produce long periods of postural instability, it is a useful alternative instrument to study motion sickness (Draper et al., 2001). However, some authors have observed that postural instability is not an essential pre-condition for motion sickness (Bos, 2010; Faugloire et al., 2007; Warwick-Evans et al., 1991). Instead, it is probably a contributing but not causative factor, but the degree of contribution in animal motion sickness is unknown.

The lack of resolution of these two theories emphasises that, despite its universal occurrence in humans and several animal species (Griffin, 1990), the many causes and mechanisms that produce motion sickness are poorly understood. Table 1 illustrates some of the many elements implicated in the causation of motion sickness (Griffin, 1990). The processing of the signals begins with activation of the visual and vestibular systems, causing awareness and then interpretation of the motion, followed by emergence of clinical signs, sweating, nausea, pallor, hypersalivation and gastrointestinal disturbances (Griffin, 1990).

The theoretical basis for motion sickness does not address the aetiology of the condition. In this sense, Bowins (2010) considered that motion sickness cannot be explained by a disease model and proposed instead an evolutionary anomaly, a theory that motion sickness evolved as a negative reinforcement mechanism to terminate an unusual motion. If individuals cannot eliminate or escape from a situation that produces motion sickness, they exhibit behaviours to reduce motion sickness effects, such as humans lying down when travelling by boat (Bowins, 2010). However, the fundamental process that produces motion sickness has not yet been confirmed (Buyuklu et al., 2009).

Table 1
Theoretical factors involved in causation of motion sickness, adapted to livestock transport (Griffin, 1990).

Motion characteristics	Animal factors
Acceleration	Experience
Frequency	Emotional state
Amplitude	Posture
	Age
	Sex
	Species/genotype

3. Symptoms and clinical signs of motion sickness

Susceptible humans show different symptoms when experiencing motion sickness that include evidence of autonomic nervous system (ANS) activity, mainly from the sympathetic branch, such as pallor, headache, loss of appetite, cold sweating, apathy, nausea, depression and reduction in cognitive function (Burton et al., 2010; Buyuklu et al., 2009; Chen et al., 2010; Lackner, 2009; Macefield, 2009). Motion sickness incidence fluctuates according to individual susceptibility and stimulus intensity (Buyuklu et al., 2009). Susceptibility to motion sickness in humans has been studied through questionnaires and experimental tests (Lackner, 2009), whereas in animals only experimental tests are possible (Kaji et al., 1990). However, humans and animals show similar gastrointestinal symptoms and clinical signs associated with motion sickness, including hypersalivation, pica (craving for and consumption of non-nutritive substances), nausea, intestinal peristalsis, defaecation and vomiting (Bos et al., 2008; Cai et al., 2010; Lang et al., 1999). Nonetheless, not all motion sickness results in vomiting (Bowins, 2010). Elevated pellet eating rates have been observed in response to heave (vertical) motion in simulated ship transport of sheep (Santurtun et al., 2013). Further research on the relationship between motion sickness and digestive disorders is warranted (Lang et al., 1999).

3.1. Nausea

Nausea is a negative sensation associated with the urge to vomit, which in mammals is less understood than the act of vomiting itself (Andrews, 2009). This is because it is not known, firstly, which sensory faculties an animal needs in order to experience motion sickness, secondly, what the criteria are for experiencing nausea (Holmes et al., 2009), and thirdly, how the feelings associated with nausea are quantifiable (Lang et al., 1999). However, there are 'behavioural equivalents' (Andrews, 2009) of nausea in animals, for example pica (McCaffrey, 1985), which are useful research tools to study nausea and motion sickness in animals. As with motion sickness, the sensory experiences cannot be studied in the same way in animals as in humans, for whom the use of questionnaires is commonplace (Golding, 2006a). In this sense, vomiting is an important and useful research indicator of motion sickness in those animal species that can perform this behaviour (Kaji et al., 1990); however, in humans at least, there is a substantial percentage of individuals that experience motion sickness but do not vomit (Shupak and Gordon, 2006). For example it has been reported that only 7% of passengers transported by sea vomit (Lawther and Griffin, 1988).

3.2. Vomiting

Vomiting, or emesis, is a protective response and coordinated reflex where upper gastrointestinal tract contents are forcefully ejected from the mouth (Frandson et al., 2009; Holmes et al., 2009). In relation to the animal species that experience motion sickness, there are few mammals (house musk shrew, cat, dog, pig, marmoset, sperm whale, ferret) and even fewer birds (pigeon and petrel), amphibians (salamander and frog), reptiles (snake and crocodile) or fish (shark and tuna) that appear to be able to vomit (Andrews, 2009; du Sert et al., 2010; Ebenezer et al., 1989; Holmes et al., 2009; Wassersug et al., 1993).

Some animal species, for example rats (Ebenezer et al., 1989; Lee et al., 2010), and mice and rabbits (Holmes et al., 2009), cannot vomit because they do not have the necessary reflex action (Andrews, 2009) as a result of their physiological and anatomical characteristics (Lee et al., 2010). Farm animals also rarely vomit because of anatomical characteristics (e.g. the horses' cardiac sphincter tone). Sheep, cows and goats rarely eject gastrointestinal contents from the mouth, except in cases where certain plant, soil or mineral toxins have been