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Charles M. El-Hage BVSc(HONS), MANZCVZ(Eq)^{1*}

Peter J. Huntington BVSc(HONS), MANZCVS(Eq)²

I.G. Joe Mayhew BVSc, DACVIM, MANZCVS³

Ronald F. Slocombe BVSc, MS, PhD, DACVP¹

Brett S. Tennent-Brown BVSc, MS, DACVIM, DACVECC¹

¹ Faculty of Veterinary and Agricultural Sciences, University of Melbourne Equine Centre, 250 Princes Highway, Werribee, VIC 3030, Australia

² Kentucky Equine Research (Australasia) Pty Ltd., 7/35 Dunlop Road, Mulgrave, VIC 3170, Australia

³ Institute of Veterinary, Animal and Biomedical Sciences. Massey University, Private Bag 11 222, Palmerston North 4442, New Zealand

* Author for correspondence; cmeh@unimelb.edu.au

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DR. CHARLES M EL-HAGE (Orcid ID : 0000-0002-6999-2664)

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C. M. El-Hage^{1*}, P. J. Huntington², I. G. Mayhew³, R. F. Slocombe¹ and B. S. Tennent-Brown¹

¹Faculty of Veterinary and Agricultural Sciences, University of Melbourne Equine Centre, 250 Princes Highway, Werribee, VIC 3030, Australia;

²Kentucky Equine Research (Australasia) Pty Ltd., 7/35 Dunlop Road, Mulgrave, VIC 3170, Australia;

³Institute of Veterinary, Animal and Biomedical Sciences. Massey University, Private Bag 11 222, Palmerston North 4442, New Zealand.

*Corresponding author email: cmeh@unimelb.edu.au

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Summary

Although described clinically for several centuries, Stringhalt remains an intriguing and enigmatic condition. In Pasture-associated Stringhalt (PSH), the clinical sign of exaggerated, prolonged hindlimb flexion is associated with a peripheral neuropathy affecting the larger myelinated axons that is thought to be the result of exposure to a plant-derived neurotoxin. It is likely that multiple host and environmental risk factors interact to produce PSH and the precise aetiology has not yet been elucidated. Drought-affected, poor-quality pasture and the presence of *Hypochoeris radicata* (commonly referred to as Catsear, Flatweed and False Dandelion) are recognised risk factors. Affected horses are typically mature adults and taller animals are considered more susceptible. Most horses with PSH recover spontaneously if removed from the presumptive source of toxin; however, recovery can be prolonged, taking several years for some horses and might be incomplete in occasional cases. A wide range of therapies have been attempted in horses with PSH including phenytoin, thiamine, taurine, infiltration of digital extensors with botulinum toxin, and lateral digital extensor myotectomy procedure. The efficacy of these treatments is uncertain since controlled trials have not been performed and the spontaneous recovery of most horses makes any response to treatment difficult to interpret.

Introduction

Stringhalt is a condition of horses characterised by excessive and prolonged flexion of one or both hindlimbs during forward or backward movement. The condition has been recognised for centuries and was referred to as “Springhalt” in Shakespeare’s play King Henry VIII, first performed in the early 1600s. Other terms for Stringhalt include Hahnentritt (German for rooster kick), and more recently, Equine Reflex Hypertonia (Hahn 2015; Mayhew 2009). Classical (or Unipedal) Stringhalt, predominantly affecting only one hindlimb, most often follows injury to one dorsal tarsal/metatarsal region (Supplementary video S4). However, the condition is considered to be idiopathic (Crabill *et al.* 1994; Sullins 2002). Pasture-associated Stringhalt (PSH) appears to be the result of exposure to a plant-derived neurotoxin or neurotoxins and subsequent peripheral neuropathy. Often referred to as Australian Stringhalt, PSH has also been termed “outbreak” or “epidemic” Stringhalt, as the condition commonly affects multiple animals in the same paddock and time period (Kendall 1887; Cahill *et al.* 1986; Gay *et al.* 1993; Araya *et al.* 1998; Takahashi *et al.* 2002; Torre 2005; Domange *et al.* 2010; de Pennington *et al.* 2011). Pasture-associated Stringhalt is most often reported in horses

grazing poor quality, drought-affected pastures and, although occasionally asymmetrical, the condition is invariably bilateral. Initially described in Australia and New Zealand, PSH has been reported in Europe, North and South America, and Asia (Cahill *et al.* 1985; Takahashi *et al.* 2002; Araujo *et al.* 2008; Mayhew 2009; Domange *et al.* 2010; de Pennington *et al.* 2011).

Clinical Presentation and Diagnosis

Horses with PSH have characteristic, though very variable, hyperflexion of both hindlimbs when attempting to move (Fig 1, Supplementary videos S1 and S2). A grading system has been proposed (I-VI) based upon prior reports in the literature (Huntington *et al.* 1989; Domange *et al.* 2010). In mild cases (Grades I and II, Table 1) the gait abnormality might only be apparent when the horse is excited or nervous, when it is turned sharply, or when it is forced to walk backward. Clinical signs can often be exacerbated while walking the horse down a slope, after a sudden stop or following hard exercise. Horses with more severe PSH exhibit a hopping action in which hyperflexion becomes progressively more exaggerated and of increased duration. In some horses, flexion is so extreme that the dorsum of the fetlock contacts the ventral abdomen and the duration of hyperflexion becomes so prolonged that both hind feet leave the ground, almost at the same time, even while walking (Grades IV and V, Table 1 and Supplementary video S2). In extreme cases, horses are unable to rise without assistance (Grade VI) (Pemberton and Caple 1980; Cahill *et al.* 1985; Huntington *et al.* 1989; Domange *et al.* 2010; Draper *et al.* 2014). Although both hindlimbs are invariably involved in cases of PSH, clinical signs can show some asymmetry (Huntington *et al.* 1989; Mayhew 2009). The gait abnormality is often worse in cold weather and appears to be worse in horses that appear agitated. The severity of hyperflexion might decrease after the initial few steps but the gait typically remains abnormal. Muscle atrophy occurs in most affected horses, with the long and lateral digital extensor muscles the most severely affected. Distal limb muscle atrophy is often obvious in the early stages of PSH. Many horses, particularly those with prominent signs of long duration, have atrophy of the thighs and adductor muscles. In such cases muscle wastage may extend to the entire hindquarters (Huntington *et al.* 1989; Domange *et al.* 2010).

A range of other gait abnormalities have been described in some cases of PSH including variation in the degree of hindlimb fetlock flexion, abduction of the limb during hyperflexion and caudal thrust of the hindlimb at the onset of protraction (Mayhew 2009). These variations in clinical signs might reflect the complexity and range of neuromuscular structures affected by the presumptive toxin. Very occasionally, the forelimbs are also affected in horses diagnosed with PSH. In these cases, knuckling, hypermetria and atrophy of the forelimb and knuckling of the hindlimb have been described in addition to hindlimb hyperflexion (Huntington *et al.* 1989). A high incidence

(up to 20% in some reports) of laryngeal hemiplegia has been reported amongst affected horses in several outbreaks of PSH and the abnormal vocalisation noted in some cases of PSH likely reflects laryngeal dysfunction (Cahill *et al.* 1985; Huntington *et al.* 1989; Araujo *et al.* 2008; Domange *et al.* 2010). The majority of horses with PSH have a normal demeanour and appetite; however, changes in behaviour have been reported. In a small number of severely affected horses, such findings have included increased aggression and obtundedness (Huntington *et al.* 1989; Domange *et al.* 2010).

Diagnosis of stringhalt has to be based on the clinical signs and absence of other neurologic and orthopaedic abnormalities. The distinction between bilateral PSH and classical unipedal stringhalt is usually apparent and can be supported by some evidence of trauma to the hock region and to presence or absence of epidemiological features discussed below. Results of routine haematologic analyses are typically non-contributory. Low plasma Vitamin E concentrations and mild increases in muscle enzymes (creatinine kinase and aspartate aminotransferase) and liver enzyme activity have been noted in occasional cases of PSH but abnormalities are inconsistent, usually mild and unlikely to be of clinical significance (Pemberton and Caple 1980; Araya *et al.* 1998; Gardner *et al.* 2005; Domange 2009; Domange *et al.* 2010). Electromyographic (EMG) studies of hindlimb muscles in horses with PSH have shown increased insertional activity, fibrillation potentials and positive sharp waves, consistent with denervation (Huntington *et al.* 1989; Huntington *et al.* 1991; Takahashi *et al.* 2002; Gardner *et al.* 2005; Armengou *et al.* 2010). Furthermore, mean conduction velocity of the peroneal nerve in four horses with PSH was approximately one third that of a control horse (Huntington *et al.* 1989). Abnormalities in EMG recordings gradually returned toward normal paralleling the clinical recovery in three horses with PSH when monitored over a period of eight months (Huntington *et al.* 1989).

The gait abnormality in PSH can vary considerably between individual horses and, as mentioned, some horses can display changes in their gait that are not typical of PSH. As a consequence, PSH might be difficult to distinguish from a number of other neuromuscular or musculoskeletal conditions particularly when the patient is mildly affected. Differential diagnoses for PSH might include Shivers, Scandinavian Knuckling Disease (acquired equine polyneuropathy), lathyrism, fibrotic myopathy and upward fixation of the patella, in addition to conditions affecting upper motor neuron innervation of the hindlimbs. It appears that horses with pain, or even irritating stimuli (such as application of a leg wrap) relating to the distal limb or hoof may produce a Stringhalt-like gait (Supplementary video S3). In most cases, careful examination of the gait and assessment of the history should allow differentiation between these conditions and PSH.

Shivers is a relatively rare movement disorder that has recently been shown to be associated with degeneration of Purkinje cell axons within the deep cerebellar nuclei (Valberg *et al.* 2015). Horses with Shivers show protracted hyperflexion of the hindlimb when the limb is manually lifted or when the horse is backed. Some affected horses will hyperextend (rather than hyperflex) their hindlimbs during backing. Forward movement is performed normally in most horses with Shivers. However, a recent study described a subset of affected animals that also had intermittent hindlimb hyperflexion during forward movement (Draper *et al.* 2014). Muscle atrophy, which is expected in Stringhalt as a consequence of the neuropathy, is also reported in horses with Shivers. Interestingly, Shivers also appears to occur more frequently in taller horses. Features of Shivers that do not seem to be present or occur infrequently in Stringhalt include abduction of the hindlimb during hyperflexion, elevation of the tailhead and facial twitching. In addition, Shivers tends to be a progressive disease rather than resolving spontaneously (Baird *et al.* 2006; Draper *et al.* 2014). Clearly there appears to be an overlap in the clinical signs of Stringhalt and Shivers and this is hampered by a lack of definitive diagnostic testing in many cases. As a consequence, the two conditions might be difficult to confidently separate clinically. Distinction might be aided by epidemiological features, particularly where there is exposure to *Hypochaeris radicata* (*H. radicata*) and the presence of multiple affected animals.

Acquired Equine Polyneuropathy (commonly referred to as Scandinavian Knuckling Syndrome) is a condition that shares some epidemiological and pathological features with PSH. However, rather than hindlimb hyperflexion, affected horses usually have symmetrical digital extensor dysfunction with knuckling of the metatarsophalangeal joint and stumbling (Hahn *et al.* 2008; Grondahl *et al.* 2012). Severely affected horses can become rapidly recumbent and many are subsequently euthanised but, as noted in PSH, mildly affected animals often make a full recovery over several months (Grondahl *et al.* 2012). Like PSH, the condition occurs in outbreaks but most cases of AEP occur between mid-winter and spring. Acquired Equine Polyneuropathy has been anecdotally linked to feeding practices (affected horses have often been fed wrapped forage) but epidemiological studies have not yet found differences in management (including feeding practices) between affected and unaffected horses (Wolff *et al.* 2014). Limited studies have shown that, as with Stringhalt, AEP is associated with injury to the peripheral nerves but it is the Schwann cells that appear to be the primary target with subsequent demyelination (Hahn *et al.* 2008; Grondahl *et al.* 2012). Both conditions may represent a spectrum of a peripheral neuropathy induced by a plant associated toxin manifesting in different clinical presentations.

Pathological Findings

To date, pathologic lesions identified in horses with PSH have been restricted to the peripheral nervous system (PNS) and to the distal muscles supplied by affected nerves. Although signs including behavioural changes may be indicative of CNS dysfunction, histopathological lesions have not yet been described in the brain of affected horses (Cahill *et al.* 1986; Huntington *et al.* 1989; Slocombe *et al.* 1992; Mayhew 2009). Additionally, histological evaluation of the spinal cord in affected animals has so far been unrewarding (Cahill *et al.* 1985; Slocombe *et al.* 1992; Domange *et al.* 2010).

At a histological level, PSH is characterised by a Wallerian-type distal axonopathy with decreased numbers of large myelinated nerve fibres within peripheral nerve trunks, and perineural fibrosis (Cahill *et al.* 1985; Slocombe *et al.* 1992; Domange *et al.* 2010). Generalised demyelination is not present. Rather, the larger, longer myelinated nerves such as the tibial, deep and superficial peroneal and recurrent laryngeal are generally most affected (Slocombe *et al.* 1992). Other common findings include Schwann cell proliferation and “onion bulb” formations indicative of demyelination and re-myelination of affected neurons (Cahill *et al.* 1986; Slocombe *et al.* 1992; Domange *et al.* 2010). Some of the early hallmark signs of distal axonopathies such as myelin digestion chambers, macrophages infiltration, myelin debris and axon fragments occur but may not be obvious in post mortem samples from more chronic cases, but might be apparent in ante mortem samples or samples collected from more acute cases (Slocombe *et al.* 1992; Armengou *et al.* 2010). Given their role in regulation of limb motion, lesions within muscle spindles might be expected, although a systematic study of these structures has not been reported to date.

Changes consistent with neurogenic atrophy have been described in the dorsal crico-arytenoid, cranial tibial, lateral digital extensor, long digital extensor, gracilis, and deep digital flexor muscles (Cahill *et al.* 1986; Slocombe *et al.* 1992; Domange *et al.* 2010). When examined, the most severe changes have been found in the dorsal crico-arytenoid muscles (Cahill *et al.* 1986; Slocombe *et al.* 1992; Domange *et al.* 2010). Histologically, muscle fibres appear shrivelled, angular and pale and in some instances muscle fibres might be completely absent leaving only compacted epimysium. In some horses, affected muscle groups are simply severely atrophic while in others there is extensive replacement with fibrous connective tissue and fat. Whether this reflects some selectivity in the mechanism of neuromuscular injury or is a result of further injury to already atrophic muscles is unknown.

Compensatory hypertrophy of scattered muscle fibres in the margins between atrophic and normal areas of muscle is a common finding (Fig 2). Histochemical staining shows that, of the remaining muscle fibres, the relative proportion of Type I (oxidative type) fibres is increased indicating a loss of Type II fibres in horses with PSH. This loss of Type II fibres, hence change in ratio of fibre types, has

been noted in other neuromuscular disorders (Andrews *et al.* 1986; van der Hoven *et al.* 1988; Slocombe *et al.* 1992).

Epidemiology

Pasture-associated Stringhalt most often occurs in horses grazing poor quality pasture on unimproved soils (Cahill *et al.* 1985; Huntington *et al.* 1989; Araya *et al.* 1998; Domange *et al.* 2010). The presence of *H. radicata* (Catsear, Flatweed, False Dandelion; Fig 3) in pastures grazed by horses that develop PSH has been consistently reported (Pemberton and Caple 1980; Cahill *et al.* 1985; Huntington *et al.* 1989; Mayhew 2009; Domange *et al.* 2010). *Hypochoeris radicata* is a perennial, edible herb native to Europe but an invasive weed that has been introduced to Australia, New Zealand, the Americas and parts of Asia. An Australian study reported that 50 of 52 horses diagnosed with PSH had grazed on pastures containing a large amount of *H. radicata* and a more recent report from France identified exposure to *H. radicata* in 69 of 70 affected horses (Pemberton and Caple 1980; Cahill *et al.* 1985; Huntington *et al.* 1989; Mayhew 2009; Domange *et al.* 2010). Other weeds, including *Taraxacum officinale* (common dandelion) and *Malva parviflora* (marshmallow, mallow weed), have very occasionally been associated with PSH cases (Pemberton and Caple 1980; Huntington *et al.* 1989; Takahashi *et al.* 2002; Armengou *et al.* 2010). Cases typically occur in late summer or early autumn after several weeks of grazing the incriminated pastures and reports of PSH typically increase during drought conditions (Kannegieter 1989; Domange *et al.* 2010). *Hypochoeris radicata* has a long tap root (Fig 3) affording the plant some resistance to drought conditions. This plant, might therefore, provide green herbage in paddocks with otherwise sparse dry forage. However, some horses have also been observed to selectively graze *H. radicata* (Pemberton and Caple 1980; Huntington *et al.* 1989). The onset of clinical signs can be delayed and disease might not become evident until one to 3 weeks after removal from affected paddocks (Huntington *et al.* 1989; Gardner *et al.* 2005). Multiple horses in the same paddock are usually affected and, occasionally all, exposed animals will develop clinical signs. However, more commonly, only a proportion of horses develop signs suggesting that exposure alone is insufficient to cause disease (Cahill *et al.* 1985; Huntington *et al.* 1989; Araya *et al.* 1998; Takahashi *et al.* 2002; Domange *et al.* 2010). Most cases of PSH occur in mature full-sized horses, with taller animals appearing to be at greater risk, although the condition has been described occasionally in ponies, younger horses and donkeys (Huntington *et al.* 1989; Araya *et al.* 1998; Araujo *et al.* 2008; Domange *et al.* 2010). It has been speculated that young or smaller animals might be protected from PSH by virtue of the shorter length of their neurons.

Cattle and sheep grazing pastures incriminated in PSH are not affected. This presumably is because toxins are broken down by rumen microflora. Other monogastric grazing species also do not appear to be affected.

Pathogenesis and Possible Aetiologies

The gait abnormalities of PSH are often exacerbated when affected horses appear agitated or disturbed. Conversely, sedation ameliorates clinical signs in some animals (Huntington *et al.* 1989; Takahashi *et al.* 2002; Domange *et al.* 2010). This, and the observation that some horses with PSH exhibit changes in behaviour, has been interpreted to support CNS involvement in the pathogenesis of PSH (Dixon and Stewart 1969; Huntington *et al.* 1989; Huntington *et al.* 1991; Domange *et al.* 2010). Despite these observations, the neurologic lesions in horses with PSH have been entirely restricted to the peripheral nerves to date. The recent report of cerebellar lesions described in horses with the allied syndrome Shivers suggesting that further investigation of the CNS in horses with PSH might be warranted (Valberg *et al.* 2015). Disease affecting the peripheral nerves is expected to produce hyporeflexia and hypotonia rather than the hyperreflexia and hypertonia observed in PSH. There is not a ready explanation for this discrepancy but a well-accepted hypothesis suggests that the neuropathy preferentially involves the larger type 1a- and 1b- afferent fibres and/or gamma motor efferent nerve fibres supplying muscle spindles and Golgi tendon organs (Fig 4) (Mayhew 2009). These axons form part of the myotatic reflexes synapsing with inhibitory interneurons within the spinal cord that modulate motor tone and the onset of volunteer and reflex alpha motor neuron firing. Dysfunction of these circuits would be expected to lead to disinhibition with resultant hypertonicity and hyperreflexia (Mayhew 2009). Involvement of the entire hindlimb is a result of the passive mechanical structures that comprise the reciprocal apparatus of horses' hindlimbs (Mayhew 2009; Hahn 2015). This hypothesis is supported by the observation that myotectomy of the lateral digital extensor reduces gait abnormalities in many affected horses (Torre 2005; Mayhew 2009; Domange *et al.* 2010; Dyson and Ross 2011).

Although there is a consistent epidemiological association between the presence of *H. radicata* and the development of PSH, attempts to experimentally induce Stringhalt have been limited and largely unsuccessful (Seddon and Belschner 1926; Araujo *et al.* 2008). Injection of a "concentrated extract of *H. radicata* into laboratory animals" did not induce clinical signs consistent with PSH (cited by (Pemberton and Caple 1980). A Stringhalt-like gait was induced in a 6-month old colt fed large amounts of *H. radicata* (9.8 kg dry matter/day) over a 50-day period, collected from paddocks that had contained horses with clinical PSH. Interestingly, the colt's gait improved when the *H. radicata* was collected from a paddock in which there were no affected horses and worsened again when

changed back to the original source (Araujo *et al.* 2008). While these findings are interesting and support the role of *H. radicata* in PSH, the rapid change, and particularly the rapid improvement, in clinical signs is not consistent with natural disease. Furthermore, young (and smaller) animals are rarely affected with PSH and further work is required in this area of investigation.

A number of studies have attempted to identify a neurotoxin or toxins produced by *H. radicata* that could explain the clinical signs of PSH. Ingestion of *Lathyrus spp.* (sweet pea) plants causes disinhibition of neural impulses and produces a hypermetric, Stringhalt-like gait (Mayhew 2009). Although several toxic agents have been identified in these plants, the neurotoxicity is thought to be due to neurotransmitter agonists (Spencer *et al.* 1986; Holbrook *et al.* 2015). Mycotoxins are well recognised causes of neurological diseases in grazing animals and have frequently been suggested as a cause of PSH (Pemberton and Caple 1980; Cahill *et al.* 1985). However, pathological lesions consistent with known mycotoxicoses have not been detected in cases of PSH. Although examination of soil samples collected from paddocks that had contained horses with PSH revealed high levels of fungal microflora, the clinical significance of this observation is unclear (Huntington *et al.* 1989). No evidence of fungal elements was found in samples of *H. radicata* or pasture litter from pastures grazed by affected horses (Barry 1956; Huntington *et al.* 1989; El-Hage 2011). Screening of leaf, flower and stem samples from *H. radicata* collected from paddocks in which horses had developed PSH using gas chromatography-mass spectrometry (GC-MS) did not reveal any evidence of fungal alkaloids including those known to be associated with neurotoxicities (El-Hage 2011).

In contrast to the many negative or inconclusive results, dose-dependent increases in urine, liver and brain concentrations of scyllo-inositol were measured in mice fed a freeze dried extract of *H. radicata* flowers although, they did not develop gait abnormalities (Domange *et al.* 2008). Scyllo-inositol is a bio-marker found in increased concentrations in several neurodegenerative diseases and supports the presence of a neurotoxic entity produced by *H. radicata*. Three guaianolides have been identified in the roots and aerial parts of *H. radicata* (Bohlmann and Bohlmann 1980). The guaianolides belong to the sesquiterpene lactone class of chemical compounds with well-known cytotoxic properties, but their role, if any, in PSH is unknown. In a recent pilot study utilising GC-MS, numerous differences in the metabolic profiles were identified between *H. radicata* samples collected from pastures that had contained horses with PSH and samples collected from pastures with no known cases of PSH. The significance of those differences remains to be determined (El-Hage 2011).

There is now evidence to suggest that *H. radicata* produces toxic metabolites only following chemical or climate induced stress (Bohlmann and Bohlmann 1980; Maruta *et al.* 1995; El-Hage

2011; MacKay *et al.* 2013). Metabolic profiles, again determined by GC-MS, differed between plants deprived of water (to simulate drought conditions) and those receiving adequate water, but it is not yet known whether these differences are related to disease in grazing horses (El-Hage 2011). Leaves of *H. radicata* exposed to the stressor copper chloride secreted fungitoxic sesquiterpenes and alkenals (Maruta *et al.* 1995). Recently, dose-dependent neurotoxicity of an extract from aerial parts of *H. radicata* plants subjected to chemical (copper chloride) stress was documented in cultured murine nerve cells (MacKay *et al.* 2013). The observed degenerative changes resembled those following exposure to repin, a sesquiterpene lactone, that is believed to be the toxic agent of yellow star thistle (*Centaurea solstitialis*) and the cause of nigropallidal encephalomalacia in horses (Roy *et al.* 1995; MacKay *et al.* 2013).

Treatment and Management Strategies for Horses with Pasture associated Stringhalt

Numerous therapies have been suggested for the treatment of PSH, and, while some of these have been described in the peer reviewed literature, none have been assessed in large-scale blinded or controlled studies (Dixon and Stewart 1969; Huntington *et al.* 1991; Kannegieter and Malik 1992; Wijnberg *et al.* 2009; Domange *et al.* 2010; El-Hage 2011). The reported success of any treatment for PSH must, of course, be considered in the context that the vast majority of affected horses recover spontaneously following removal from the putative source of toxin. Most horses recover over a period of 6 to 18 months, but mildly affected horses (Grades I-II) can recover more quickly. It should be noted that recovery can be extremely prolonged requiring over 2 years for complete resolution in some severe cases. Severely affected horses might be euthanised if they become recumbent for prolonged periods. A very small number of horses never appear to recover completely, perhaps as a result of severe muscle wastage or fibrosis (Cahill *et al.* 1985; Huntington *et al.* 1989; Domange *et al.* 2010).

Myotectomy of the Lateral Digital Extensor (LDE): Surgery for Stringhalt has been reported for over 300 years (Markham 1644); whilst myotectomy has been advocated for Classical Stringhalt, efficacy of the procedure in the management of PSH has been a point of some contention (Cahill *et al.* 1985; Huntington *et al.* 1989; Crabill *et al.* 1994; Sullins 2002; Domange *et al.* 2010; Dyson and Ross 2011). Some horses with PSH have been reported to improve by several grades immediately following LDE myotectomy; although, others only recovered after a prolonged convalescence (Cahill *et al.* 1985; Huntington *et al.* 1989; Domange *et al.* 2010). This has created some doubt regarding the value of the procedure in light of the spontaneous recovery of most horses (Cahill *et*

al. 1985; Huntington *et al.* 1989), however, one report has suggested that the technique can be successful in many (11/13) horses with PSH (Torre 2005).

Botulinum toxin infiltration: Botulinum toxin type A (Botox A) is a potent neurotoxin that has been used to manage a number of hypertonic or spastic muscular conditions in humans (Pathak *et al.* 2006). Horses are considered exquisitely sensitive to the effects of Botox A and a single report of its use in two horses presenting with Classical Stringhalt provided some encouraging results. Following infiltration of 100 IU four times over 21 days into either the lateral or long digital extensor muscles and 300 IU into the vastus lateralis at day 28, improvements in electromyographic and clinical parameters were observed over 2 to 3 months (Wijnberg *et al.* 2009). Anecdotal reports of prolonged clinical improvement following Botox A administration provide some basis for further investigation of its use in horses affected with Stringhalt.

Muscle Relaxants: Mephenesin, a centrally acting muscle relaxant, produced rapid reduction of the hypertonia in a single horse with PSH following systemic (intravenous or intramuscular) administration of mephenesin but this effect was transient (Dixon and Stewart 1969). The authors proposed that mephenesin might modulate the response of CNS centres and neural tracts that might be responsible for the hypertonicity in PSH. Because of its short duration of action and potentially serious side effects in human patients, mephenesin now has limited availability and has been largely replaced by methocarbamol. Although anecdotally used to treat PSH, the efficacy of methocarbamol in PSH has not been reported.

Baclofen, an analogue of the inhibitory neurotransmitter gamma amino butyric acid (GABA), is used as a skeletal muscle relaxant primarily to treat spastic muscular conditions in humans (Standaert and Roberson 2010). Baclofen has been trialled in horses with PSH on the basis that it might down-regulate hyperactive stretch reflexes (Pedersen *et al.* 1974). Administration of baclofen (1 mg/kg orally q 8 h) to 10 horses that had had signs of PSH for between 4 months and 4 years resulted in improvement in clinical signs in 8 of the 10 horses. However, clinical signs regressed slightly or returned to pre-treatment levels in 7 of those 8 cases once treatment was withdrawn (Kannegieter and Malik 1992). A later report indicated no changes in a horse with PSH following a 9-day course of baclofen starting at 7.1 mg/kg orally q 12 h and increasing to 10 mg/kg orally q 8 h for the last 7 days of treatment (Takahashi *et al.* 2002).

Phenytoin has perhaps been the drug most often administered to horses with PSH and, at least anecdotally, appears to be the most consistently effective. Phenytoin is a sodium channel blocker that has been suggested to modulate tetanic muscle contractions and aberrant nerve activity (Woodbury 1982). Oral administration of phenytoin at dosages of 15 mg/kg q 12 to 24 h to

horses with PSH generally result in a degree of clinical improvement while the drug is administered. Additionally, EMG recordings from the long digital extensor muscle indicate a reduction in abnormal spontaneous electrical activity in horses receiving phenytoin (Huntington *et al.* 1991; Takahashi *et al.* 2002). It has been suggested that the mild tranquillisation that often accompanies phenytoin administration might be responsible for amelioration of clinical signs and, for most horses, clinical deterioration occurs after cessation of treatment (Takahashi *et al.* 2002; Gardner *et al.* 2005; Domange *et al.* 2010). Variations in pharmacokinetics have been reported following both oral and intra-venous administration and might explain the lack of efficacy in some horses (Soma *et al.* 2001). Given this variability, monitoring plasma concentrations is recommended particularly for long term administration (Beech *et al.* 1988; Huntington *et al.* 1991; Soma *et al.* 2001). However, aside from mild sedation few if any adverse effects have been noted in horses administered phenytoin for extended periods (Takahashi *et al.* 2002; Domange *et al.* 2010). It is of course important to recognise that the administration of phenytoin (and other drugs) might be regulated in competitive animals.

Miscellaneous treatments: Thiamine (vitamin B1) is an essential co-factor in neuronal cell metabolism, neurotransmitter production and myelin synthesis (Puri 2014). It has been commonly used in the treatment of human peripheral neuropathies (Ang *et al.* 2008). Although there is little evidence to support any benefits of administration in humans, thiamine has been used with anecdotal reports of success in the treatment of PSH (Huntington *et al.* 1989; Domange *et al.* 2010). Taurine is a sulphur-containing compound with metabolic activity in many tissues including the mammalian nervous system where it appears to modulate neuronal excitability (Birdsall 1998). In a small number of horses, taurine (10 g orally q 24 h) appeared to have a quite dramatic and repeatable calming effect in horses with behavioural changes thought to be related to PSH (Domange *et al.* 2010). Anti-oxidants including tocopherol (Vitamin E), ascorbic acid (Vitamin C) and dimethyl sulfoxide (DMSO) have been administered to horses with PSH presumably to reduce oxidative injury to distal axons of the long nerves (Armengou *et al.* 2010). Other supplements recommended for the treatment of PSH include L- tryptophan, magnesium, and potassium bromide for purported neuromodulatory effects. Mycotoxin binders have been promoted for both the prevention and treatment of PSH. There is little theoretical basis for many of these supplements and no scientific evidence to support the use of any of them.

Conclusions and Future Directions

Despite continuing advances in diagnostic and analytical technologies, there are still many gaps in our understanding of PSH. The techniques used in some of the earlier landmark studies may have been insufficient to detect the pathologic lesions that would explain the clinical manifestation of PSH. The recent finding of cerebellar changes in horses with Shivers suggests that the application of newer diagnostic techniques might also bear fruit in horses with PSH. The use of non-invasive technologies such as magnetic motor evoked potentials to assess the function of descending motor tracts may provide further insight into horses with PSH. Similarly, magnetic resonance imaging techniques may identify peripheral nervous system dysfunction. This modality is a well reported PNS imaging modality in humans. These potentially valuable techniques have not been commonly used in cases of PSH to date and may aid both diagnosis and assessment of disease severity. Targeted scrutiny of the neuromuscular relays (including muscle spindles) suggested to be involved in the pathogenesis of the gait abnormality of PSH might also be rewarding. Definitive identification of a causative toxin and an understanding of the exposure required to cause disease would be a major advance in the management of grazing horses in high risk pastures. The *in vitro* and laboratory animal studies evaluating extracts from (stressed) *H. radicata* appear promising and might provide sensible targets for a preventative strategy. However, until a definitive lesion and/or toxic agent are identified, recommendations for the prevention and management of PSH must remain empirical. Pasture-associated stringhalt is well known to occur in certain geographical regions and might even be restricted to specific paddocks. Reducing known risk factors by improving poorer pastures with fertilisers and/or introduced grasses such that *H. radicata* and other weeds are out-competed would seem logical. Supplementary feeding to reduce reliance on grazing might be a useful strategy if horses cannot be moved to another paddock. Once the clinical signs of PSH develop, the affected horse almost certainly has a distal axonopathy of the longer nerves. A prolonged recovery period is required, although nearly all affected horses will eventually recover once removed from the offending pasture.

Authors' declaration of interests

No conflicts of interest have been declared.

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Authorship

Each author contributed substantially to the preparation of this review manuscript.

Table 1: Grading scheme for categorisation of Stringhalt affected horses adapted from Huntington *et al.* (1989) and Domange *et al.* (2010).

GRADE	DESCRIPTION
I	Only noticeable when horse was backed, turned or stressed.
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V	Hindlimb held hyperflexed for prolonged periods when initiating movement which is characterised by plunging, leaping, hopping motion.
VI	Prolonged recumbency following grade IV-V stringhalt gait

Figure legends

Fig 1: Horse with clinical signs of Pasture associated Stringhalt displaying exaggerated hyperflexion of the hindlimb.

Fig 2: Masson's trichrome stained digital extensor muscle x100 from the hindlimb of a horse with longstanding Grade III-IV Pasture-associated Stringhalt. Normal muscle bundles are on the left with muscle bundles that have undergone extensive atrophy on the right. The two regions of this muscle are separated by a dense band of connective tissue. Affected muscle appears hypercellular, and

individual muscle cells are small, angular and irregular in outline, and show marked fibre size variation. Bar indicates 100 μ m.

Fig 3: *Hypochoeris radicata* (left) present in a paddock with horses displaying signs of Pasture-associated Stringhalt. Note lack of improved pasture species. On the right is an uprooted specimen highlighting the length and nature of the tap roots. Note branched stems and rough haired leaves differentiating the plant from the common dandelion (*Taraxicum officinale*). Image courtesy of Dr Xenia Newland.

Fig 4: Diagrammatic representation of the myotatic reflex arc involving the neuromuscular spindle. Note (larger diameter) 1-a afferent fibres from spindle to CNS relays. The [alpha motor] descending tracts can initiate extrafusal muscles to voluntarily contract. However other tracts, via gamma motor axons, set the contraction of intrafusal muscle fibres. Changes in tension on annulospiral endings in the neuromuscular spindle indirectly change tone and timing of contraction of the main striated muscles. This could then heighten muscle tone and initiate early, prolonged contraction. Reproduced with permission from I.G. (Joe) Mayhew, Large Animal Neurology, Wiley Blackwell Publishing.

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586 **Supplementary Information**

587

588 Supplementary Video S1: Horse with Grade II-III Pasture-associated stringhalt (courtesy Dr Soodi
589 Ilkhani)

590

591 Supplementary Video S2: Horse with Grade V Pasture-associated stringhalt (courtesy Dr Jemma
592 Bergfeld)

593

594 Supplementary Video S3: Stringhalt-like gait induced by application of travel wraps to hindlimbs
595 limbs (courtesy Miss Amy Waldron)

596

597 Supplementary Video S4: Horse with Classical (unilateral) Stringhalt following a proximal metatarsal
598 injury (courtesy Dr Kayo Kawaguchi)

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Author/s:

El-Hage, CM;Huntington, PJ;Mayhew, IG;Slocombe, RF;Tennent-Brown, BS

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