

Review

Possible Mechanisms of Acupuncture for the Treatment of Chronic Inflammatory Disorders of the Equine Lower Airway

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ABSTRACT

Inflammatory airway disease, recurrent airway obstruction and summer pasture associated obstructive pulmonary disease are chronic inflammatory disorders of the equine lower airway. They are characterized by chronic bronchitis, an increase in respiratory resistance and an increase in the number of neutrophils in the bronchoalveolar lavage fluid. Besides the conventional treatment with bronchodilators and anti-inflammatory agents, acupuncture (AC) and electro-acupuncture (EA) can provide a safe and effective alternative therapy that possesses little or no negative side effects. Possible therapeutic mechanisms of AC and EA for chronic inflammatory disorders of the equine lower airway are proposed based on research in other species. These therapeutic mechanisms include an increase in the mucociliary action of the airway epithelium, reduction in airway and pulmonary tissue inflammation, activation of cholinergic anti-inflammatory responses, alteration of the immune response, modulation of the autonomic nervous system and alteration in the peripheral sensory input from the inflamed pulmonary tissue.

Key words: Acupuncture, electro-acupuncture, veterinary, RAO, recurrent airway obstruction, horses, lower airway inflammatory disease.

Respiratory disease is one of the most common health problems in veterinary medicine.¹ It affects animals at all ages, all breeds, and all physiological states. Severity of a respiratory disease depends on several factors including the age of the animal, immunological status and the cause of the disease. Performance animals such as horses and greyhound dogs that are being kept for recreational sports are unable to participate in their routine training program during sickness. A decrease in their maximal performance capability is commonly seen in severe cases of respiratory disease due to residual damage. Opportunities to develop other pulmonary disease-associated complications such as pleuropneumonia, pleuritis, emphysema and fibrosis are increased if an

appropriate treatment is delayed. Irreversible changes in the histological structure and severe compromise of the normal respiratory physiological function may lead to the termination of an animal's athletic career or, in severe cases, euthanasia.

Cardiopulmonary fitness is a major factor contributing to equine athletic performance. Diseases and abnormalities that are capable of compromising normal airflow may originate in the upper or lower airways.¹ Disorders of the upper airway that increase airway resistance and limit the normal air flow include recurrent laryngeal hemiplegia, pharyngeal lymphoid hyperplasia, dorsal displacement of the soft palate, nasopharyngeal collapse, subepiglottic cyst and entrapment of the epiglottis. Inflammatory airway disease (IAD), heaves or recurrent airway obstruction (RAO) and summer pasture associated obstructive pulmonary disease (SPAOPD) are the most common diseases that lead to an increase in the resistance of the lower airway.

Treatments of IAD, RAO, and SPAOPD

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are complex due to an incomplete understanding of the causes of these diseases and their pathogenesis. Conventional therapy requires both long-term medication and an adjustment of the stable environment to remove the likely inciting cause.² Significant improvement in clinical signs may be seen in some cases after making changes in the environment. Bronchodilator, mucolytic and anti-inflammatory agents alone or all together are normally prescribed in order to control the clinical signs of dyspnea and cough. Improvement in the clinical signs does not guarantee a complete resolution of the disease. Horses with a history of previous lower airway inflammatory disease are more likely to experience a recurrence of their clinical signs when the inciting factors are re-introduced. Failure or delay in a proper treatment may lead to chronic irreversible changes in the histopathological structures of the small airway and pulmonary parenchyma.^{1,2} Increase in airway resistance and decrease in pulmonary compliance and gas exchange capacity are common consequences of these changes.

Besides the medical managements mentioned, alternative treatments such as acupuncture and electro-acupuncture (AC/EA) and Chinese herbs have been used as adjunctive therapies. They are intended to decrease the dosage requirements of bronchodilators and anti-inflammatory agents and improve the quality of life of animals suffering from chronic respiratory diseases.³ In the United States, an integration of AC and EA into the conventional veterinary practice is relatively new as compared to China, and it was not widely practiced until 1974 when the International Veterinary Acupuncture Society was founded.⁴ Research on laboratory animal models have suggested that AC/EA possess therapeutic benefits for respiratory problems.⁵

AC/EA treatment of respiratory diseases in veterinary medicine requires stimulation of multiple acupoints. The points selected in the treatment strategy are intended to replenish the normal lung function, reduce cough, alleviate the clinical signs of heaves and dyspnea and to improve the animal's immune function. Selected acupoints can be stimulated by a dry acupuncture needle or with an electrical current applied to the inserted acupuncture needle. Studies of EA analgesia have shown that 15 to 30 minutes of electrical stimulation produced short-term improvement in

pain when determined by the visual analog scale, while the hypoalgesic effects after 30 minutes of stimulation were similar to those that occurred after 45 minutes of stimulation.⁶ Most of the studies also indicated that a low frequency (range from 2-30 Hz) of electrical stimulation produced greater analgesia than that of the frequency above 100Hz.⁷⁻⁹ The low frequency stimulation has been shown to cause release in β -endorphin, enkephalin, and met-enkephalin.¹⁰ These endogenous opioid substances were suggested to play an important role in AC/EA therapeutic mechanisms. Further study also demonstrated that high frequency stimulation induced a release of dynorphin, another type of endogenous opioid substance, which potentially synergistically interacts with other endogenous opioid substances released by the low frequency electrical stimulation to produce greater therapeutic benefits.¹¹

According to the "Acupuncture and Electroacupuncture: Evidence-based Treatment Guidelines" published by the Council of Acupuncture and Oriental Medicine Associates, typical duration of each EA application is 15 to 30 minutes.¹² The duration may be increased up to 45 minutes. Stimulation amplitude should be adjusted only to the level where the patient feels a slight sensation or the acupuncturist observes a small movement of the needles or slight muscular contraction. Suitable treatment frequency for chronic respiratory conditions in humans is 2-3 times per week for 4 weeks during the initial course of the treatment. The treatment can be reduced to one to two treatments per week thereafter for another six to eight weeks.¹² It should be kept in mind that it is unnecessary to electrically stimulate all the selected acupoints. The number of acupoints capable of being stimulated by electricity at one time depends on the number of leads of the electrical stimulator (electroacupunctoscope). Most electroacupunctoscopes can not stimulate more than 12 to 14 acupoints simultaneously. Frequently used acupoints and their functions are listed in Table 1.^{13,14} Chinese herbal medicines are often combined with AC/EA when treating chronic respiratory disorders.¹⁵

In IAD, RAO, and SPAOPD, the affected animals develop clinical signs of respiratory discomfort due to a reduction in airway ventilation, impairment of the gas exchange capacity, increase

Table 1: Acupoints commonly used to treat chronic respiratory diseases of horses

Acupoint	Location	Attributes and Indications
BL-13	At the caudal edge of the scapular cartilage (8 th intercostal space), 3 cun from the dorsal midline	Lung Back <i>Shu</i> Association point to strengthen the Lung
BL-23	At the 2 nd Lumbar intercostal space (L2-L3), 3 cun from the dorsal midline	Kidney Back <i>Shu</i> Association point to strengthen the Kidney
CV-17	On the ventral midline at the level of the 4 th intercostal space (caudal border of the elbow)	Cough, dyspnea
CV-22	On the ventral midline in the depression just cranial to the manubrium of the sternum	Cough, dyspnea
LU-9	On the medial side of carpus at the junction of the radius and the first row of the carpal bone, at the level of accessory carpal bone.	Chronic cough, asthma, and heaves
<i>Ding-chuan</i>	0.5 cun lateral to the midline at the level of GV14 (on the dorsal midline between C7-T1)	Chronic lung problems, cough, asthma, and dyspnea
<i>Fei-pan</i>	On the caudal edge of the scapula, 1/3 from the upper border	Lower airway problems, heaves and cough
<i>Fei-men</i>	On the cranial edge of scapula, 1/3 from the upper border	Upper airway disease
LI-4	At the level of upper one third of the cannon bone in a depression between the 2 nd and 3 rd metacarpal bone.	Immune deficiency
LI-11	In a depression cranial to the elbow (lateral aspect), in the transverse cubital crease, cranial to the lateral epiconyle of the humerus	Immune deficiency
ST-36	3 cun distal to ST-35 and 0.5 cun lateral to the cranial aspect of the tibial crest over the cranial tibial muscle	General weakness
GV-14	Dorsal midline between the dorsal spinous processes C7-T1.	Cough, heaves, and immune modulation

in the production and accumulation of airway secretion and increase in the inflammatory reaction of the lower airway.² Alleviation of any one or more of these disease mechanisms may reduce the severity of the clinical signs. AC treatment in humans suffering from asthma has been shown to improve the pulmonary function parameters and significantly improved the quality of life of the patients.^{16,17} However, a more scientific explanation on how AC works in treating respiratory diseases is still needed. From current scientific research on AC/EA in animal models, several different mechanisms can be proposed for how AC/EA

alleviates respiratory diseases.

Improved Mucociliary Action of the Airway Epithelium

An increase in mucus production is a common consequence of airway inflammation. In normal circumstances, mucus is immediately removed by epithelial mucociliary action. Disruption of the normal mucociliary clearance is a common consequence of chronic airway inflammation and is thought to be caused by neutrophil-derived elastase.¹⁸ Besides alteration of

a normal mucociliary function, neutrophil-derived elastase also induces goblet cell metaplasia and mucin production in response to antigens. The increase of mucus and its accumulation decrease airway diameter and increase total airway resistance. Until recently no scientific evidence had directly demonstrated the effect of AC or EA on mammalian mucociliary clearance. However, Tai et al. demonstrated that EA at acupoints LU-1 and CV-22 significantly increased the rate of tracheal mucociliary transport in quails when compared to a control group.¹⁹ Moreover, EA at these acupoints significantly reversed the decrease in mucociliary transport caused by the administration of human neutrophil-derived elastase.

Several mediators have been shown to regulate the rate of mucociliary clearance. These included endogenous nitric oxide (NO) and substance-P.^{20,21} However, whether these substances are involved in AC/EA-mediated mucociliary clearance needs further investigation.

Reduced Airway and Pulmonary Tissue Inflammation

Analgesic and anti-inflammatory effects of AC/EA are well recognized in both somatic and visceral tissues.^{22,23} Major acupoints that are indicated for these purposes include LI-4, LI-10 and ST-36.^{22,24,25} Mechanisms associated with these analgesic and anti-inflammatory effects have been linked to a release of endogenous opioid substances in the central nervous system (CNS) during AC/EA treatment.²⁶ These endogenous substances activate opioid receptors in the CNS tissues, such as the substantia gelatinosa of the spinal cord and the periaqueductal grey of the midbrain and produce analgesia.²⁷ This endogenous opioid-dependent anti-nociception has been shown to be modulated via the mu-opioid receptor.²⁷

Immunocytes such as macrophages, monocytes, and polymorphonuclear cells possess opioid receptors on their cell surfaces and the mu-opioid receptors predominate.²⁸ Once activated, the receptor induces an anti-inflammatory response via the down regulation of the transcription factor, nuclear factor kappa-B (NF- κ B).²⁹ The NF- κ B down regulation has been demonstrated to reduce the mRNA expression of other inflammatory cytokines including TNF- α , IL-1 β , IL-6, nitric oxide synthase (iNOS), and metalloproteinase.³⁰ Carneiro et al. demonstrated that EA reduced the

inflammatory cell infiltration in the peribronchial tissue and in the pulmonary perivascular spaces in rats with ovalbumin-induced bronchial asthma.⁵ Moreover, the number of total nucleated cells and the percentages of neutrophilic and eosinophilic leukocytes in bronchoalveolar fluid (BALf) were significantly decreased when compared to control and sham EA groups. In this study, the EA was performed on acupoints GV-14, BL-13, LU-1, CV-17, ST-36, SP-6, and *Ding-chuan* (0.5 cun lateral to GV-14) which mimicked the acupoints used to treat human asthma.

Inflammatory cell infiltration into pulmonary tissues is governed by inflammatory cytokines released by vascular endothelial cells, tissue resident macrophages and local immunocytes. The AC treatment in the study of Caneiro et al. reduced pulmonary tissue inflammation and improved BALf cytology.⁵ These results directed the researchers to hypothesize that the anti-inflammatory action of EA in this experiment is partly associated with down regulation of the eosinophil and neutrophil chemotactic cytokines IL-5 and IL-13 respectively.

Cholinergic Anti-Inflammatory Effects

The vagus nerve is a major parasympathetic nerve that contains afferent and efferent nerve fibers for both somatic and visceral tissues.^{31,32} Acetylcholine is a major neurotransmitter in both preganglionic and postganglionic parasympathetic nerve synapses. The cholinergic influence on immunological functions has been demonstrated in laboratory animals. Direct electrical stimulation of the vagus nerve attenuated the in-vivo release of TNF- α and prevents lipopolysaccharide-induced endotoxic shock. Moreover, acetylcholine attenuated the in-vitro release of IL-1 β , IL-6, and IL-18, but not the anti-inflammatory cytokine, IL-10 in lipopolysaccharide-stimulated human macrophage culture.³³ This cholinergic dependent anti-inflammatory pathway suppresses the non-specific, innate immune response and may explain how AC/EA works in treating other diseases affecting visceral organs.

Tian et al studied the anti-inflammatory benefit of EA at ST-36 in rats with induced colitis.³⁴ He reported a significant decrease in circulating TNF- α and a down regulation of its mRNA expression in inflamed colonic tissue. The

results from this study are important because in Traditional Chinese Veterinary Medicine, ST-36 is one of the most commonly used acupoints for treating several deficiency ailments including respiratory diseases. It is a *He-sea* point, which can be used as a general *Qi* tonic and for Lung *Qi* Deficiency.¹³

Increase in the parasympathetic tone after AC/EA stimulation at selected acupoints has been demonstrated. Examples include a decrease in heart rate, an increase in gastrointestinal myoelectric activity and an increase in gastric motility.³⁵⁻³⁷ In one study, EA at ST-36 reversed a stress-induced delay in gastric emptying and alleviated the stress-induced acceleration of the colonic transit time.³⁸

The direct effect of AC/EA on the cholinergic-associated anti-inflammatory response in visceral organs has never been demonstrated; however, the effect of EA on the cholinergic anti-inflammatory response of somatic tissue has been shown in rats with collagen-induced arthritis.²² In this model, EA at ST-36 showed significant analgesic and anti-inflammatory properties. These analgesic and anti-inflammatory activities of EA were inhibited by the administration of a muscarinic cholinergic receptor antagonist. This suggests that EA at ST-36 may activate a local cholinergic anti-inflammatory mechanism to prevent the inflammatory reaction and thus to reduce the painful sensation. It is also possible that endogenous opioids and cholinergic pathways work together to create the anti-inflammatory action associated with AC/EA when treating internal medical problems like lower airway disease.

Alteration of Immune Responses

When treating chronic respiratory diseases with AC/EA in clinical practice, acupoints that benefit the immune system are routinely stimulated along with the acupoints that have specific indications for respiratory problems. This treatment strategy is mainly based on the knowledge in conventional medicine that allergic airway inflammatory diseases are the result of an imbalance of Th1 and Th2 associated immunological responses.³⁹ Acupoints that are normally used for this purpose include LI-4, LI-11, ST-36 and GV-14.⁴⁰⁻⁴³

Studies of AC/EA performed at these acupoints have shown to modulate cellular immunity, secretory functions of inflammatory

cells, and humoral immunity. Multiple AC treatments at LI-11, GV-14, ST-36 and SP-10 in healthy human volunteers reduced circulating leukocytes and lymphocytes without affecting the level of circulating corticosteroid.⁴³ This study suggested that alterations of the hematological parameters of interest are unlikely to be driven by the physiological stress response. Another study demonstrated that EA at LI-4 and LU-6 normalized the patterns of leukocytes in granulocytosis/lymphocytopenia and granulocytopenia/lymphocytosis.⁴⁴ Moreover, EA at ST-36 corrected the splenic NK cell activity suppression induced by a lesion at the anterior hypothalamus.⁴² The mechanism by which AC/EA normalizes the population of circulating leukocytes has not been proposed.

Besides the effect on circulating leukocytes, AC/EA at ST-36 has been shown to increase phagocytic activity of rat peritoneal macrophages and attenuate TNF- α release *in-vitro*.⁴⁵ Moreover, an *in-vivo* attenuation of TNF- α release has been demonstrated in experimentally induced ulcerative colitis in rats.³⁴ In this study, EA at ST-36 not only decreased the level of circulating TNF- α , but also down regulated the colonic tissue TNF- α mRNA expression. The suppression of activated macrophage TNF- α production by EA could be partly antagonized by naloxone, the opioid antagonist.⁴⁶ This would suggest that the opioid receptors are partly involved in the mechanism of EA suppression of the macrophage immune response. In another study, EA at LI-11 significantly increased the respiratory burst of peripheral-circulating neutrophils. Neutrophils possess a highly phagocytic activity and are capable of eliminating pathogens by producing reactive oxygen species.⁴¹ This innate immunological activity is an important primary immune response in both peripheral circulation and local tissues.

EA at ST-25 and CV-6 significantly reduced IL-1 β , IL-6, and TNF- α secretion by monocytes during experimentally induced colitis in rats and alleviated the decrease in apoptosis rate of peripheral circulating neutrophils.⁴⁷ Prolonged apoptosis of neutrophils is thought to be a result of the presence of pro-inflammatory-triggering substances. Alleviation in the suppression of neutrophil apoptosis may be due to the reduction of inflammation of local tissue and a decrease in the

production of inflammatory cytokines. EA may possess an activity similar to that of direct electrical stimulation of the vagus nerve, thereby possibly also stimulating the cholinergic anti-inflammatory pathway, inhibiting macrophage activation, and decreasing the production of TNF- α , IL-1 β , IL-6, and IL-18.⁴⁸ A down regulation of TNF- α released by AC/EA also has been demonstrated in other experimental models.^{34,46,49}

The humoral immunity effects of AC for human asthma were shown to be a reduction of the levels of circulating IgM and IgE and an increase in the level of IgG without a change in the level of IgA.⁵⁰ Successive EA at ST-36 in DNP-KLH-immunized mice significantly reduced total IgE and the antigen specific IgE to DNP-KLH.⁵¹ Production of TH2 specific cytokines, including IL-4 and IL-13, was also significantly reduced. This suggested that EA at ST-36 suppresses the immune response to the injected antigen by modulation of the TH2 cytokines and inhibition of IgE production. EA at LI-4, ST-6, ST-36 and LU-6 inhibited a decrease of secretory IgA and increased the cortisol in saliva of human athletes in training.⁵² Moreover, salivary and nasal IgA and serum IgE of asthmatic patients are significantly decreased after AC treatment.⁵³

Modulation of the Autonomic Nervous System

Equine airway smooth muscle and lymphoid tissue are innervated by both sympathetic and parasympathetic nervous systems.⁵⁴ Alterations of the activity in both systems has been demonstrated following AC treatment.⁵⁵ It has been proposed that the spinal cord and the vagus nerve are essential to relay AC/EA sensory signals to higher relay centers such as the brain stem and the hypothalamus.⁵⁶ All the afferent signals converge at these relay centers and send a signal to a cortical area in order to generate a physiological response.

Bradycardia following AC/EA has been commonly seen in both clinical practice and laboratory experiments demonstrating the parasympathomimetic and sympatholytic properties of AC/EA.^{44,57} EA at ST-36 decreases the excitability of the cardiovascular system manifested by bradycardia, which may be associated with sympathetic inhibition and modification of the central baroreflex arch.⁵⁸ Its modulation of the sympathetic response also has been reported in a study of AC performed at PC-6.⁵⁹ Chan et al. reported that AC can be used to treat post-traumatic

sympathetic dystrophy in humans with a 70% improvement in the clinical signs.⁶⁰ In another study, EA at LI-11 and LI-4 produced moderate hypoalgesia in humans paralleled by a significant increase in muscle sympathetic nerve activity.⁶¹ Based on the observation of the c-Fos expression in neurons, the location of the CNS tissue that is activated by AC/EA performed at particular acupoints can be observed.⁶² AC at ST-36 increased gastric motility and the number of c-Fos immunopositive cells at the mediocaudal and caudal nucleus tractus solitarius (NTS) in rats. This finding suggested that somatic afferent signals activated by AC at ST-36 were conveyed to the medio-caudal and caudal NTS and stimulated the dorsal motor nucleus of the vagus.⁶² NTS is located in the brain stem and known as a primary relay center for visceral organs including respiratory, cardiovascular, and gastro-intestinal systems.⁶³ It is adjacent to the dorsal motor nucleus of the vagus, and it is a part of the dorsal vagal complex (DVC). These findings support the theory that AC at ST-36 increases gastric motility by activating the parasympathetic nervous system. AC at ST-25, on the other hand, reduces the gastric motility by activating the rostral ventrolateral medulla, which is known as a pre-motor sympatho-excitatory preganglionic neuron in the intermediolateral nucleus of the spinal cord.⁶²

Several studies indicate that AC/EA possesses an immediate mild to moderate bronchodilator effect. A clinical study of human asthma found that AC at LU-7, LI-4, PC-6, ST-40, LI-11 and PC-3 for 15 minutes induced bronchodilation manifested by an increase in forced expiratory volume in the first second (FEV1).¹⁶ Improvement in pulmonary function parameters (pleural pressure, tidal volume, minute ventilation, peak inspiratory flow and peak expiratory flow) in RAO-affected horses also has been demonstrated after a single AC treatment.⁶⁴ In this study, the authors concluded that the improvements were due to animal handling. Whether AC/EA induces immediate bronchodilation in obstructive lesions of the airway by modulation of autonomic nervous system is uncertain and needs further investigation.

Alteration in the Peripheral Sensory Input from Inflamed Pulmonary Tissue

Although the non-myelinated nerve fibers (C fibers) have never been described in the equine

airway, they can be identified in several animal species and it might be assumed that this type of nerve fiber is also present in equine species.^{65,66} It is a vagally mediated non-myelinated nerve fiber and serves as a polymodal receptor, which can be activated by tissue damage, edema and inflammatory mediators.⁶⁷ Irritation, created by inhaled particles or mediators released by airway resident inflammatory cells, activates this nerve fiber. Activation leads to airway hyper-responsiveness, a classical clinical manifestation of airway diseases.

Attenuation of nociceptive signal conduction by C fibers has been hypothesized as the main analgesic mechanism of AC/EA.⁶⁸ This is also known as the Gate Control Theory, proposed by Melzack and Wall. In this theory, sensory input from AC conducted by the A β myelinated nerve fiber reaches the spinal cord at a faster speed than does the nociceptive signal that travels through the non-myelinated nerve fiber. When the sensory signal reaches the substantia gelatinosa of the spinal cord, it stimulates the inhibitory interneuron and prevents subsequent neuronal conduction of the slow nociceptive signal transmitted by the non-myelinated C fiber and A δ fiber. Based on this theory, ascending non-nociceptive signals originating from AC/EA may modulate the nociceptive input from C fibers residing in the airway at the level of the spinal cord and alleviate the airway hyper-responsiveness.

Other mechanisms

AC/EA has been shown to induce the release of NO in several tissues.^{69,70} NO is an important biological signaling molecule. It is also known as the endothelium-derived relaxing factor (EDRF), which is capable of inducing vasodilation in the pulmonary vascular bed.⁷¹ It is mainly produced by the endothelium. Although it has a short half-life, it diffuses freely across the cell membrane and functions as a paracrine to communicate with the surrounding tissue such as the smooth muscles of the blood vessels. NO is capable of activating, and is produced by, macrophages, monocytes, neutrophils and NK cells as a part of the primary immune response.⁷²⁻⁷⁴ It possesses antimicrobial activity, relaxes the smooth muscle of the endothelium, and signals other inflammatory cascades.^{71,75}

Positive therapeutic effects following sham

AC/EA in human studies suggest a psychogenic influence that leads to a placebo effect.⁷⁶ Whether the same placebo effect is present in other animals is questionable, because animals do not know if the treatment is good for them (unless the investigator predictably rewards the animal following the treatment). Subjective parameters that require human assessment of an animal, such as appetite and degree of pain, are unavoidably affected by human bias. Also, it should be kept in mind that handling during the experiment potentially plays a major role in producing stress, which is frequently demonstrated by elevation of the endogenous corticosteroids.

Unlike in humans, a study of thoracic pain associated with respiratory discomfort and cough has never been published in animals suffering from IAD, RAO, and SPAOPD. However, thoracic pain is thought to be present in horses that are affected by chronic lower airway inflammatory diseases.⁷⁷ Immediate relief in respiratory discomfort following AC/EA can be a result of a non-specific nociceptive inhibition by a diffuse noxious inhibitory control mechanism (DNIC). In DNIC, noxious stimulation, including AC needle insertion, may evoke analgesia by a non-specific attenuation of the afferent painful stimulation.⁷⁸ Alleviation of respiratory discomfort following AC/EA at acupoints located around the thoracic region, including BL-13, *Fei-men* (1/3 the way along the cranial border of the scapula from dorsal to ventral), *Fei-pan* (1/3 the way along the caudal border of the scapula from dorsal to ventral) and *Ding-chuan* (0.5 cun lateral to GV-14), may be the result of this mechanism (Table 1).

Conclusion

In summary, it seems certain from experimental studies in several species that AC/EA provides benefits as a treatment for chronic lower airway diseases, but more investigation of mechanisms specific to horses is needed. Ideally, future studies should involve natural occurring cases of equine respiratory disease. Based on laboratory animal and human studies, it seems that AC/EA may treat the chronic lower airway diseases via modulation of the nervous and the immune systems. Among all of the proposed mechanisms, the cholinergic anti-inflammatory effects seem the most logical. The cholinergic anti-inflammatory system receives sensory input from both somatic

and visceral tissues and is capable of generating a physiological response at the effector tissues. However, which pathway the body uses to generate the physiological response from AC/EA stimulation also needs further investigation.

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TCVM NOTES

Trigger Points

Trigger points are discrete, hyperirritable foci usually located within a taut band of skeletal muscle. They are composed of a well-circumscribed area in which pressure produces intense local pain and potentially a characteristic referred pain. Digital pressure produces muscle twitching and fasciculation and usually there is restriction of motion or function of the affected area. The pathogenesis is unclear but there are many theories. One thought is that they are due to the sensitization of nerves and the pain results from a decrease in the pain threshold. Another thought is that they arise from acute trauma or repetitive micro-trauma to the muscles.

Trigger points are categorized as active or latent. Active trigger points are a specific area with spontaneous pain or pain in response to movement or palpation. They are associated with an acute onset of pain or lameness and are more common in animals that undergo physical exertion. Pain can be felt with movement and/or at rest. Referred pain, which is pain felt at a distant site when the trigger point is compressed, can also be present. Affected muscles are weakened in strength and have restricted movement. Latent trigger points cause pain only in response to compression of the trigger point. They develop over time and can persist for months or years. They can become activated by over-stretching, overuse, excess cold, nutritional deficiencies or stress.

Trigger points cause pain and stress in the muscles or muscle fibers. As the stress increases, the muscles become fatigued and more susceptible to activation of additional trigger points. They weaken the muscles, prevent full lengthening of the muscles and eventually lead to dysfunction. They are identified by deep palpation or inferred by inhibition of either active or passive range of motion and feel firmer than surrounding tissues. Treatment is based around the release of the taut band of skeletal muscle.

In TCVM, trigger points are called the “a-shi” points. *Sun Si-miao* of *Tang* Dynasty (618-907 A.D.) stated “Needle wherever there is tenderness”. Trigger point stimulation causes localized trauma-induced vasodilation and segmental release of endogenous opioids. Dry needle, electro-acupuncture, aqua-acupuncture and *Tui-na* can provide relief of trigger point sensitivity. For treatment, first locate the trigger point, insert the acupuncture needle (or hypodermic needle) into the muscular knot and gently probe until you produce a localized involuntary twitching of the muscle. This reaction has the effect of fatiguing the tight muscle and producing an immediate reduction or elimination of the tightness. Trigger points should feel less firm and less painful upon palpation with subsequent treatments.

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