

## Review on gastric ulcer in horses

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**Abstract:** Equine Gastric Ulcer Syndrome (EGUS) describes a unique set of conditions that are manifested as erosions and ulcerations in the distal esophagus, non glandular and glandular stomach, and proximal duodenum of horses. There is no established etiology of gastric ulcer in horses although association with risk factors such as stress/disease, treatment (iatrogenic factors), exercise, management and environmental factors, dietary factors and factors associated with withholding of feeds are indicated. The pathogenesis gastric ulcers are uncertain but exposure of squamous mucosa to the acid is probably involved in the development of ulcers in most horses. Management has an important impact on the treatment of gastric ulcer in horses. Horses with gastric ulcers experience a spontaneous healing when removed from the training and kept at the pasture. Prevention of gastric ulcer disease in athletic horses centers up minimizing factors that promote ulcer development. Many studies have been done in different parts of the world in search of knowledge of equine gastric ulceration syndrome emphasis being given to the risk factors, pathogenesis, and clinical presentation of the case, diagnosis, treatment and control and prevention measures that can be applied in order to alleviate the problem.

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### 1. Introduction

Equine Gastric Ulcer Syndrome (EGUS) describes a unique set of conditions that are manifested as erosions and ulcerations in the distal esophagus, non-glandular and glandular stomach, and proximal duodenum of horses. It is common in race horses, and in the study by Orsini *et al.* (1997) 82% (571/798) of standard breed and thorough breed race horses had gastric ulcers. Horses with moderate to severe ulcers are more likely to suffer subtle clinical signs such as poor body condition, rough hair-coat, interrupted and/or slow (picky) eating, and low-grade colic, especially after eating a grain meal. These signs are subtle and nonspecific, which may not be recognized by the owners and trainers. The presence of gastric ulcers has also been correlated with poor racing performance in horses (Vatistas *et al.*, 1999)

The cause of EGUS is likely due to secreted and/or organic acids in gastric juice and the resultant low stomach PH. Lorenzo and Merritt (2002) recently showed that intra-gastric pressure sharply increased at a trot and gallop due to increased intra-abdominal pressure related to the exercise. They hypothesized that this increase pushed gastric contents into the proximal region of the stomach, exposing the non glandular squamous mucosa to acidic inorganic and organic acids. This may explain the high prevalence of

gastric ulcers in the race horses in the study by Orsini *et al.* (1997).

The findings by Lorenzo and Merritt (2002) are consistent with observations that the lesions regress or disappear when a horse is taken out of training or treated with an acid-suppressing drug. The corrosive potential of the gastric content might also be influenced by the diet and feeding schedule to which race horses are subjected (Lorenzo and Merritt, 2002).

The mainstay of EGUS treatment involves inhibition of gastric acid and raising intra-gastric pH above 4.0 for a 24-hour period. The various medications used in the race horses includes buffers (anti-acids), sucralfate, compounded omeprazole, and proprietary omeprazole paste (Gastrogard, Merial) at 2 and 4 mg/kg body weight daily (Frank and Andrews, 2004).

Therefore the objectives of this paper are:

- To describe epidemiology, pathogenesis and risk factors of gastric ulcer.
- To highlight treatment, control and prevention of gastric ulcer in horses.

## 2. A Review Ofgastric Ulcer In Horses

### 2.1 Etiology

There is no established etiology of gastric ulcer in horses although association with stress is indicated. In adults the disease is common in horses undertaking

regular exercise and might be related to decreased stomach volume and subsequent exposure of the squamous mucosa of the proximal parts of the stomach to acid during exercise (Radostitis *et al.*, 2007). Individual cases of gastric ulcers are associated with parasitic gastritis such as in horses infested with gastrophilus species and *Habronema megastoma* larvae. Tumor of the stomach such as gastric squamous cell carcinoma or lymphosarcoma may cause ulceration of the gastric mucosa. Gastric phyto-bezoars and persimmon seeds (*Diospyros virginiana*) have been associated with gastric impaction and ulceration of glandular portion of stomach of the horse (Cummings *et al.*, 2001).

Many infectious diseases with major systemic or alimentary tract signs are able to produce gastric lesions. Systemic states such as uremia and endotoxemia, blister beetle (*Epicauta species*) intoxication induced by the cantharid contained in these insects may cause necrosis and ulceration of distal esophagus, pars esophagi and intense hyperemia of glandular epithelium with 88% of mucosa of the stomach being affected (Jubbs, 2005). Once gastric ulcers present other bacteria have been implicated in inhibiting ulcer healing. Bacteria including *E.coli* were cultured from stomach of horses (Al Jassim *et al.*, 2006).

## 2.2. Epidemiology

### 2.2.1. Prevalence

Erosion and ulceration of gastric mucosa (equine gastric ulcer syndrome – EGUS) is a common finding in race horses. According to various authors, prevalence of the disease varies from 55% to 100% (Vatistas *et al.*, 1999; McClure *et al.*, 1999). According to Murray *et al.* (1999) EGUS is a very common disease of stomach of foals and adult horses with reported prevalence of up to 50% and 75% respectively. This percentage does heavily depend on habitat, breed performance and age of the population. The prevalence of erosion ulcer of gastric glandular and non glandular mucosa detected by gastroscopic examination reaches about 50% in foals less than 2 months of age that do not have signs of gastric ulcer disease. Lesions of squamous mucosa are present in 45% of foals where as lesions in the glandular mucosa occur in less than 10% of foals less than 4 months age. A disease attributable to the duodenal and gastric ulcer occurs in approximately 0.5% of foals although the prevalence is greater in foals with other diseases, such as pneumonia and septicemia (Radostits *et al.*, 2007). Despite wide spread use of anti ulcer drugs the prevalence of EGUS remains high. This is due to cost of antiulcer agents which results in shorter than prescribed treatment course, administration of sub therapeutic doses or substitution of compounded

medications or feed supplements that are ineffective according to Orsini *et al.* (2003).

The prevalence of gastric ulceration in competitive horses varies from 11% in riding horses to 93% in race horses (Sandin *et al.*, 2000). Also the prevalence of horses' gastric ulceration is high in horses with bowel, liver and esophageal lesions. Prevalence of up to 93% in thoroughbred race horses and severity of equine gastric ulceration syndrome has been associated with the type of training and different management practices (Lutherson *et al.*, 2009).

## 2.3. Risk factors

There are different risk factors associated with the occurrence of gastric ulceration in horses and these include factors associated with animals, treatment (iatrogenic factors), exercise, management and environmental factors, stress/disease, dietary factors and factors associated with withholding of feed. While each of the risk factors can be considered separately it is likely that many are related and act in concert to increase the risk of development of lesions (Radostits *et al.*, 2007).

### 2.3.1. Animal risk factors

Major animal risk factors include age and sex. Age has different degree of effect on the occurrence of gastric ulcer both in adults and foals. It is an important risk factor for the ulceration of squamous epithelium with 88% of foals less than 9 days of age compared to the 30% of foals more than 70 days of age developing the problem. Among adult horses age and sex are weak risk factors. Gastric lesions tend to be more severing in adults and females are more susceptible than males (Rabuffo *et al.*, 2002).

### 2.3.2. Exercise risk factors

There is a definite association between equine training and gastric ulceration. Even non-intensive training is associated with a high prevalence of stomach ulcer. It has been shown that blood flow to the stomach (which helps to remove acid) decreases with exercise - while increased pressure in the abdomen during exercise pushes acid up into the sensitive portion of the stomach (Lorenzo and Merit, 2002).

Intense exercise is common risk factor for equine gastric ulceration. This is associated with release of acid contents to the proximal region of stomach following the compression of the stomach by the abdominal viscera and diaphragm. This results to injury due to exposure to the acid (Radostits *et al.*, 2007).

### 2.3.3. Iatrogenic risk factors

Some long-term medications can produce adverse gastric effects, as they may inhibit production of the substances that help to protect the stomach. Treatments with NSAIDS have been shown to cause

gastric ulceration in horses ([http://www.lochlevnequine.co.uk/gastric\\_ulcers.pdf](http://www.lochlevnequine.co.uk/gastric_ulcers.pdf).)

According to Garris and Kirkwood (1997) when prostaglandin synthesis is inhibited erosion and ulceration of gastric mucosa may occur. This is apparently the mechanism by which aspirin and other NSAIDS cause acute gastric ulcer. The association of the use of NSAIDS and the occurrence of gastric ulcers was indicated by Andrews *et al.* (2006). According to this study phenylbutazone 2.2mg/kg bwt po every 12hr for 5 days or 1.1mg/kg bwt every 12hr for 5 days were administered. Later it was found that plasma protein and albumin were found decreased and non glandular ulcer score was significantly higher in horses treated (Andrews *et al.* 2006).

#### 2.3.4. Stress/disease

According to Radostits *et al.* (2007) stress and disease are important factors for the development of ulcers of glandular mucosa. Lesions of glandular mucosa occur in 27% of foals with other disease but in 3% of healthy foals. Gastric ulcers can occur in response to physiological stress. Shock, respiratory disease and traumatic injury may play a role. Equine transportation and stable confinement are proven risk factors in causing ulcers (MacClure *et al.*, 2005). While psychological stress is difficult to evaluate in horses and foals, stressful conditions may adversely influence feed intake, resulting in periods of increased stomach acidity (Furr *et al.*, 2004).

Transporting horses has been implicated as a risk factor for EGUS. Transportation of horses has been associated with dehydration, increased threat of respiratory illness (pleuritis, pleura pneumonia), and immune suppression (Watson, 2002). During transport, water and feed consumption is usually decreased which may cause an increased incidence of EGUS. Transportation has been shown to increase the severity of gastric ulcers in horses (MacAllister and Sangiah, 1993).

#### 2.3.5. Management and environmental risk factors

Race horses in training have a higher prevalence of ulcers than do spelling (not in active training) horses and horses that are racing regularly have a higher prevalence of gastric ulcer than horses that are resting or in training but not racing (Radostits *et al.*, 2007). Although many factors can contribute the development of likelihood of a horse having gastric lesions. Management has great role in occurrence of gastric ulcer and for treatment of it (Murray, 1999).

#### 2.3.6. Dietary risk factors

As horses are 'trickle feeders' there is a continuous secretion of acid within the stomach, so prolonged periods without food to neutralize that acid can lead to ulceration. When horses are denied free access to feed or fail to eat, ulcers develop rapidly

(Murray and Eichorn, 1998). Use of concentrated feeds may also contribute to ulcer risk by reducing the time spent feeding and increasing gastrin levels (Smyth *et al.*, 1997). High roughage diet causes prolonged chewing and production of salivary bicarbonate that bathes the stomach and protects against gastric ulcers (Radostits *et al.*, 2007).

On the other hand little and starch meals emptied from the stomach significantly faster than larger and high starch diets, which are fermented to the VFAS, and lactic acid causing gastric ulceration and should then be avoided from horses meal. According to Nadeau *et al.* (2000) alfalfa was shown to protect horses against EGUS by increasing the stomach PH. This was confirmed by that gastric juice PH and ulcer scores were lower in horses fed a diet containing alfalfa hay compared to the same horse fed dietary feed brame or costal Bermuda (Nadeau *et al.* 2000).

#### 2.3.7. Feed withholding/starvation

Feed withholding causes gastric ulcers in horses probably because of buffering of acid produced during periods when the stomach is empty. This is due to the absence of the feed material in the stomach and a decreased production of saliva which has a buffering effect in the gastric acids. It is likely that intermittent access to the feed that occurs in many stables results in period of time during each day when horses do not have feed in the stomach. Horses grazing at pasture eat frequently and have food in their stomach all times (Radostits *et al.*, 2007).

### 2.4. Pathogenesis

The lining of the equine stomach is divided into an upper squamous mucosa (non glandular) and the lower glandular mucosa. The squamous mucosa does not absorb or secrete anything, but has the primary role of aiding the mechanical breakdown of ingested foods. The glandular mucosa, on the other hand, produces hydrochloric acid (HCl), pepsin, lipase, gastrin, histamine, and several other physiologic mediators (Flemstrom and Isenberg, 2001).

The glandular mucosa of the stomach has a complex mechanism for protecting itself from its own corrosive agents. The essence of this barrier is a combination of mucus and bicarbonate that are produced by cells of the gastric glands, and through which secreted acid and pepsinogen can easily move out in to gastric lumen, but not vice versa (Flemstrom and Isenberg, 2001).

Thus, under normal conditions, the pH of the area just adjacent to the glandular mucosal surface is biologically neutral. Equine stomach is comparatively small relative to the size of the gastrointestinal tract and is divided into two parts. These are the proventricular or non glandular part and glandular part both separated by the margo-plicatus, a slightly raised irregular serrated border (Radostits *et al.*, 2007).

Stomach churns and mixes the feed with the digestive juice, secretes enzymes and mucus that digest the protein and secretes intrinsic factors which are necessary for the absorption of vitamin B<sub>12</sub> from the intestine. Few highly lipid soluble substances are also absorbed in a moderate quantities (Abrams, 2003).

The secretion of enzymes is highly controlled by the parasympathetic nervous system, presence of food in the mouth, and by the smelling or looking of the feed. Once produced gastric acid is released by the activation of any enzyme system like H<sup>+</sup>-k<sup>+</sup>ase at the surface of parietal cells. This enzyme system acts as a gastric acid /proton pump to remove gastric acid from parietal cells in the mucosal lining of the stomach into the stomach lumen (Murray *et al.*, 1996).

Auto digestion of the stomach wall and ulcer formation are normally prevented by the cell protective effects of the mucus secretion, dilution effects of the food, prevention of back diffusion of HCL from the stomach lumen back to the gastric mucosal lining. The presences of some prostaglandins also have some importance, therefore gastric ulceration is considered to result from an imbalance between aggressive factors (HCL, pepsin) and protective factors (mucus, bicarbonate barrier, PGF<sub>2</sub>, mucosal blood flow, cellular restitution and growth factors that promote angiogenesis). Gastric emptying is also important, because delayed gastric emptying and prolonged gastric emptying have been implicated in the pathogenesis (Murray, 1999).

Most gastric lesions in horses occur in the gastric mucosa of the non glandular part of the stomach. This none glandular part is glistening white in color and is composed of stratified squamous epithelium and glandular part is covered by the thick layer of viscous mucus. Glandular mucosa contains mucus secreting fundic glands, HCL producing parietal cells and

pepsinogen secreting chief cells (Radostits *et al.*, 2007).

The glandular mucosa elaborates mechanisms including the mucus bicarbonate barrier, prostaglandins, mucus blood flow and cellular restitution to protect itself from peptic injury. The horse is continuous variable HCL secretor and the pH of equine gastric contents in the pylorus and antrum is often less than 2.0. The administration of the H<sub>2</sub> antagonists like ranitidine during feed deprivation reduces the area of lesion in the gastric epithelial mucosa (Murray and Eichorn, 1998). The pathogenesis gastric ulcers are uncertain although exposure of squamous mucosa to the acid is probably involved in the development of ulcers in most horses. During exercise intra-gastric pressure increases from approximately from 14mmHg at rest to as high as 50mmHg. Stomach volume decreases and acidity of the fluid within the proximal part of the stomach declines from 5-7 to 2-4 (Lorenzo and Merrit, 2002).

The combination of reduced blood flow and exposure to the low PH increases the likelihood mucosal damage, loss of protective mechanisms and development of gastric mucosal lesions. Other factors including physical injury to the gastric mucosa reflux of bile acids from the duodenum and the presence of volatile lesions fatty acids in the stomach may all contribute to the development of gastric ulcer (Nadeau *et al.*, 2000).

## 2.5. Clinical findings

The vast majority of horses with gastric mucosal lesion including ulceration do not have clinical signs. Among rare horses signs of poor physical performance, feed refusal, and fussy eating (feed consumption at an abnormal rate) have been associated with the presence of gastric ulcers (Dionne *et al.*, 1999).

**Table 1:** Clinical signs and risk factors of Equine Gastric Ulcer Syndrome

Clinical signs	Foals	Risk factors
Acute colic	Diarrhea	Stress
Recurrent colic	Abdominal pain	Transportation
Excessive recumbency	Restlessness	High grain diet
poor body condition	Rolling	Stall confinement
Partial anorexia	Lying in dorsal recumbency	Intermittent feeding
Poor appetite	Excessive salivation	Intense exercise
Poor performance/training	Bruxism	Racing
Attitude changes	Intermittent nursing	Illness
Stretching often to urinat	Poor appetite	NSAIDS
Inadequat exercise		Management change

**Source:** Orsini *et al.* (1997).

Gastric outflow obstruction due to pyloric or duodenal stricture secondary to pyloric or duodenal ulceration is also seen in 2-5 months old foals (Traub-Dagartz *et al.*, 2001). Typical signs of gastric ulcer in foals include depression, teeth grinding, excessive salivation and abdominal pain ranging in intensity from very mild to acute and severe (Becht and Byars, 2003).

### 2.6. Diagnosis

The approach to diagnosis of EGUS requires a thorough history, physical examination, and gastroscopic examination. Identifying risk factors and clinical signs are helpful in diagnosing EGUS (Table 1). However, gastroscopy is the only definitive diagnosis of EGUS currently available. Gastroscopic examination is the only means of assessing their extent and severity. Endoscope of 2.5-3m long for adults and 1-2m long for foals is used. The horse should be withheld off food for at least 12 hrs and water at least for 4hrs. Because of the presence of the food material within the stomach prevents the complete examination of stomach mucosa. Material adherent to the mucosa should be washed away flushing water through the endoscope. Endoscopic examination under estimates the number of lesions as compared to the necropsy examination and doesn't accurately predict the severity and depth of lesions (Anderws *et al.*, 2006).

Naso-gastric intubation, even though it causes pain and gagging in foals, may be used in case gastric outflow obstruction is suspected. Reflux of the material through the tube confirms the diagnosis. Contrast radiographic examination may demonstrate filling defects in the gastric wall that is consistent with ulcers. The principal use of radiography is establishing delays in gastric emptying which may be delayed from the normal of 10-20 ml/kg bwt of barium sulphate per 2 hours of administration in foals (Radostitis *et al.*, 2007).

Therefore, the combination of compatible clinical signs, endoscopic demonstration and favorable response to the treatment after elimination of other diseases permits confirmatory diagnosis of gastric ulcer disease. In adult horses there is no impaired gastric outflow as in foals (Baker, 2004).

### 2.7. Clinical pathology

There are no specific laboratory tests for gastric ulceration in horses. But in diseased animals there is a higher concentration of creatinine and alkaline phosphate in the serum (Vatistas *et al.*, 1999). Horses with gastric disease are not typically anemic. Sucrose is absorbed intact across damaged gastric mucosa and excreted in the urine (Hewetson *et al.*, 2006). In foals there are no diagnostic changes in the serum biochemical profile or haemogram and serum

pepsinogen levels are of diagnostic use (Wilson, 2000).

### 2.8. Necropsy findings

Ulcers which may be single or multiple and are located in the squamous epithelial mucosa adjacent to the margoplicatus along the lesser curvature of the stomach are seen. They may be linear or irregular in shape and circular in appearance. Ulcers in the glandular zone are less common and usually circular or oval depressions surrounded by the intense zone of inflammation. When perforation has occurred there is an area of local peritonitis, adherence of the stomach the tip of the spleen and supportive splenitis. If the stomach is full at the time of perforation large quantities of ingesta will spill in to the peritoneal cavity (Radostits *et al.*, 2007).

### 2.9. Treatment

The promotion of healing by reducing gastric acidity and enhancing mucosal protection, enhancement of gastric emptying and provision of nutritional and metabolic support and treatment of other diseases are principles of treatment of gastro duodenal ulcers in foals (Radostits *et al.*, 2007). The goals of clinical treatment are to eliminate clinical signs, promote healing of ulcers and prevent recurrence. Currently, pharmaceutical treatment of gastric ulcers relies upon the use of H<sub>2</sub> receptor antagonist drugs (i.e. ranitidine) and proton pump inhibitors (i.e. omeprazole). In addition to pharmacological therapy, dietary and environmental modification has also been shown to assist healing; particularly sufficient roughage in the diet which aids to absorb gastric secretions so does not contact the susceptible mucosal lining. (Buchanan and Andrews, 2003).

Cimetidine increases gastric pH by binding with histamine to prevent its binding to the H<sub>2</sub> receptor, ranitidine decreases HCl by binding at the H<sub>2</sub> receptor, and sucralfate creates a protein bandage of mucus over the ulceration. Sucralfate can be helpful in controlling the severity of glandular gastric ulcer disease, with rationale for its use in its putative ability to induce mucosal protection by activating PGF<sub>2</sub> synthesis. However, clinical trials of its efficacy in treatment have not been very convincing (Murray, 1999).

NSAIDS such as phenyl butazone or flunixinemeglumine are ulcerogenic and should not be used in the treatment of foals with the gastroduodenal ulcers unless absolutely necessary (Carrick *et al.*, 1995). Treatment can be achieved by different methods (i.e. acid suppression, use of gastric antacids, use of protectants and applying management changes (Rdostits *et al.*, 2007).

Gastric antacids given orally neutralize stomach acid to form water and neutral salt. They are not

absorbed and reduce pepsin activity binding to the bile salts in the stomach and stimulate local prostaglandin. The short duration of action, minimal and transient action on the PH and the need for the administration of volumes doses orally renders these products less optimal. Examples of such compounds are aluminum hydroxide and magnesium hydroxide (Vatistas *et al.*, 1999).

Protectant compounds sucral fat are antiulcer drugs with cytoprotective effect on the gastric mucosa. This is due to sucrose octasulfate that polymerizes to a viscous sticky substance that creates a protective effect by binding to the ulcerated mucosa. It is administered at 22 mg/kg BWt orally every 8hour (Orsini *et al.*, 1997). Management has also an important impact on the treatment of gastric ulcer in horses. Horses with gastric ulcers experience a spontaneous healing when removed from the training and kept at the pasture. Using feeding practices that minimize or eliminate periods when horses do not have access to the feed, example using hay constantly, is managmental aspects for the treatment of gastric ulcers (Radostits *et al.*, 2007).

Another dietary strategy is feeding alfalfa hay. Because of its superior buffering capacity, alfalfa protects the squamous mucosa from acid damage and has been shown to decrease gastric ulcers in horses. Horses fed alfalfa hay and exercised showed decreased gastric ulcer scores compared to horses fed a coastal hay diet. (Lybert *et al.*, 2007).

### 2.10. Control and prevention

Prevention of gastric ulcer disease in athletic horses centers up minimizing factors that promote ulcer development. This may involve chronic administration of omeprazole at adose rate of 1 mg/kg Bwt orally once daily (MacClure *et al.*, 2005). But this should include an attention to the dietary and feeding practices that minimize the time that horses have no feed in their stomach. All horses in the athletic training and confined to the stalls should be considered at a high risk of development of gastric ulcer and should be managed accordingly. Horses at pasture such as, brood mares are at minimal risk of development of gastric ulcer disease and no specific control measures are indicated (Radostits *et al.*, 2007).

Horses could develop gastric ulcer in as little as 8 days after initiation of heavy to light training and omeprazol past administration reduces the incidence. Management changes are essential to prevent gastric ulcers after treatment is discontinued (Nadeau *et al.*, 2000).

Since large amounts of VFAs are produced in the stomach of horse fed on high concentrate diets it has been suggested that concentrates should be at less than 0.5kg/100kg BWt and horses with EGUS should be fed concentrates with caution (Lybbert *et al.*, 2007).

### 3. Conclusion And Raecommendation

Many studies have been done in different parts of the world in search of knowledge of equine gastric ulceration syndrome emphasis being given to the risk factors, pathogenesis, and clinical presentation of the case, diagnosis, treatment and control and prevention measures that can be applied in order to alleviate the problem. Over the recent decades the studies used to induce this disease experimentally have mainly involved exposing to risk factors of equine gastric ulceration. Nonetheless this work has facilitated significant progress in our understanding of the disease and improved option of disease control. The generation of new infection protocols including an ability to expose animals to the controlled and potentially low risk factors of equine gastric ulceration will allow further understanding of the occurrence of the disease and allow greater progress to combat the ongoing threat posed by equine gastric ulceration. Therefore, by taking the foregoing facts the following points are recommended.

- ✓ Strict hygienic practices should be practiced in the farm to reduce contamination from feces, urine and other contaminants in order to cut the cycle of gastric ulceration.

- ✓ Newborn foals should be kept in a good management that should not have stress and avoid administration of NSAIDS.

- ✓ Race horses should never be held off feed.

- ✓ Race horses having race and hard work should take omeprazole or antacids to prevent auto-digestion of stomach wall by HCl and pepsin.

- ✓ Further researches should be conducted on etiology, risk factors, and pathogenesis of the problem in naturally susceptible animals in order to understand the establishment and progress of the disease for designing effective control measure.

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