TOXIC PLANTS AND MYCOTOXINS AFFECTING CATTLE AND SHEEP IN URUGUAY

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Summary

Plant poisonings and mycotoxicosis affecting cattle and sheep in Uruguay include the intoxications by *Phalaris spp., Cynodon dactylon, Paspalum spp, Solanum bonariense, Senecio spp., Echium plantagineum, Cestrum parqui, Wedelia glauca, Myoporum laetum, Ammi majus, Festuca arundinacea, Solanun malacoxylon, Nierembergia repens, Baccharis coridifolia, Cichorium intybus,* and *Amaranthus spp*. Bloat caused by *Trifolium* spp., nitrite intoxication caused different grasses and cyanide poisoning caused by sorghum are also frequent. Mycotoxicosis caused by *Ramaria flavo-brunnescens, Fusarium solani* (*Ipomoea batata*)*, Claviceps purpurea* and *Pithomyces chartarum* are reported. This paper reviews some less known plant intoxications observed between 1980 and 1999 in Uruguay, including the intoxications by *Xanthium cavanillesii, Cycas revoluta, Lantana camara, Erichtites hieracifolia, Nerium oleander, Halimuim brasiliense, Cynodon dactylon, Anagallis arvensis, Quercus spp, and Nierembergia hippomanica.* Chronic copper phytogen intoxication caused by *Trifolium repens and Trifolium pratense* and intoxication by *Ramaria flavo-brunnescens* in sheep are also reported.

Plant intoxications and mycotoxicosis in ruminants in Uruguay

Hepatotoxic plants and mycotoxins

Plants causing hepatic necrosis. *C. parqui, Xanthium cavanillesii, Wedelia glauca, Cycas revoluta.*

Plants causing hepatic fibrosis. *Senecio spp., Echium plantagineum, Erichtites hieracifolia*.

Plant and mycotoxins causing hepatogenous photosensitization. *Myoporum laetum, Lantana camara, Pithomyces chartarum*

Plants causing primary photosenstization.

Ammi majus.

Plants affecting the heart
Nerium oleander.
Plants and mycotoxins causing neurological disorders
Solanum bonariense, Paspalum notatum, Paspalum dilatatum, Phalaris spp., Halimium brasiliense, Cynodon dactylon, Ramaria flavo-brunnescens (in sheep).

Plants causing nephrosis
Amaranthus spp., Anagallis arvensis, Quercus spp.

Plants affecting the digestive tract
Baccharis coridifolia, Nierembergia hippomanica, Chicorium intybus, Trifolium repens, Trifolium pratense, Medicago sativa.

Cianogenic plants
Sorghum spp.

Plants causing systemic calcification
Solanum malacoxylon, Nierembergia repens

Plants with oestrogenic activity.
Trifolium pratense

Mycotoxins affecting the respiratory system
Fusarium solani toxins (*Ipomoea batata*).

Plants causing nitrate/nitrite intoxication
Lolium multiflorum, Triticum aestivum, Avena sativa, Trifolium repens, Trifolium repens, Lotus corniculatus.

Plants causing chronic phitogen copper intoxication
Trifolium repens, Trifolium pratense.

Mycotoxicosis causing Ergotism
Festuca arundinacea, Claviceps purpurea

Mycotoxicosis affecting diverse systems
Ramaria flavo-brunnescens

Some plant intoxications observed between 1980 and 1999 in Uruguay

Intoxication by *Xanthium cavanillesii* in cattle

Outbreaks of this intoxication had been observed in Rio Grande do Sul and Uruguay during spring (September and October). It occurs in the borders of rivers

or creeks, in sandy soils, after floods. One or two weeks after the withdrawal of the water there is a massive germination of the plant and the animals can eat enough amounts of the newly germinated seedlings in their cotyledonary stage to became intoxicated. Mortality varies between 3% and 82% (Mendez *et al*, 1997).Clinical signs, observed a few hours after the ingestion of the plant, are characterized by depression, muscle fasciculation, increased respiratory and cardiac frequencies, opisthotonous, sternal or lateral recumbence and terminal paddling movements. The animals die after clinical manifestation periods of 12-24 hours. On necropsies, the liver is swollen and dark reddish, and the wall of the gall bladder is edematous. The cavities have yellowish fluid. Petechiae and ecchymosis are seen on serous membranes. Dry feces with blood or mucus are frequently observed in the rectum. Microscopically, the liver has an hemorrhagic centrilobular necrosis. Necrosis frequently extends to the periportal hepatocytes (Mendez *et al*, 1997).The intoxication was produced experimentally in calves dosed with 7.5-10g per kg of body weight (g/kg bw) of cotyledons (Mendez *et al*, 1997).

Intoxication by *Cycas revoluta* in cattle

An outbreak of acute intoxication by *Cycas revoluta* was observed in Uruguay in September 1995 (Riet Correa et al, 1996). Two bulls had signs of aggressiveness, incoordination and diarrhea, 7-10 days after been introduced in an area where *C. revoluta* had been cultivated as an ornamental plant. At necropsies the liver was swollen, dark reddish and mottled. The gall bladder wall, the mesentery, and the abomasum wall were edematous. Hemorrhages were observed in the digestive tract. Microscopically there was a centrilobular liver necrosis. Hepatocytes of the midzonal and periportal regions were vacuolated. The disease was produced in a calf given 20g/kg bw of green leaves of *C. revoluta* collected in the area where the outbreak was observed. Clinical signs and lesions were similar to those observed in natural cases.

Intoxication by *Lantana camara* in cattle and sheep

Two outbreaks of intoxication by *Lantana camara* in sheep and one in cattle were observed in Northwestern Uruguay. Mortalities of 87% and 33% were observed in two flocks of 200 and 600 sheep, respectively. The outbreaks occurred after the transportation of the flocks to parks where *L. camara* had been cultivated as an ornamental plant. Sheep stayed in the paddocks for 24 hours. Many animals showed clinical signs after been removed from the area. Another outbreak affected two cows introduced in a park where the plant was also present. Clinical signs in sheep were characterized by severe photodermatitis affecting mainly the face and ears, anorexia, restlessness, jaundice, brown urine, weight loss, ruminal stasis, drooling of saliva, lacrimation, and occasionally keratitis. Serum GGT, and AST were increased. Some sheep died 24-48 hours after the onset of signs, but in most animals the clinical manifestation period varies from 5

to 20 days. Jaundice, subcutaneous yellow edema and swollen ochre coloured liver with distended and edematous gall bladder were observed at necropsies. Microscopically, the liver had severe vacuolation of periportal hepatocytes and mild proliferation of bile duct cells. A mild tubular nephrosis was also observed. The outbreak observed in cattle affected 2 cows that died after being sick for 24-48 hours. Clinical signs and lesions were similar to those observed in sheep. The green plant was administered experimentally, in unique doses, to cattle and sheep at 25-40 g/kg bw. Clinical signs were similar to those observed in field cases. The cattle that received 25g/kg bw died after 7 days after the administration of the green plant. The two sheep died 24-36 hours after the administration of a single dose of 40g/kg bw of leaves and flowers (Riet Correa et al, 1996).

Intoxication by *Erechtites hieracifolia* in cattle

The intoxication by *Erechtites hieracifolia* was observed in Eastern Uruguay in March 1993, in a herd of 120 one year old Aberdeen Angus cattle (Riet Correa et al, 1996). Eight animals were affected and died. Clinical signs were characterized by progressive weight loss, wasting, abdominal straining, protracted scouring and prolapse of the rectum. At necropsies there was excessive abdominal fluid, edema of the mesenterium and wall of the abomasum, and pale hard liver with enlarged and edematous gall bladder. Microscopic lesions of the liver were characterized by diffuse fibrosis, megalocytosis and proliferation of bile duct cells. The plant contained 0.2% pyrrolizidine alkaloids.

Intoxication by *Nerium oleander* in cattle

Nerium oleander is an ornamental plant found commonly in Uruguay. The toxicity of *N. oleander* results from several cardiac glycosides, mainly oleandrin. An outbreak of the intoxication was observed in Northwestern Uruguay in a paddock where an eucalyptus forest had been trimming and a plant of oleander was also cut. Eighty, 2 years old heifers, were introduced in the area and five of them died 24-48 hours after been in the paddock. Some animals were found dead. Others had clinical signs characterized by depression, weakness, anorexia, ataxia and diarrhea. No significant lesions were observed at necropsies. Oleander leaves were found in the rumen. The disease was produced in three calves given singles doses of 1g/kg bw, 0,5g/kg bw and 0,25 g/kg bw of leaves of *N. oleander* collected at the farm. The animals that received a single dose of 1 and 0,5g/kg bw died between 6 to 36 hours after the administration, with clinical signs of weakness, ataxia, anorexia, tachypnea, and severe tachycardia with arrhythmia. Post-morten findings were of little significance. The principal histologic lesions were in the heart and consisted of multifocal myocardial edema, degeneration and necrosis. The calf intoxicated with 0,25g/kg bw showed anorexia, weakness, bradycardia in the first 24 hours, becoming normally after 72 hours (Riet Correa et al, 1996).

Intoxication by *Halimium brasiliense* in sheep

Poisoning by *Halimium brasiliense* in sheep is characterized by transient seizures with muscular tremors, ventroflexion of the neck, opisthotonos, nystagmus, tetanic spasms and limb paddling movements. The intoxication has been observed in 2 farms in the municipality of Rio Grande in Rio Grande do Sul, Brazil, and in at least 36 farms in the departments of Lavalleja, Maldonado, Cerro Largo, Durazno and Treinta y Tres, in Uruguay. The illness is seasonal with most cases occurring from August to November, but few cases are also observed from May to July. Most sheep recovered when removed to other pastures. The frequency varies between farms and between years. There are also variation between different paddocks within farms. Morbidity varies between 1% and 15%, but some farmers reported a frequency of up to 50% in years when draught conditions prevailed. In farms where affected sheep are removed from the paddocks after the observation of the first clinical signs, mortality is between 1% and 5%. Nevertheless, in draught conditions, in some farms where this measure is not practised, it is as high as 35% (Riet-Correa *et al*, 1995). Macroscopic lesions are not significant. The main histologic lesion was the presence of vacuoles, sometimes containing macrophages or axonal residues, in the white matter of the brain and spinal cord. On electron microscopy the lesions were characterized by axonal degeneration followed by balloned myelin sheaths with disappearance of the axoplasm. It is probably that convulsions are secondarily inducing death of neurons which results in Wallerian degeneration or that the plant causes an axonal degeneration. A pigment identified as ceroid-lipofuscin is also present in neurons, astrocytes, Kuppfer cells and macrophages of the spleen and lymphonodes. This pigmentation was apparently non related with the clinical signs. Feeding trials in sheep demonstrated that the disease is caused by the ingestion of *Halimium brasiliense* in amounts ranging from 2100 to 3000g/kg bw (Riet-Correa *et al*, 1995).

Intoxication by *Cynodon dactylon* in cattle

Two outbreaks of intoxication by *Cynodon dactylon* (Bermuda grass) in cattle, were reported in Northwestern Uruguay in July l996 and August 1998. Both outbreaks occurred during winter time after heavy frost. One outbreak affected Hereford heifers, in a paddock covered by a dense and dry pasture composed mainly by *C. dactylon*. The other affected 2-3 years old Hereford steers grazing in a Eucalyptus forest with an abundant presence of the plant. Morbidity was 23,6% and 8,7 % and mortality 1% and 1,4 %. Clinical signs were muscle tremors and twitching, marked incoordination, weaving and bobbing of the head and inability to rise. Some animals appeared to be stiff legged, and others showed marked weakness of the hind limbs. Most of the dead animals died accidentally, as a result of the nervous disorder, mainly drowned in streams or ditches.

Intoxication by *Anagallis arvensis* in cattle and sheep

Ten outbreaks of intoxication by *A. arvensis* were diagnosed in the Department of Paysandú, Uruguay, during December, 1994, January, 1995, and December 1996 and 1997 (Rivero et al, 1998). Cattle morbidity varied between 3.2% and 53.2% and lethality between 42.6% and 100%. Sheep morbidity was 2.8% to 42.9% and lethality 81.3% to 100%. In 9 outbreaks the animals were grazing on wheat or barley stubbles. In 8 occasions the animals were introduced in the stubble 2-10 days before first clinical signs and in 1 outbreak 25 days before. In all outbreaks *A arvensis* was in the vegetative state and in bloom, covering the soil and with high predominance over other species. Flowers of red and blue colour were observed. Other nephrotoxic plants, as *Amaranthus* spp, or *Quercus* spp, were not observed. The remaining outbreak occurred on a paddock ploughed in winter but not cultivated, that remained without animals until December, when it was covered by a dense and green pasture basically composed of *A arvensis*. At the beginning of December, 1200 yearling sheep were introduced on this paddock and started to die 36 hours later. In 4 outbreaks where cattle and sheep had been grazing together, both species were equally affected. In one outbreak cattle were less affected because they remained in the paddock for no more than 36 hours; while sheep were kept in the pasture until the first clinical signs occurred. No differences in frequency were observed in animals of different age or sex. Two outbreaks occurred in the same paddock of the same farm in different years (Nov. 1994 and Dec. 1996). The farms were located on basaltic or cretacic soils. Clinical signs for both species were characterized by weakness, loss of body condition, ruminal atony, diarrhea (occasionally stained with blood), slow gait, staggers, convulsions in some animals, coma and death within 12 to 48 hours. Blood serum values of creatinine, urea and magnesium were increased, and levels of calcium were decreased. Gross lesions were characterized by a ventral subcutaneos edema and petechiae, submandibular edema, presence of fluids in cavities, and edema and petechial hemorrhages of the mesenterium. Kidneys were edematous, pale or yellowish in colour with petechiae on the cortex. Erosive and ulcerative lesions were observed in the esophagus. Edema of the abomasal submucous, and hemorrhagic abomasitis and enteritis were also observed. Cardiac and skeletal muscles were pale and flaccid. Some animals had congestion and edema of the lungs. The most significant histological lesion was a severe nephrosis, with tubular degeneration and necrosis, hyaline cylinders, moderate intratubular hemorrhages, and interstitial edema and congestion. Catharral enteritis with hemorrhages, focal necrosis and mononuclear infiltration of the mucosa and lamina propia with gland hyperplasia and increased secretion were observed in the gut. Liver congestion, and in some cases moderate hepatocitic granular degeneration and mild proliferation of Kupffer cells were also seen. *A. arvensis* in its vegetative period was collected in a farm where an outbreak was occurring in December 1996. Immediately the plant was carried to the laboratory and maintained at 4-5ºC until administration. Leaves, fruits, and fine stems were milled and administered through a stomach tube. The plant was administered to 2 sheep. The administration started 24 hours after the

plant collection. One ewe received a daily dose of 40 g/kg bw for 4 consecutive days. Another was dosed daily with 32 g/kg bw for 7 days. The 2 sheep that died as a consequence of the experimental intoxication were necropsied and examined histologically. One sheep showed depression, anorexia and weakness, and died on day 5, 12 hours after the onset of signs. The second sheep had weakness, anorexia, ruminal atony, diarrhea and staggers; it died on day 9, 36 hours after the onset of signs. Macroscopic and histologic lesions were similar to those observed in field cases. Prevention of intoxication should be based on avoiding grazing in areas severely infected by the weed during the season and under the conditions of this report. One reason for the presence of large amounts of the plant in certain areas could be the use of commercial seeds contaminated by seeds of *A arvensis*.

Intoxication by *Quercus spp*. in cattle

An outbreak of intoxication by *Quercus spp*. was observed in Eastern Uruguay, in May, 1997, in a herd of 90 one year old Hereford, Aberdeen Angus and crossbreed cattle. They were grazing in a forest of *Quercus* sp. with accumulation of acorns from the trees and dead forage. Morbidity was 16,6% and mortality 4,4%. Clinical signs were characterised by weakness, loss of body condition and dark diarrhea. Gross lesions were characterized by erosive and ulcerative lesions in the esophagus, oedema and petechial hemorrhages of the mesenterium, necrosis of buccal and rumen papilla. Kidneys were edematous, pale or yellowish in colour with petechiae on the cortex. The most significant histological lesion was a severe nephrosis with tubular necrosis, diffuse epithelial regeneration, hyaline cylinders, moderate intratubular hemorrhages, discrete fibrosis, and mononuclear infiltration.

Intoxication by *Nierembergia hippomanica* in cattle

Outbreaks of intoxication by *Nierembergia hippomanica* had been frequently diagnosed in cattle in Northwestern Uruguay. Morbidity is 10%-80% and deaths do not occur. Most outbreaks are observed in milking cows or in 3-4 years old steers. Younger cattle appear to be more resistant. The intoxication occurs at any time of the year from January to November. All outbreaks occurred in cultivated pastures or in wheat or barley stubble fields. Invasion of pastures by the plant is apparently due to the use of seeds contaminated by *N. hippomanica* seeds. Clinical signs are characterized by salivation, diarrhea, restlessness, abdominal pain and periodic motion of the head and limbs. Milking cows have a decreased milk production. Affected animals recovered within 1 week after the withdrawal from the pastures (Odini *et al*, 1995). The green plant was administered experimentally to cattle and sheep at 10-50g/kg bw. The lower toxic dose was 10-15g/kg bw. No differences were observed in the toxicity of plant samples collected in winter or spring. Clinical signs were similar to those observed in field cases. All animals recovered in 1-8 days, except one calf that died after the ingestion of 50g/kg bw. The main lesions were focal hemorrhages in

the large intestine and enteritis in the small intestine. The dried plant was not toxic to cattle and sheep. One steer that received 10 daily doses of 5g/kg bw showed clinical signs after the last dose, demonstrating an accumulative effect in the plant (Odini *et al*, 1995). Two sheep that received 20g/kg bw of the plant presented anorexia, diarrhea, abdominal pain, restlessness and excessive salivation (Odini *et al*, 1995). A previous description of the spontaneous intoxication in sheep reported nervous signs and deaths of some animals (Riet Alvariza, 1979). A pyrrole-3-carbamidine has been identified as the toxic principle of *N. hippomanica* (Buschi & Pomilio, 1987).

Chronic phytogen copper intoxication in sheep

This intoxication is associated with pastures containing normal levels of cooper, but considerable low levels of molybdenum. In Uruguay, it occurs in sheep grazing pastures of *Trifolium repens* and *Trifolium pratense.* From 1980 to 1985, 12 outbreaks were diagnosed in different regions of the Country. From 1983 to 1988, 25 outbreaks of the intoxication were diagnosed in Northwestern Uruguay. Twelve of these outbreaks occurred during 1988. The increase in the frequency of the intoxication was due to an increment on the ovine production in Uruguay, due to a good international wool price. By consequence, areas previously used for agriculture or cattle production, like the Northwestern region, were partially used for sheep breeding in *Trifolium repens* and/or *Trifolium pratense* pastures. After 1988 the outbreaks of chronic phytogen copper toxicosis decreased because, due to a drop in the wool price, it was not more profitable to graze sheep in cultivated pastures. From 1997 to 1999, the frequency of the disease in the Northwestern region increased again due to the use of mainly *T. pratense* pastures, for the production of fattening lamb for exportation. First cases are often seen after three months grazing in pastures of *Trifolium repens* and/or *Trifolium pratense,* mainly in animals in good nutritional state. The intoxication is observed all over the year but is more frequent in spring. The onset of the disease is commonly associated with stress factors like vaccination; insemination; dipping; transportation; and reduction in forage availability. Morbidity varies between 1% to 12% and lethality is nearly 100%. There was not variation in susceptibility between breeds (Corriedale, Ideal, Romney Marsh, Merilin and Merino), but most outbreaks occurred in Corriedale because this breed represents 70% of sheep population in Uruguay. The animals showed depression, anorexia, jaundice, hemoglobinuria, anaemia and liquid, fetid and dark feces. In most sheep the death occurred in 24-96 hours. Few sheep survived to the haemolytic crisis. At necropsies, the main gross lesions were jaundice; subcutaneous yellow edema; serous liquid in cavities; swollen friable ochre coloured liver with distended and edematous gall bladder; dark kidneys with edema and diminished consistency; and dark urine. Microscopically, the liver had enlarged pleomorphic and vacuolated hepatocytes, and, ocassionally, centrilobular necrosis, biliary stasis, mild proliferation of bile duct cells with fibrosis in the portal space, and proliferation of Kupffer cells with abundant cytoplasm and

granules containing copper. The most significant histologic lesion in kidneys was a severe hemoglobinuric nephrosis, with tubular degeneration and necrosis, with the presence of haemoglobin and cooper in the epithelial cells. Chronic phytogen copper intoxication occurs in pastures with low molibdenum, less than 0,36 ppm (Pereira & Rivero,1993), and normal cooper concentration in pastures of *Trifolium repens* and/or *Trifolium pratense.* The diagnosis is based on the epidemiological data, clinical signs, macroscopic and histologic lesions, and the determination of Cu levels in the liver (over 500ppm) and kidneys (over 80 ppm). For the prevention of the intoxication in Uruguay, grazing periods of no more than 3 months in pastures with predominance of *Trifolium repens* or *Trifolium pratense* are recomended.

Intoxication by *Ramaria flavo-brunnescen* in sheep

Several outbreaks by *Ramaria flavo-brunescens*, a well known disease in cattle in Uruguay and Rio Grande do Sur, Brazil, have been recently reported in sheep in Uruguay. The disease occurred mainly in the Northwestern region with a morbidity of 7% to 35% and a mortality of 7% to 26% (Riet Correa et al, 1996).The intoxication occurs between March and July, in eucalyptus forests where the fungus is found. Clinical signs in sheep are characterized by nervous disorders with convulsions, muscle tremors, ataxia, hypermetria, nystagmus and opisthotonos. Some animals remain in recumbence and died. Hypertemia, polyuria, ulcers in the tongue and necrotic lesions in the extremities characterized by a hyperhemic line with crusts at the coronary band were also observed in experimental intoxications.
The increase in the frequency of this intoxication in the last years in cattle and sheep in Uruguay, is due to the increase of the forestation area with eucalyptus. These new forests are also used for breeding livestock.

References:

Buschi C.A. and Pomilio A.B. (1987). Pyrrole-3-Carbamidine: a lethal principle from *Nierembergia hippomanica*. Phytochemistry 26, 863-865.

Méndez, M.C.; Santos, R.C. and Riet-Correa, F**.** (1997). Intoxication by *Xanthium cavanillesii* in cattle and sheep in southern Brazil. Vet. Hum. Toxicol. 40 (3): 144-147.

Odini, A.; Rivero, R.; Riet-Correa, F.; Mendez, M. C. and Giannechinni, E. (1995). Intoxicación por *Nierembergia hippomanica* en bovinos y ovinos. Veterinaria, Uruguay 30, 3-12.

Pereira, D.; Rivero, R**.**. 1993. "Intoxicação crónica fitógena por Cobre". In: Riet-Correa, F; Mendez, M.C.; Schild A. L. (ed). Intoxicações por Plantas e Micotoxicoses en animais domésticos. Brasil, Ed. Hemisferio Sul do Brasil. p. 299 - 307.-

Riet Alvariza F. (1979). Comunicación de un caso de intoxicación por *Nierembergia hippomanica.* Apuntes de toxicología veterinaria. Dirección General de Extensión Universitaria, Montevideo, pp. 165-166.

Riet-Correa, F.; Méndez, M.C. and Schild, A.L. (1993). Intoxicações por plantas e micotoxicoses em animais domésticos. Editorial Hemisferio Sur. Montevideo, 340p.

Riet-Correa, F.; Méndez, M.C.; Barros, C.S.L. and Gava, A. (1994). Poisonous plants of Rio Grande do Sul. In: SM Colegate & P. Dorling (ed.), Plant associated toxins. CAB International, Willingford, UK. p. 13-18.

Riet-Correa, F.; Mendez, M.C.; Pereira Neto, O.; Soares, M.P.; Vieira, M.A.; Silva, E.A.; Soares, M.P. (1995). Intoxicação por *Halimium brasiliense* em ovinos. Boletim do Laboratório Regional de Diagnóstico, Nº 15, pp. 32-37.

Riet Correa, F.; Rivero, R.; Dutra, F.; Mendez MC. 1996. "Intoxicaciones en Rumiantes en Río Grande del Sur y Uruguay". Publicación 6to. Congreso Nacional de Veterinaria. Noviembre 1996. Montevideo - Uruguay.-

Rivero, R.; Quintana, S.; Feola, R. y Haedo, F.1989 et al. "Principales enfermedades diagnosticadas en el área de influencia del Laboratorio de Diagnóstico Regional Noroeste del C.I Vet. "Miguel C. Rubino" Publicación XVII Jornadas Uruguayas de Buiatría. 16 - 18/06/89 - Paysandú, Uruguay.-

Rivero R., Zabala A., Gil J., Gianneechini R.E., Moraes J. 1998. "Intoxicación por *Anagallis Arvensis* en Bovinos y Ovinos del Uruguay. Publicación de las XXVI Jornadas Uruguays de Buiatria. Junio 1998 .p: 26-29