EFFECTS OF CHRONIC INGESTION OF TARBUSH (FLOURENSIA CERNUA) ON EWE LAMBS

E FREDRICKSON, J THILSTED, R ESTELL, K HAVSTAD

Reprinted from **Veterinary and Human Toxicology,** Vol. 36, No. 5, October 1994, pp. 409-415

ORIGINAL RESEARCH

Effect of Chronic Ingestion of Tarbush (Flourensia Cernua) on Ewe Lambs

Ed Fredrickson PhD
USDA-ARS, Jornada Experimental Range, Las Cruces, NM 88003-0003

John Thilsted DVM, PhD
New Mexico Department of Agriculture, Veterinary Diagnostic Services, Albuquerque, NM 87196-4700

Rick Estell PhD
USDA-ARS, Jornada Experimental Range, Las Cruces, NM 88003-0003

and

Kris Havstad PhD
USDA-ARS, Jornada Experimental Range, Las Cruces, NM 88003-0003

ABSTRACT. Efforts to increase livestock utilization of tarbush are being coupled with studies to examine tarbush toxicity. Thirty-eight (19/treatment) ewe lambs were assigned at birth to receive either tarbush or alfalfa (15%, dry matter basis) in a sorghum-based growing ration. Lambs were pen-fed this diet 60 d pre-weaning and 60 d post-weaning. No differences existed between treatments in feed consumption. In the tarbush group, 1 lamb died of unknown causes at 90 d of age, while 3 lambs died between 115 and 120 d of age. There were no deaths in the alfalfa group. Shortly before death, lambs fed tarbush appeared lethargic, disoriented and anorectic. At 122 d of age, 5 lambs were randomly selected from each group. Feces and jugular blood samples were obtained from each lamb before being euthanized and necropsied the following day. All fecal samples were negative for occult blood. Serum gamma glutamyl-transpeptidase ($P<0.001$) and aspartate aminotransferase ($P<0.001$) activities and platelet counts ($P<0.05$) were elevated in lambs fed tarbush, while serum calcium concentrations tended ($P<0.10$) to be greater. Histologic examination revealed diffuse liver apoptosis in lambs fed tarbush. These data indicate tarbush leaves cause liver damage when fed for extended periods of time.

Tarbush (Flourensia cernua DC) is an edemic plant species of the Chihuahuan Desert, with its northern range in southeastern Arizona, the lower Rio Grande and Pecos Valleys in New Mexico, and the Trans-Pecos and Edwards Plateau regions of Texas. In Mexico it can be found in much of Chihuahua, Coahuila, Durango and Zacatecas (1). Tarbush is usually less than 1 m high (1), but can be 1 to 2 m high in some locales (2). It is a multibranched, deciduous (semi-evergreen) shrub (3) with blackish stems and leaves that are entire, ovate to oval, and covered with an aromatic resin. During years that it flowers, it has yellow discoid flowers borne from terminal and axillary axes. Other names include varnish-bush, hojase and blackbrush. In Mexico, the leaves and flower heads are sold in drug markets as "hojase" or "hojasen" for indigestion (1,4).

Within the Chihuahuan Desert, tarbush has rapidly increased in dominance over the past century in areas previously classified as desert grasslands. The increase appears to be ongoing and associated with a decline in grasses and other herbaceous species. The shift from grasslands to land dominated by tarbush negatively impacts forage production, water quality and esthetic land values. Past efforts to control the spread of tarbush involved chemical and mechanical procedures, but these procedures have not been cost effective. Research to examine the potential use of sheep as a biological control for tarbush is ongoing. Tarbush has a nutrient profile similar to alfalfa (unpublished data), which suggests that dietary tarbush may have nutritional benefits.

Because the fruit and flowers of tarbush have been reported to be toxic to antelope (Antilocarpa americana) (5), goats (6,7), sheep (6) and rabbits (7), periods of intense grazing may need to be restricted to non-flowering periods. Previous research (6,7) indicated that tarbush leaves are not toxic; however, that research was performed using large doses administered over short periods of time. The possibility of toxicosis from tarbush leaves at doses that reflect observed levels of intake (8) over time has not been examined. Research presently reported was conducted to evaluate potential toxicosis in sheep from tarbush leaves when consumed at rates previously reported for grazing sheep (8).

MATERIALS AND METHODS

This study was conducted in conjunction with research to evaluate effects of previous exposure on the dietary preference of lambs for tarbush. Thirty eight (19/treatment) ewe lambs of Polypay x Rambouillet breeding were assigned at birth to receive either tarbush or alfalfa in a sorghum-based, pelleted growing ration. The ration dry matter consisted of 53% grain sorghum, 25% cottonseed meal, 5% molasses, 0.5% trace mineral salt and 1.5% calcium carbonate. The remaining 15% was either tarbush or alfalfa. The tarbush was collected during August 1991 from a singular 3 ha location on the Jornada Experimental Range in southcentral New Mexico. Neither flowers nor fruit were present on tarbush during collections. Each bush was harvested at the base and air-dried for 96 h. Leaves were then separated from the stems by threshing and winnowing. Mixing and pelleting into 6.4 mm pellets was performed by a local mill (Valley Feed Mills, Clint, TX). Nutritive analyses of the pellets was performed by the Northeast DHIA Forage Analysis Laboratory (Ithaca, NY) using conventional procedures. Results are listed in Table 1.

TABLE 1. Dry matter composition of pelleted rations containing either 15% tarbush or 15% alfalfa and fed to growing ewe lambs.

Item	15% Tarbush	15% Alfalfa
Dry matter, g/100 g as fed	92.7	92.8
Crude protein (N X 6.25), g/100 g	20.7	23.0
Available protein, g/100 g	19.4	21.5
Neutral detergent fiber, g/100 g	13.3	19.2
Acid detergent fiber, g/100 g	8.0	11.5
Total digestible nutrients, g/100g	78.0	76.0
Calcium, g/100 g	0.87	0.36
Phosphorus, g/100 g	0.52	0.54
Magnesium, g/100 g	0.35	0.32
Potassium, g/100 g	1.21	1.23
Sodium, g/100 g	0.391	.111
Iron, mg/kg	432	178
Zinc, mg/kg	104	44
Copper, mg/kg	34	10
Manganese, mg/kg	63	25
Molybdenum, mg/kg	1.7	1.5

Lambs were fed the above diets from birth until weaning at 60 d and 60 d post-weaning. During the pre-weaning period, barriers were constructed that restricted the lamb's access to solid feed other than their respective experimental feeds. Likewise, dams were prevented from consuming the pellets intended for lambs. Water and trace mineralized salt blocks (Acco Feeds, Amarillo, TX) were available at all times. No differences existed between treatments in feed consumption during either the pre- or post-weaning periods.

In lambs fed tarbush, 1 lamb died of unknown causes at 90 d of age, and 3 lambs died between 115 and 120 d of age. Necropsies were not performed on these 4 animals. There were no deaths of lambs fed the pellets containing alfalfa. Shortly before death, lambs fed tarbush appeared lethargic, disoriented and anorectic. At 122 d of age, 5 lambs from each treatment were randomly selected from the remaining lambs and evaluated for signs of toxicosis related to chronic consumption of tarbush.

Also at 122 d of age, fecal samples were obtained rectally from each lamb and tested for occult blood. Whole blood was collected into sterile tubes containing 0.5 ml 15% EDTA via jugular venipuncture and immediately analyzed for hemoglobin, hematocrit, and red and white blood cell counts using automated cell counters. At that time, approximately 10 ml of blood was collected into sterile serum separator tubes, allowed to clot for 30 min at room temperature, and centrifuged at 2,000 x g for 20 min at 4 C. Separated serum was stored at -20 C until serum constituents were analyzed using an automated serum analyzer. Analyses of occult blood, complete blood count and serum clinical profile (electrolytes, metabolites and enzymes) were contracted to Southwest Medical Laboratories (Las Cruces, NM).

The following day, lambs were euthanized and necropsy examinations performed. Skeletal muscles, reproductive tract, central nervous system, and thoracic and abdominal organs were examined grossly, and samples of tissues were fixed in 10% formalin solution for histopathology. Tissues for histopathology were sectioned at 5 μm and stained with hematoxylin and eosin (H&E).

Data were analyzed using SAS Institute Inc procedures (1989) for a 2-sample t-test (df = 4).

RESULTS

The test for fecal occult blood was negative for all lambs. Blood cell counts (Table 2) and morphology were similar ($P < 0.10$) between dietary treatments, except that platelet counts were elevated in 4 of the 5 lambs fed tarbush.

Blood serum constituents values are listed in Table 3. Gamma-glutamyl transpeptidase (3.8 times; $P < 0.005$) and aspartate aminotransferase (12.6 times; $P < 0.0005$) activities were greater in lambs fed 15% tarbush. Likewise, serum concentrations of potassium ($P = 0.15$) and calcium ($P < 0.1$) also were greater in tarbush-fed lambs. One lamb fed the alfalfa pellet had a very high serum lactate dehydrogenase activity (4,359 U/L); the remaining lambs had lactate dehydrogenase concentrations that were only mildly elevated (467 to 605 U/L) (9). In contrast, all lambs fed tarbush had serum lactate dehydrogenase activities (1029 to 3784 U/L) that were markedly elevated. A similar pattern was noted for creatine kinase but was less pronounced.

TABLE 2. Hematology determinations for ewe lambs fed either a 15% tarbush or a 15% alfalfa growing ration.

	15% Tarbush						15% Alfalfa					
Item\ID	5693T	2319T	5682T	5720T	5694T	Mean ± SE*	5688A	5691A	2355A	5700A	5723A	Mean ± SE*
White blood cells, x 10^3/µl	6.7	7.9	7.6	7.0	3.6	6.6±0.77	6.6	5.1	9.1	6.5	5.3	6.5±0.71
Red Blood cells, x 10^6/µl	6.0	5.9	4.6	5.7	5.7	5.6±0.24	5.2	5.1	5.1	5.6	5.3	5.3±0.09
Hemoglobin, g/dl	13	11.5	11.0	11.8	13.5	12.2±0.47	12.7	12.8	11.2	12.5	12.6	12.4±0.29
Hematocrit, %	26.5	24.8	20.9	24.7	26.0	24.6±0.98	23.6	23.2	22.0	25.1	24.3	23.6±0.52
Platlets, x 10^3/µl**	661	564	571	266	1356	683±181	394	408	180	267	314	312±42

*Treatment mean ± standard error of the mean (n = 4)
**Treatment means within row differ ($P < 0.05$)

Body weights and visceral organ weights did not differ between dietary treatments ($P > 0.10$; Table 4). During gross necropsy observations, it was noted that the ruminal mucosa of the lambs fed tarbush were dark grey with considerable amounts of lightly adherent feed particles in comparison to the light grey-tan color and relatively few adherent feed particles of the animals fed alfalfa. No evidence of rumenitis was found in the histopathology of the rumens.

TABLE 3. Blood serum constituents of ewe lambs fed either a 15% tarbush or a 15% alfalfa growing ration.

	15% Tarbush						15% Alfalfa					
Constituent\ID	5693T	2319T	5682T	5720T	5694T	Mean±SE*	5688A	5691A	2355A	5700A	5723A	Mean±SE*
Albumin, g/dl	4.0	3.9	3.5	3.7	3.6	3.7±0.09	3.6	44.2	4.1	3.9	3.7	3.9±0.11
Globulin, g/dl	2.6	2.0	1.8	2.2	2.1	2.1±0.13	2.0	1.8	2.3	2.7	2.2	2.2±0.15
Total protein, g/dl	6.6	5.9	5.3	5.9	5.7	5.9±0.21	5.6	6.0	6.4	6.6	5.9	6.1±0.18
Total bilirubin, mg/dl	0.4	0.2	0.4	0.2	0.2	0.3±0.05	0.2	0.2	0.3	0.3	0.2	0.2±0.03
Direct bilirubin, mg/dl	0.1	0.1	0.1	0.1	0	0.1±0.02	0	0	0.1	0.1	0	0±0.01
Indirect bilirubin, mg/dl	0.3	0.2	0.3	0.2	0.1	0.2±0.03	0.2	0.2	0.2	0.3	0.2	0.2±0.02
Alkaline phosphatase, U/l	1164	786	582	1040	1205	955±118.6	511	1256	672	1055	975	894±134.1
Creatine kinase, U/l	203	234	264	225	296	244±16.2	149	155	243	203	114	173±22.6
Gamma glutamyl transpeptidase, U/l***	458	210	221	231	238	271±46.8	59	84	73	59	84	71.8±5.6
Aspartate amino- transferase, U/l***	2317	663	3452	1460	1812	1941±464.0	143	158	87	92	289	154±36.5
Lactate dehydrogenase, U/l	3784	1029	1737	1907	1551	2002±469.3	568	605	4359	467	547	1309±762.8
Glucose, mg/dl	80	91	81	84	94	86±2.8	84	90	87	92	86	88±1.4
Cholesterol, mg/dl	109	60	68	87	87	82±8.5	33	54	52	80	55	55±7.5
Triglyceride, mg/dl	34	12	17	17	20	20±3.7	25	20	10	9	21	17±3.2
Blood urea nitrogen, mg/dl	21	30	19	26	24	24±1.9	33	30	29	30	34	31.2±1.0
Creatinine, mg/dl	1.0	1.0	0.8	1.0	0.9	0.9±0.04	1.2	1.1	1.0	1.0	1.1	1.1±0.04
Sodium, mEq/l	143	142	142	145	147	144±1.0	142	145	142	142	143	143±0.6
Potassium, mEq/l	4.8	4.8	4.8	5.1	4.9	4.9±0.06	4.4	4.9	4.1	4.4	4.4	4.4±0.13
Chloride, mEq/l	101	103	102	106	109	104±1.5	100	104	105	104	102	103±0.9
Bicarbonate/CO_2, mEq/l	29	27	31	25	25	27±1.2	31	26	29	29	30	29±0.8
Calcium, mg/dl**	10.6	10.4	11.5	10.6	9.8	10.6±0.27	9.6	9.7	9.3	9.8	9.3	9.5±0.10
Phosphorus, mg/dl	8.8	8.5	8.0	9.8	9.2	8.9±0.31	8.6	11.2	7.3	7.4	9.2	8.7±0.71
Iron, µg/dl	238	249	312	214	239	250±16.4	331	208	146	145	214	209±33.9
Calculated osmolality, mOsm/l	287	289	284	293	296	290±2.1	289	295	287	288	291	290±1.4

*Treatment mean ± standard error of the mean (n = 4).
**Treatment means within row differ ($P < 0.05$).
***Treatment means within row differ ($P < 0.01$).

TABLE 4. Body weight (kg) and relative weight of selected organs (g/kg body weight) of ewe lambs fed either a 15% tarbush or a 15% alfalfa growing ration.

	15% Tarbush						15% Alfalfa					
Item\ID	3569T	2319T	5682T	5720T	5694T	Mean ± SE*	5688A	5691A	2355A	5700A	5723A	Mean ± SE*
Body weight	34.5	37.2	27.2	29.5	30.8	31.8±1.78	27.2	37.6	28.6	26.3	24.0	28.8±2.34
Heart	5.2	4.5	4.6	5.6	5.0	5.0±0.19	5.2	5.3	5.7	5.7	5.3	5.4±0.10
Liver	20.6	23.4	21.3	19.0	21.1	21.1±0.71	21.3	22.3	23.4	22.0	23.7	22.6±0.45
Kidneys	3.5	4.1	4.0	5.2	3.6	4.1±0.31	5.0	3.5	3.7	6.7	7.0	5.2±0.73
Spleen	2.8	2.4	2.3	2.4	2.3	2.4±0.09	2.4	1.8	1.8	2.6	2.4	2.2±0.18

*Treatment mean ± standard error of the mean (n = 4)

All lambs receiving alfalfa developed urinary calculi, whereas none of the lambs fed tarbush developed calculi. Urinary calculi samples from 3 animals were sent to Dr P Osborne (Minnesota Urolith Center, St Paul, MN) for analysis. Urinary calculi from lamb 5723A was composed of calcium phosphate (30%; carbonate form), calcium phosphate (60%; hydroxyl form) and protein (10%); uroliths from lamb 5688A was composed of magnesium hydrogen phosphate trihydrate (86%), calcium phosphate (10%; hydroxyl form) and protein (4%); uroliths from lamb 5700A was composed of magnesium hydrogen phosphate trihydrate (60%), magnesium orthophosphate tetrahydrate (25%), calcium phosphate (10%; hydroxyl form) and protein (5%). A protein matrix was evident in all samples.

Microscopic examination of samples of liver taken at necropsy revealed ($P < 0.05$) moderate hepatic apoptosis (individual cell death) in all lambs fed tarbush and minimal apoptosis in alfalfa-fed lambs. The hepatic apoptosis was characterized by scattered shrunken hyalinized hepatocytes which had pycnotic fragmented nuclei. Some affected hepatocytes had been phagocytized by adjacent cells. No inflammatory cell infiltrate was associated with the apoptotic hepatocytes (Fig 1). The number of apoptotic hepatocytes/10 high power fields are listed in Table 5.

Several lesions identified were considered unrelated to tarbush consumption because they were not consistently observed between treatment groups, or were found in only 1 or 2 animals. These lesions include suppurative microfocal hepatitis (lambs 2319T, 5693T, 5682T, 5720T, 5691A, 5723A and 5688A), hepatic sinusoidal dilatation (lamb 5682T), nonsuppurative microfocal myocarditis (lambs 2319T, 5691A and 5723A), degenerative myopathy (lambs 2319T, 5682T and 5688A), microfocal eosinophilic adrenitis (lambs 5693T and 5723A), bile duct hyperplasia (lamb 5682T), chronic ulcerative cholecystitis (lamb 5720T) and pulmonary hemorrhage (lamb 5723A). Focal interstitial nephritis (lambs 5720T, 5691A, 5723A, 5688A, 5700A) and pyelitis (lambs 5720T, 2355A, 5691A, 5723A, 5700A) were found primarily in lambs fed pellets containing alfalfa and were probably related to the urolithiasis seen in these lambs.

DISCUSSION

Over a 3-y period, Mathews (6) collected the ripe fruit of tarbush from 6 different locations in West Texas to examine tarbush toxicity in both sheep and goats. Pulse doses, divided evenly between am and pm administrations to both fasted and fed animals, caused death in some animals within 18 h. Slight toxicosis was denoted by loss of appetite that lasted not more than 72 h. Signs of severe, acute toxicosis included listlessness, nasal mucus discharge, grinding of the teeth and an expiratory grunt. These animals appeared "very sick" for a period lasting 5 to 6 d before recovery occurred. In fatal cases, initial symptoms included excessive salivation followed by muscular twitching, groaning and grinding of teeth within 2 to 4 h post-dosing. Animals avoided movement while remaining on their feet with an arched back until just prior to death. Post mortem examination revealed severe inflammation of the abomasum and the first 30 to 60 cm of the proximal duodenum. Additionally, there was "marked congestion" of the liver and kidneys. Doses as small as 9 g/kg body weight produced death in some animals while 10 g/kg body weight had no effect in others. This variation was attributed to animal tolerance of the toxin(s) since it could not be attributed to either location of tarbush collec-

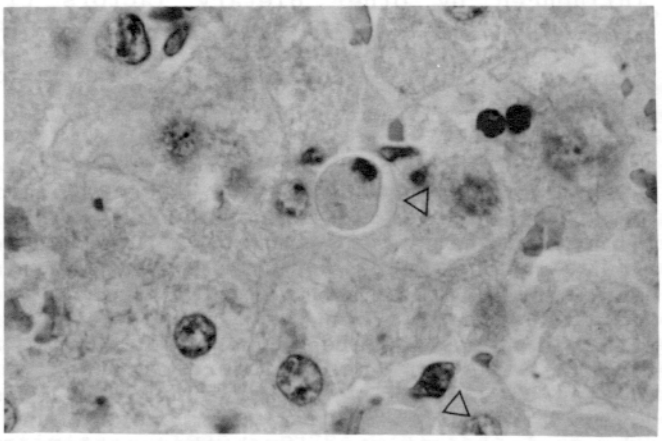

Figure 1. Apoptotic hepatocytes (arrowheads) in the liver of tarbush-fed sheep #2319T. H&E X 950.

TABLE 5. Number of apoptotic hepatocytes per 10 high-power fields of ewe lambs fed either a 15% tarbush or a 15% alfalfa growing ration.

	15% Tarbush						15% Alfalfa					
Item\ID	5693T	2319T	5682T	5720T	5694T	Mean ± SE*	5688A	5691A	2355A	5700A	5723A	Mean ± SE*
Apoptotic hepatocytes*	77	59	18	43	53	50±9.7	7	12	2	12	7	8±1.9

*Treatment mean ± standard error of the mean (n = 4)

tions or year of collection. In Matthew's study (6), the feeding of green leaves failed to produce toxic effects.

Dollahite and Allen (7) later determined that all phenological stages of tarbush reproductive tissue were toxic (flowering to mature fruit) to goats and rabbits. The signs and pathology of their study were similar to those described by Mathews (6), but congestion of the brain, heart, lungs and spleen were also described. One goat was fed green leaves for 13 d (5.3 g/kg body weight on days 1 and 2 and 10.6 g/kg body weight thereafter). This animal lost 46.6% of its body weight in 16 d. The animal recovered, and regained its weight after being fed a bermuda grass-grain diet. This loss of weight was attributed to the low levels of digestible nutrients in tarbush. This conclusion was not substantiated by the results obtained in our laboratory. Using slight modifications of a conventional in vitro organic matter disappearance technique (10), we found the in vitro organic matter disappearance of tarbush leaves to be similar to alfalfa (63 g organic matter disappearance/100 g tarbush organic matter). In sacco ruminal digestion of tarbush and alfalfa leaves yielded similar results. The body weight loss described by Dollahite and Allen (7), and in studies conducted at our laboratory (unpublished data), must be attributed to other factors, perhaps anti-nutritional phytochemical components of tarbush. In the present study, growth rates of ewe lambs consuming 15% tarbush in a growing ration were not different ($P > 0.10$) from growth rates of animals consuming the same ration containing 15% alfalfa rather than tarbush. The degree of toxicosis elicited by a specific toxicant is often influenced by other dietary factors (11); therefore, observations in this study may differ from other studies using diets more representative of ruminants grazing native rangeland.

Hailey et al (5) described tarbush toxicosis in pronghorn antelope (Antilocapra americana) that were also experiencing malnutrition. In that case, hemorrhagic lesions were found in the lungs, rumen, omasum, abomasum and small intestine. Similar lesions were not found in pronghorn antelope in which ruminal digesta appeared free of tarbush. Although pronghorn antelope were observed dying during the flowering and fruiting of tarbush, only tarbush leaves and stems were found in the ruminal contents. Such observations suggest pronghorn antelope avoided the most toxic components (flower and fruit), while selecting the less toxic components (leaves) of tarbush.

In our study, 4 of 19 ewe lambs fed 15% tarbush pellets died of unknown causes, while no deaths occurred in the control group. Of the ewe lambs necropsied, congestion of the viscera or severe hemorrhagic lesions were not found; however, elevated activities of serum gamma glutamyl transpeptidase, aspartate aminotransferase and lactate dehydrogenase indicated some tissue damage occurred from feeding tarbush. Another important finding was the distinct liver apoptosis in the lambs fed tarbush. Apoptosis is characterized by phagocytosis by adjoining cells or macrophages before cell lysis occurs; during morphological degeneration cell contents are retained without inducing inflammation (12, 13). Elevated serum concentrations of aspartate aminotransferase and gamma glutamyl transpeptidase are indicative of liver damage (14,15). During apoptosis, phagocytosis is extremely rapid, and the number of detectable apoptotic cells may appear low even when cell death rate is high (12). Regardless, it is clear that phytochemical compounds in tarbush either induce or mimic apoptotic stimuli in the liver. From other studies in our laboratory, we observed that wethers consuming diets consisting of 20 and 30% tarbush for 21 d similarly had higher activities ($P < 0.05$) of the same serum enzymes when compared to wethers consuming 0 or 10% tarbush (unpublished data). Although this difference was statistically significant, we did not view it as being physiologically relevant at the time.

Elevated activity of serum lactate dehydrogenase and a tendency for greater creatine kinase activity and serum potassium concentrations in the lambs fed tarbush may indicate myopathies or brain damage (15). One control lamb and 2 tarbush-fed lambs had histologically evident muscle degeneration; however, these observations were not consistent among treatment groups. Greater numbers of experimental units are required to establish actual differences or trends.

With the exception of lambs 5694T and 2355A, platelet counts were within normal limits (250,000 to 750,000 count/μL of blood) (16); however, platelet counts were ($P < 0.05$) elevated in the tarbush group. Platelets may be elevated for a multiplicity of reasons. Of the factors commonly known to increase blood platelet counts (16), hemorrhage, chronic infection or inflammatory conditions seem the most probable causes in this study.

Chronic infection is considered a likely cause, since sheep consuming tarbush in this study and others (unpublished data) seemed to require more attention for various illnesses than their counterparts not consuming tarbush. An alternate hypothesis should consider that the platelet counts in the control group (15% alfalfa) may have been depressed.

Hematocrit values for both dosed groups were unusually low (16). We believe these low values were the result of automated cell counters which were not correctly calibrated for sheep blood. Therefore, the small-sized erythrocytes of sheep (16) may explain the low red blood cell counts and affect hematocrit calculations.

The cause of the phosphatic calculi was probably the high phosphorus content of the control diet relative to calcium concentration (Table 1) (17). Even though the diets were designed to be similar between treatments, obtaining a pelleted feed from most commercial operations that meets specifications for minor constituents is difficult when mixing small batches of feed. A proper calcium-to-phosphorous ratio (1.5:1 to 2:1) should have prevented phosphatic calculi.

The phytochemistry of tarbush has rarely been examined, even though aspects of its chemistry appear to be highly unusual. Extracts of intact tarbush leaves have yielded approximately 100 different compounds that appear to be chiefly mono- and sesquiterpenes (18). Of those compounds we have positively identified, many have been attributed with various biological activities in mammals. Other researchers (19) isolated 2 unusual sesquiterpenes, the eremophilane flourensic acid and the aromadendrane flourensadiol from the inflorescence of tarbush. The compound 1,3-arachidobehenicin was also isolated. These authors concluded, based upon goldfish assays, that the greatest toxicity was in "petrol-soluble" plant extracts.

A vinylic-substituted aromatic hemiterpene, which is a type of compound that rarely occurs in nature, has also been isolated in tarbush (20). To our knowledge, the biological activity of this compound has not been determined. Several benzopyrans and benzofurans isolated from tarbush have hemolytic properties that are increased substantially in the presence of UV light (21), with benzopyrans having greater hemolytic activity than benzofurans (21). Another Asteraceae (Isocoma wrightii) found in the Chihuahuan Desert contains a family of benzofurans that are responsible for "milk sickness" in humans and "trembles" in cattle (22).

Four methoxylated flavones in tarbush foliage have been described (23,24). Of these compounds, hispidulin is a cytotoxin found in several Eupatorium species (25). Five 6,8-di-C-glycosylflavones have also been characterized (23). Flavonoids may be a major component of tarbush. We have found that phenolic compounds represent about 9% of the leaf dry weight, with condensed tannins being a minor component (less than 0.5% of the dry weight) of the phenolic compound (unpublished data).

SUMMARY AND IMPLICATIONS

Tarbush leaves caused toxicosis in ewe lambs when consumed as 15% of a pelleted creep feed for 120 d. The feeding of tarbush was associated with a death loss higher than that of the control group. Toxicosis was manifested as apoptotic liver damage and probable damage to muscles and other tissues. The toxin(s) have not been identified, but are thought to occur in the nonpolar fraction of plant extracts. Further research designed to increase utilization of tarbush by domestic animals will need to characterize tarbush toxicosis and identify tarbush toxin(s). Knowledge of spatial and temporal distribution of toxins within plant and across plants, and of variations in animal responses to these toxins, will be essential to identifying the optimal periods of tarbush use by livestock.

ACKNOWLEDGEMENTS

The authors wish to thank Greg Jillson, Larry Shupe and Drs Dean Anderson, Melissa Behr, Marta Remmenga and Stan Smith for their valuable assistance.

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