UNIVERSITY OF CALIFORNIA
AGRICULTURE & NATURAL RESOURCES

SIERRA FOOTHILL RESEARCH & EXTENSION CENTER

Presents:

Annual Beef & Range Field Day

Beef Cattle Husbandry

APRIL 9, 2011

In Cooperation With:
University of California Cooperative Extension
Dept. of Animal Science, UC Davis

Beef and Range Field Day
UC Sierra Foothill Research & Extension Center

Saturday, April 9th , 2011

AGENDA

8:30 – 9:15 AM **Registration** -- $15 Adults (includes proceedings, refreshments & lunch)
$8 Children under 12 yrs.

9:15 AM **Welcome, Introductions & Overview** – Art Craigmill, Director, Sierra Foothill Research & Extension Center (SFREC)

9:30 AM **Using DNA information on commercial cattle ranches** – Alison Van Eenennaam, Extension Specialist, Dept. of Animal Science, UC Davis

10:10 AM **Beef Niche Market Update** -- Roger Ingram, UCCE Farm Advisor, Placer/Nevada Counties

10:40 AM **The UC-SFREC Herd Health Program – Herd health management**
Glenn Nader, UCCE Farm Advisor, Yuba/Sutter/Butte Counties
John Maas, Extension Veterinarian, School of Vet Med, UC Davis
Bruce Hoar, Asst Professor, School of Vet Med, UC Davis

11:30 AM **Laws and regulations regarding transport of ag equipment and livestock** –
California Highway Patrol

Noon **Tri-tip BBQ Lunch** – Served by the Yuba-Sutter Cowbelles and SFREC Staff

12:20 **California Cattlemen's Association – Industry Update**
California Beef Cattle Improvement Association Update

1:15 – 3:30 PM **Concurrent Repeating Sessions and Demonstrations/displays:**

Chute-side health for producers – Bruce Hoar, Asst Professor, School of Vet Med, UC Davis and Justin Tindall, SFREC Animal Health Tech.

Poisonous plants – Dr. Bob Poppenga, Professor & Dr. Snehal Tawde, Resident, Toxicology Section, California Animal Health & Food Safety Lab System, Davis, CA

Calving school -- Sponsored by the UCD Dept. of Animal Science and UC Cooperative Extension
John Maas, Extension Veterinarian, School of Vet Med, UC Davis
Dan Myers, Staff Research Associate, Dept. of Animal Science, UC Davis

Rotary wiper for selective pasture weed control demonstration – Dustin Flavell, SFREC Superintendent and Roger Ingram, UCCE Farm Advisor, Placer/Nevada Counties

For more information about the Sierra Foothill Research & Extension Center, go to:
http://ucanr.org/sites/sfrec/

Annual Beef & Range Field Day Proceedings
April 9, 2011

LIST OF CONTENTS

Uses of DNA Information on Commercial Cattle Ranches (Page 1)
Alison Van Eenennaam, Cooperative Extension Specialist, Animal Biotechnology & Genomics, Dept. of Animal Science, UC Davis

Beef Niche Market Update (Page9)
Roger Ingram, UCCE Livestock and Range Farm Advisor, Nevada/Placer County

The UC-SFREC Heard Health Program (Page 13)
Compiled by the SFREC Animal Research Committee

Vet Views **(**Page 19)
John Maas, DVM, MS, DACVN, DACVIM Extension Veterinarian, University of California, Davis

Livestock-Poisoning Plants of California (Page 23)
Excerpt from UC Agriculture and Natural Resources Publication 8398

Update on Controlling Smutgrass in Irrigated Pastures (Page 33)
Glenn Nader, UCCE Livestock and Range Farm Advisor, Butte/Sutter/Yuba Counties
Josh Davy, UCCE Livestock and Range Farm Advisor, Tehama/Glenn/Colusa Counties
Larry Forero, UCCE Livestock and Range Farm Advisor, Shasta County

Rotating Wipers for Weed Control (Page 35)
Glenn Nader, Josh Davy, Larry Forero, UCCE Livestock and Range Farm Advisors

Uses of DNA information on Commercial Cattle Ranches

Alison Van Eenennaam
Cooperative Extension Specialist, Animal Biotechnology and Genomics
Department of Animal Science, UC Davis

For the past two years we have been collecting DNA, performance and carcass data from all of the calves produced by approximately 2,500 crossbred cows and 120 Angus bulls located on four large Northern California commercial cow-calf operations. This sample collection is part of a USDA-funded project entitled "**Integrating DNA information into beef cattle production systems.**" The overall objective of this project is to develop a large phenotyped and genotyped population to use as a resource to compare different approaches to predict the genetic merit of commercial beef bulls. The research objectives are to compare the current means of genetic prediction of herd sires, which include breed association-derived expected progeny differences (EPDs) with DNA-assisted genetic predictions, and "commercial ranch" genetic evaluations based on offspring performance under field conditions. Additionally, we are interested in exploring the costs and benefits associated with DNA testing in the beef cattle industry.

VALUE OF DNA TESTS FOR PARENTAGE DETERMINATION

In the commercial cow-calf sector, the principal determinants of income are the number of sale animals and the value per sale animal. Most commercial producers sell their calves at weaning or shortly thereafter. Their financial returns are therefore very closely tied to the number of calves produced. A bull has two qualities of value to producers. One is his ability to impregnate cows, and the second is having genes for superior performance to pass on to his offspring. The latter is predicted by EPDs. However, the former is hard if not impossible to predict, and if a bull sires no calves then his EPD values are irrelevant.

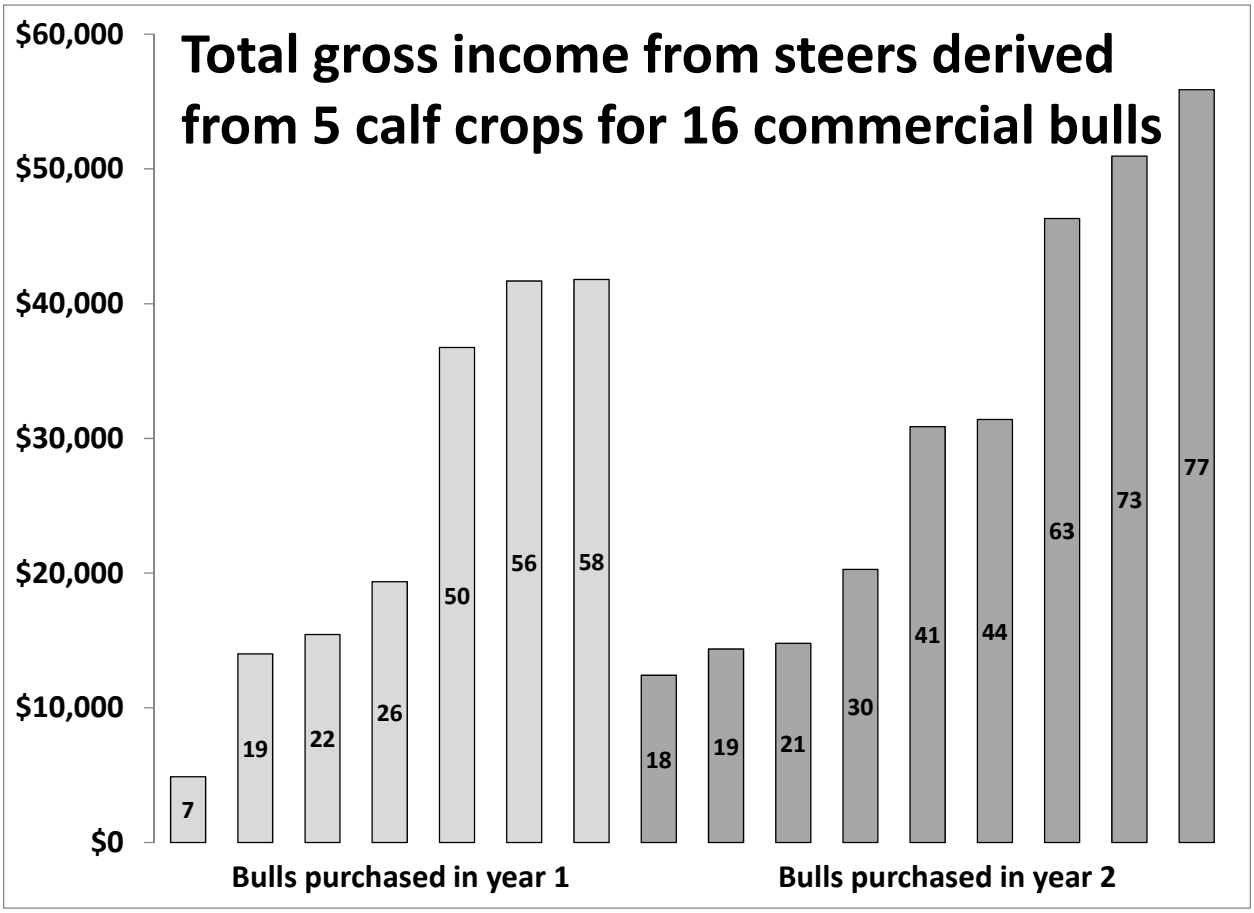
Recently we compiled data on all steer progeny derived from two cohorts of commercial bulls purchased in successive years on one of the commercial ranches in our study. Both year groups were comprised of apparently healthy bulls that had passed a breeding soundness examination. Both year groups served in multisire breeding pastures for 5 consecutive breeding seasons. Having multiple sires present with a group of cows has a number of benefits including higher fertility, precluding sire failure, and reducing the calving interval. It also minimizes the number of pastures needed, thereby facilitating better pasture management (Van Eenennaam et al., 2007).

We used a DNA parentage panel to assign the paternity of each calf to a bull. Offspring were sold at an average age of 10 months to a commercial feedlot, and although the ranch did not retain ownership, they participated in a program that required selection for specific carcass attributes and rewarded carcass quality with a premium paid back to the producer. The average gross return including the quality premium derived from the steer progeny of each sire was $721 and did not differ greatly among the bulls, but the total gross revenue derived from all male offspring of each bull ranged from $4,881 to $55,889 due to differences in sire prolificacy (Figure 1). This huge discrepancy in calf numbers shows how certain bulls in a team may disproportionately influence affect profit.

VALUE OF DNA TESTS TO IMPROVE THE ACCURACY OF EPDs

Genetic improvement on commercial ranches is largely realized through the purchase of bulls from seedstock producers. There may be some value in using DNA testing to improve the accuracy of identifying above-average herd sires. However, producers would want this information at the time of purchase and so DNA testing costs have typically been paid by the seedstock producer, and recouped by an increased price at the time of bull sale. Until recently, DNA tests results (stars, scores, MVPs) have been presented in sire catalogs as a piece of information separate from EPDs. This has led to confusion especially when the two sources of information offered contradictory information regarding a bull's genetic merit for a given trait. This has often begged the question of which to use. Sometimes it has led to the belief that one must be incorrect (Spangler and Van Eenennaam, 2010).

Figure 1. Total gross revenue (number of male calves sired shown inside the bars) derived from all of the steer progeny produced by two cohorts of yearling bulls purchased in successive years. Both groups served in multisire breeding groups for 5 breeding seasons on a US commercial ranch. Offspring were marketed at an average of 314 days of age to the feedlot, and an additional carcass-based quality premium was paid to the commercial producer.



The DNA testing industry has matured from marker tests involving a handful of markers explaining a relatively modest amount (0-10%) of the genetic variation in the target trait, to panels involving hundreds or thousands of markers. This is an exciting development because many of the production traits of interest to beef cattle producers are likely to be controlled by a large number of genes. High density panels containing more than 50,000 (50K) markers, or panels of several hundred of the best markers derived from the high density panels, are likely to be more predictive than previous commercial test offerings. It stands to reason that as the number of markers included in the test increases, so will the proportion of genetic variation explained by the test.

The proportion of genetic variation explained by a test provides producers with a way to quantitatively evaluate the merit of commercial products. Factors that affect this include the number of animals and records that were present in the "training" population used to develop the test. Larger training populations result in better DNA tests. It is also easier to develop accurate DNA tests for traits with high heritability as compared to traits with low heritability. Tests that are developed in one breed have generally not worked well in other breeds, with the exception of the test for tenderness. Because many factors can influence the accuracy of DNA tests, it is important to develop estimates of the accuracy of DNA tests currently on the market. Independent estimates are not available for many traits. Absence of this metric is a major deficiency in the information that is available to producers to evaluate the utility of including DNA tests in their selection system (Van Eenennaam, 2010).

DNA tests can be used much more effectively when they are incorporated into and presented as EPDs. Such an approach is appealing as it presents results in a format that is familiar to producers, and it eliminates the choice that is implicitly associated with the current practice of publishing traditional EPDs and marker information separately. The promise of using DNA information to improve the accuracy of EPDs on young animals is starting to be realized, at least for Angus cattle. First there was an agreement between Angus Genetics Inc. (AGI) and IGENITY to calculate genomic-enhanced expected progeny differences (GEPDs) for multiple carcass traits using American Angus Association carcass and ultrasound data. Then, in November 2010, AGI announced an agreement to similarly accept Pfizer Animal Genetics' High-Density 50K test results for incorporation into GEPDs.

Knowledge of the amount of genetic variation associated with the test is also required to incorporate DNA test information into EPDs. Ideally this value is high, such that DNA tests results can help improve the accuracy of EPDs. Some estimates are available for Angus cattle (Table 1). The Igenity estimates were developed in the US Angus population, whereas the Pfizer estimates were developed in the Australian Angus population. To incorporate Pfizer HD 50K data in the American Angus EPDs, these estimates will need to be calibrated in the US Angus population (Van Eenennaam, 2011).

Table 1. Percentage of genetic variation associated with different DNA tests for various traits. "na" means no estimate is publicly-available or published. h^2 = trait heritability.

Trait	**h^2**	**Igenity® Angus Profile**		**Pfizer HD 50K**	
		Included	% Genetic variation[1]	Included	% Genetic variation [2]
Average Daily Gain	0.28	X	na	X	1-10
Net/residual Feed Intake	0.39	X	na	X	0
Dry matter intake	0.39			X	4-5
Tenderness	0.37	X	na	X	na
Calving Ease (Direct)	0.10			X	6
Birth weight	0.31			X	12-16
Weaning Weight	0.25			X	12-19
Yearling Weight	0.60	X	na		
Calving ease (maternal)	0.10	X	na	X	4
Milking Ability	0.25			X	10-14
Heifer Pregnancy	0.20	X	na		
Stayability	0.10	X	na		
Docility	0.37	X	na		
Yield grade	0.64	X	na		
Carcass weight	0.39	X	29	X	6-13
Backfat thickness	0.36	X	25	X	14-19
Ribeye area	0.40	X	34	X	10-20
Marbling score	0.37	X	42	X	4-11
Percent choice		X	na		

[1] MacNeil, M. D., S. L. Northcutt, R. D. Schnabel, D. J. Garrick, B.W. Woodward, and J. F. Taylor. 2010. Genetic correlations between carcass traits and molecular breeding values in Angus cattle. 9th World Cong. Genet. Appl. Livest. Prod. Leipzig, Germany. August, 2010. http://www.kongressband.de/wcgalp2010/assets/pdf/0482.pdf

[2] Animal Genetics and Breeding Unit (AGBU). 2010. Evaluation of Pfizer Animal Genetics HD 50K MVP Calibration. http://agbu.une.edu.au/pdf/Pfizer_50K_September%202010.pdf

As mentioned previously, independent estimates of the amount of genetic variation associated with DNA tests are not available for all breeds and tests on the market. Breeds other than Angus are also going to have to derive these genetic estimates for any DNA test targeted to be used in that breed. To date, data suggest that tests developed for one breed are unlikely to work very well in another. Even within Angus, tests trained in North American Angus were associated with less genetic variation when used in the Australian/New Zealand Angus population, and required regional recalibration for that population and production system. Although there is a lot of work to be done, this move towards incorporation of DNA information into National Cattle Evaluation (NCE) aligns with the Beef Improvement Federation (BIF) guiding philosophy which reads: "*BIF believes that information from DNA tests only has value in selection when incorporated with all other available forms of performance information for economically important traits in NCE, and when communicated in the form of an EPD with a corresponding BIF accuracy. For some economically important traits, information other than DNA tests may not be available. Selection tools based on these tests should still be expressed as EPD within the normal parameters of NCE.*"

These genetic tests target a suite of traits ranging from fertility and longevity to growth and carcass merit. A question that often arises in conversations with producers is "What is the value of these tests?" The answer to that question depends on what the tests are being used for. Some breeders are just listing the results as an additional source of information in sale catalogs. If this adds value, increasing the animal's sale price beyond the cost of the test, then this makes economic sense. Other people are using tests to make culling or selection decisions on traits that are not currently in breed EPDs (e.g. feed efficiency or tenderness). Working out whether this pays is a little more complicated.

While estimates of genetic merit for new traits may have value, the amount of emphasis to put on them is dependent on their economic value to the commercial producer. For example, should you eliminate animals from your herd based solely on a poor feed efficiency DNA test result? That depends partly on how accurate the test is at predicting superior versus inferior animals. The more accurate a test is, the more opportunity there is to accelerate genetic improvement. This decision also depends on the importance of feed efficiency versus all the other traits contributing to your overall profitability.

One way to make this decision is to develop a "selection index" that weights all traits on their relative economic importance. Indexes consider both the "input" or expense side and the "output" or income side of selection decisions and enable cattle producers to make balanced selection decisions, taking into account the economically-relevant growth, carcass and fertility attributes of each animal to identify which animals are the most profitable for their particular commercial enterprise. Melton (1995) suggested that US cow-calf producers should have a relative economic emphasis of 47% on reproduction, 24% on production, and 30% on carcass traits whereas producers in an integrated system should have a relative economic emphasis of 31% on reproduction, 29% on production and 40% on carcass traits. This relative emphasis will depend on how much the value derived from genetic gain in carcass traits is shared with the producer in the integrated system. If the value of genetic improvement for carcass traits is not paid back to the producers making the selection decisions, there is no economic value to them for continuing to emphasize carcass performance at the expense of reproduction or production.

VALUE OF DNA TESTS TO IMPROVE COMMERCIAL BULL SELECTION

The response to selection and therefore the value associated with a particular DNA test is dependent upon how much that test information improves the accuracy of genetic evaluations at the time of selection, and the value of a unit of genetic improvement. To determine that value I recently did a simulation study to determine the value of DNA tests to increase the accuracy of commercial beef bull selection (Van Eenennaam et al., 2010) I used selection indexes to estimate the value derived from using information from a hypothetical DNA test to increase the accuracy of selection. I expressed these values on a per DNA test basis.

In order to do this simulation study I had to make a number of assumptions about where and how the DNA tests were used. I modeled an example where the seedstock breeder was incurring the

costs of DNA testing to improve the accuracy of bull selection. In this example the seedstock tier consisted of a closed nucleus of 600 breeding females. It was assumed that in the absence of DNA test information, EPDs on young, untested bulls were informed by their own performance records, along with those of their sire, dam and 20 paternal-half sibs.

Each year the top 8 bulls were selected to be stud sires, and 125 (remaining bulls from the top half of the calf crop) were made available for sale to commercial producers. Commercial sires were then used to sire four calf crops at a mating ratio of 25 females: 1 male (i.e. they were exposed to a total of 100 cows). DNA test information was combined with performance records to increase the accuracy of EPDs. This increased $Index selection response 20-41% over that obtained with performance recording alone, depending upon the traits included in the breeding objective.

The value derived from using DNA test information to enable more accurate selection of genetically-superior commercial bulls ranged from AU$61-135 per bull. Assuming that the entire bull calf crop (n = 267) was tested and that the top half of the bulls (n=125) were sold as commercial sires, the breakeven value of the genetic gain derived from DNA testing ranged from **$30-67** per DNA test depending upon the market being targeted. These values assumed commercial producers were willing to pay a price premium for genetically-superior bulls, and some form of industry vertical integration or profit sharing between sectors such that the rewards for improvement in processor traits (e.g. dressing %, marbling score, etc.) were transferred along to commercial producers and breeders. The value of DNA tests to improve traits of direct value to commercial cattle enterprises (e.g. fertility) would be less than this value. For example, 69% of the returns from including DNA data in commercial sire selection for an integrated system index were derived from traits that generate a direct return to processors (e.g. marbling score).

These values were based on using a relatively powerful hypothetical DNA test panel that accurately predicted ALL of the traits in the selection index. DNA tests are most valuable for traits that are not routinely recorded before selection decisions are made. For example, a DNA test accounting for 50% of the genetic variation in a trait like feed efficiency for which EPDs do not exist is likely be more valuable than a DNA test accounting for 50% of the variation in an easily measured trait like weaning weight.

VALUE OF DNA TESTS TO IMPROVE REPLACEMENT HEIFER SELECTION

The cow-calf industry could benefit greatly from tests that accurately predict maternal performance. It has been proposed that DNA information be used to select replacement commercial females, most of which have no EPD information. The value of testing heifers will depend upon the cost of the test relative to the return on investment, the accuracy of the test, and how many heifers are being selected relative to the number available from which to select. I calculated that the breakeven cost of testing all of the potential replacement heifers in a commercial herd with a replacement rate of 20% (i.e. selecting 20 heifers) from the 45 potential replacement heifers born per 100 cows per year using a DNA test with an index accuracy (i.e. correlation between the test result and the true genetic merit) of 0.25 was less than **$5** per test, using the assumptions outlined in the previous example and assuming that the commercial producer was not performance recording his females (i.e. had no other data upon which to base heifer replacement decisions). This low value is due in part to the fact that commercial cows do not produce very many progeny, and so the value derived from spending money on improving the genetic merit of females is spread over few offspring relative to bulls.

It should be noted that unless DNA tests have high accuracy, they should ideally be used in conjunction with available phenotypic data. And here is the quandary when developing tests for replacement female selection. Traits that are of the most economic value to cow-calf producers for replacement heifer selection are low heritability reproductive traits including age at first calving, reproductive success and replacement rate. Research suggests that very large numbers of records will be required to obtain accurate DNA tests for low heritability traits. Further such tests are the most difficult to evaluate as there are few cattle populations with sufficient phenotypic data to enable estimation of the accuracy of DNA tests for these traits. However as most commercial producers have no EPD information upon which to base their replacement heifer selection decisions, even moderately powerful tests will provide previously-absent criteria upon which to base selection decisions.

VALUE OF DNA TESTS FOR GENETIC DEFECTS

DNA information can also be used to test for simply-inherited traits including coat color, horned-status, and recessive genetic defects. There are a large number of genetic abnormalities in cattle occurring in a variety of breeds. These defects have had a significant impact on specific cattle populations. Naturally-occurring recessive genetic defects are common in all species, including humans. The average human carries approximately 2,000 deleterious recessive alleles, of which one to two are thought to be lethal. Recessive conditions only become evident when certain lines of cattle are used very heavily, such that both cows and bulls have common ancestors in their pedigree, thereby allowing a rare genetic defect to become homozygous in their offspring.

Genetic defects are often propagated as a result of specific trait selection. In dogs it has been noted that each of the top 50 breeds has one aspect of breed type that predisposes the breed to a genetic disorder. For example bull dogs are prone to airway obstruction syndrome, and King Charles spaniels are affected by a reduced-size malformation of the skull. Perhaps the most famous example of a genetic defect in 20th century beef breeding was "snorter" dwarfism which became an issue in Angus and Hereford cattle during the 1940s and 1950s. This genetic defect was uncovered as a result of strong selection pressure for animals with small stature. Ultimately the cause of this mutation was traced back to a bull named St. Louis Lad, born in 1899. A 1956 survey of Hereford breeders in the USA identified 50,000 dwarf-producing animals in 47 states.

The Angus breed has recently had to manage three recessive genetic conditions. These include two lethal conditions Arthrogryposis Multiplex (AM; "Curly Calf Syndrome"), and Neuropathic Hydrocephalus (NH). The first is caused by a chromosomal deletion that occurred in Rito 9J9 of B156 7T26 (AAA Registration No. 9682589; born 10/29/1979). The second occurred as a result of a single DNA base pair mutation in his grandson, the widely-used GAR Precision 1680 (AAA Registration No. 11520398; born 9/6/1990). The widespread use of this superior carcass trait bull spurred on by an increased selection emphasis on carcass traits increased the probability of this bull showing up on both sides of many Angus pedigrees, thereby uncovering the presence of any recessive lethal mutations. The third condition is a non-lethal autosomal genetic defect called Congenital Contractural Arachnodactyly (CA; "Fawn Calf Syndrome") that is caused by a ~54,450 base pair deletion.

Genetic tests for AM, NH, and CA became available 1/1/2009, 6/15/2009 and10/4/2010, respectively. The speed with which these genetic tests were developed is testament to the power of having access to the bovine genome sequence information, and is perhaps the greatest success story of genomics never told. The proactive response of the breed association in making genotypes available also helped to rapidly and transparently address the problem (Table 2).

CONCLUSION

The advent of molecular information in the form of both tests for simply inherited traits and complex traits has created both excitement and confusion. The lag between discovery and application has been decreased, allowing for technology to be rapidly delivered to industry. In some cases this has caused confusion surrounding the methods for incorporating this technology into breeding schemes. DNA marker tests results should not be used to replace traditional selection based on EPDs and economic index values, but rather should be seen as providing an additional source of information from which to predict genetic merit. When included in the estimation of genetic predictions, DNA information provides valuable information on young animals which could improve the accuracy of genetic predictions. DNA testing holds the greatest promise for economically-relevant traits which are too expensive to measure, and for which no good selection criteria exist (e.g. residual feed intake or reproduction). Commercial companies have started to offer genetic tests for such traits. Meaningful incorporation of these traits into national cattle evaluations will be required to make the best use of DNA information, and such efforts will call for further collaboration between DNA companies, producers, scientists, and breed associations.

ACKNOWLEDGEMENTS

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Table 2. Dates that Arthrogryposis Multiplex (AM; "Curly Calf Syndrome"), Neuropathic Hydrocephalus (NH), and Congenital Contractural Arachnodactyly (CA; "Fawn Calf Syndrome") were recognized as recessive genetic defects, and subsequent availability of genetic tests and American Angus Association registration cutoff dates.

DATE	AM	NH	CA
Recognized as genetic defect	November 15, 2008	June 12, 2009	July 14, 2010
Commercial test becomes available	January 1, 2009	June 15, 2009	October 4, 2010
Number of carriers recorded (current as of March 2011)	34,653	32,193	5,088
HEIFERS: Must test & all can register if born before or on	December 31, 2011	June 14, 2012	October 4 , 2013
HEIFERS: Only non-carriers can be registered if born on or after	January 1, 2012	June 15, 2012	October 5, 2013
BULLS: Must test & all can register if born before or on	December 31, 2009	June 14, 2010	October 4, 2011
BULLS: Only non-carriers can be registered if born on or after	January 1, 2010	June 15, 2010	October 5, 2011

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Niche Beef Update

Roger Ingram
County Director and Farm Advisor
Placer and Nevada Counties

There is considerable interest in the niche beef business. It is primarily centered on a grass-fed and finished product. The emphasis on grass-fed and finished is because it allows you to differentiate a unique product from what is available at retail stores that are grain finished. Grass-fed and finished beef is a tiny segment of the overall beef industry. Less than ten thousand head of grass-fed and finished beef is raised and marketed in California and that estimate may be too optimistic. This article will update on you on some key issues, marketing tips, and inventory management considerations.

Scale

One of the biggest challenges for any niche beef business is increasing the scale of the operation to an economically viable unit. This can take some time, as there are phases to go through before you can make the decision to expand. The first phase understand your product. What does it taste like? What is the product yield? What will be individual cuts and what will go the hamburger? Where will you get the animal harvested and processed? How long will you age the carcass? Where will you store the meat? What does your market need? Where will you market?

This is further complicated by the biology of a cow. The gestation period is nine months. The time from conception to carcass would be 20-24 months in most cases. One you have answers to the questions above and an analysis shows it can be profitable, Then you could move towards increasing scale – the number of animals marketed in a year. This will then move you towards identifying the needed forage resources to support the operation. Do you have enough irrigated pasture and rangeland? If you expand, do you need more labor?

USDA Inspected Processing

In order to sell individual cuts of meat, you must have USDA inspected processing on both the harvest and processing (cut and wrap). Each individual package will have a safe food handling and processor label containing their identification number. You can get your labels made up that include your name and the processor information. If you ever change processors, you would need to get a new label.

There are few USDA inspected harvest and processing facilities in Northern California. There is great interest in seeing more facilities built or remodeled to meet USDA inspection requirements. The key issue is whether a facility will have enough consistent demand throughout the year to justify the capital investment. If the demand is not there, a facility can quickly experience cash flow issues, as there is not enough income to meet expenses. UC Cooperative Extension is conducting an 18 county survey to better determine demand for USDA inspected harvest and processing. A Rural Business Enterprise Grant is supporting the project.

There is a need for producers to communicate and collaborate with existing processors to help cut costs from both sides. Doing small number of custom work can incur higher costs on the processing side. Coordinating animal delivery and pick-up of meat could potentially cuts costs for producers. It costs just as much to haul one head as it does a full trailer load. Being able to supply a consistent number of animals on a weekly basis is crucial in helping processors schedule help and cut costs.

Marketing and Sales Considerations

- Selling the whole animal – and attaining premium prices for all cuts – is a huge challenge:
- Selling steak cuts – even at very high prices – is not the biggest challenge.
- Selling chuck and rounds is far more difficult.
- Even the better chuck and round cuts can present difficult challenges.
- Otherwise saleable chuck and round cuts often end up in the grinder at plants or at stores.
- If you can find retailers or consumers that will commit moving balanced carcass proportions, it will make and enormous difference in profitability."
- Must insist that retailers take as much of the animal as possible –manage trim.

Education is helpful in creating awareness of product. Education is usually based on casting a very board net, communicating to a broad audience with diverse interests and motivations. However, education does not have a selling message. It only seeks to inform and clarify.

Marketing is different. Marketing consultant Robert Reynolds of Reynolds Consulting noted: "As marketers, our responsibility is to sell products to consumers, over and over and over again. To do that, we provide them information, convey selling messages that direct them to our products, and convince them to buy and to buy again. It is for our benefit. Effective marketing must be laser-focused on selling, not educating. It must convey a selling message to consumers who are prepared to buy. If we do not sell, we have failed."

This is why identifying your target markets is crucial. Each will need unique selling messages. You must focus time and effort on those markets prepared to buy once they are identified.

Other Tips:

- Easy to understand information – Aimed at target customers. Please note that customer might be the end user. It could be an intermediate seller, distributor, restaurant, or retail store.
- Simple, targeted information is essential.
- Point of Sale materials that can be provided to consumers can be helpful.
- Be prepared to find your POS under the counter. You will have to replace it and put in back up.

- Be prepared for turnover of retail meat cutter or distributors and other intermediate sellers.
- Prepare high quality packaging that conveys the brand image, the qualities of the product, and the selling message. This will take time and patience in working with your processor to achieve high quality and visually attractive packaging.
- Be prepared to sell all the way to the ultimate retail consumer - it is the only way to assure the message gets all the way to them.

Inventory Management

Inventory management should be a key area of focus for your operation. You want to guard against inventory continually increasing. For example, you may develop a restaurant customer that wants all of your rib and loin cuts. This will only work if you can find other customers who will take cuts from the chuck, round, and trim which will prove more difficult to sell. Other times, restaurants may be able to buy specific cuts in quantity if see inventory starting to build and you have a good relationship with them.

In 2005, a survey was conducted with four restaurants interested in carrying grass-fed beef on their menu. The following table summarizes demand by cut based on survey results:

Cut	Amount	Frequency	Amount of cut in one beef carcass (lbs) from grass-finished heifers	Number of Animals Needed Weekly to Meet Demand
Flat Iron	50-60 lbs	Weekly	3.6 lbs	14-17 head
Tri-Tip	40 lbs	Weekly	3.3 lbs	12 head
Filet	54 lbs	Weekly	8.5 lbs	7 head

To fill the flat iron order, you would need to have 12-15 animals ready on a weekly basis. This would translate to 48-60 animals per month or 576-720 animals per year. The average retail cut-out on the grass-fed heifers was 322 pounds. For each individual animal, markets would need to be identified for the remaining 307 pounds of meat left the above order.

One Nevada County rancher manages inventory by identifying retail stores that who have butcher shops that can take the entire carcass. He only sells quarters and halves direct to the consumer for the same reason. A Yuba County rancher sell through a meat buyers club and has successfully moved inventory on individual cuts by running specials. One Mendocino County rancher markets through farmers markets by selling a 50 lb box that contains cuts from all primals. They offer a 25 lb box of hamburger. The economic situation in 2011 has turned many people into buying more cuts from the chuck and rump in order to save money. This helps your ability to sell the entire carcass.

The following table shows the percentage of pounds and dollars sold in 2010 through a northern California Meat Buyers Club.

Primal	% Lbs Sold	% of Total Sales Dollars
Chuck	17	15
Rib	7	12
Loin	17	30
Round	7	6
Foreshank, Brisket, Flank, Plate	13	10
Ground Chuck Hamburger	24	19
Other – Stew Meat, Stir Fry, Summer Sausage	11	6
Bones	3	2

The more desirable rib and loin cuts represented 24% of all pounds sold and 42% of the total sales dollars. Ground chuck hamburger made up almost 24% of all pounds sold and 19% of total sales dollars. The same percentage of pounds of chuck was sold as the loin.
You should develop some sort of inventory tracking system to help you know what cuts you are long on and other that you are short. If you know your inventory and have past sales figures, it would allow you to project how long current inventory will last. This can prove helpful in planning harvest dates for your cattle. The system could be as simple as a pad or small notebook or a spreadsheet.

Final Thoughts

Many UC Cooperative Livestock and Natural Resources Advisors have been working in the areas of niche meat marketing. You can use them as an information resource. A new niche beef publication is being developed and hopefully will get published this year. Be sure you have done your planning as you move from learning, to start-up, to expansion to be sure you can be successful. There will be many challenges along the way. There is opportunity as well.

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CALVES (unweaned):		**Age**
Birth:	Tag, Weigh, Nolvasan™ solution on naval if assisted birth or exposed to contaminated areas.	**1 day**
Branding:	Hot Iron "UC"	**2.5 - 5 months**
Castration:	"Knife" Method/ E-Z Bloodless Castrator	**2.5 - 5 months**
Dehorn:	Most are polled	**< 3 mos.**
	Dehorner, Horn Iron, AG Guide	**> 3 mos.**
	Use local anesthesia if greater than 1 month of age	
General:	Provide supplemental selenium to all calves by injection, boluses or supplemental feeding	
	Provide pinkeye vaccine by implant or injection	**2 weeks prior to weaning**
Prophylaxis:		
1st Vaccination	Modified Live vaccine - BVD, IBR, PI3, BRSV (+ Lepto 5-way)(OK because cows have 2 previous Bovishield™)	**2.5 - 5 months**
	Clostridial 8-way	**2.5 - 5 months**
	Histophilus somnii vaccine (e.g. Somnus)	**2.5 - 5 months**
Booster	Modified Live Vaccine - BVD,IBR, PI_3, BRSV (+ Vibrio/Lepto 5-way)	**2 weeks prior to weaning**
	Feedlot Steers and heifers - Mannheimia hemolytica vaccination	**2 weeks prior to weaning**
	Histophilus somnii vaccine (e.g. Somnus)	**2 weeks prior to weaning**
Pink Eye Vaccine	Administer timely - 2nd dose to be administered 3 weeks prior to peak fly season	**Prior to Peak Fly Season**
Anaplasmosis	Anaplasma marginale vaccination - After selection of replacements (for cattle < 11 months of age)	**5-10 months of age**
Anthelmintics	Dectomax™, Ivomec™, Valbazen™ (Rotation suggested)	**Wean/Postwean**
Tetanus	Clostridium tetani antitoxin and vaccination at castration, at separate sites (only if castration is by banding)	**2-3 mos.**
Shipping	Prefer 45 days preconditioning after weaning before shipping.	
Treatments:		
Scours	LA-200™ (Sustained release oxytetracycline) & long acting sulfamethazine 1 - 3 x every other day; Banamine™ daily as necessary. Oral fluids/electroytes if needed	
Pneumonia	LA-200™ (Sustained release oxytetracycline) & long acting sulfamethazine 1 - 3 x every other day; Banamine™ daily as necessary. 2nd treatment -Baytril™ (enrofloxacin) 1x, 3rd treatment Nuflor™ (florfenicol) 1x, 4th treatment Excede™ (ceftiofur)	
Anaplasmosis	LA-200™ (Sustained release oxytetracycline) 1-3x every other day, or Oxytetracycline 3x daily	
Pinkeye	Nuflor™ (florfenicol), 2nd treatment LA-200™ (sustained release oxytetracycline) & Patch 1x	
Coccidiosis	Long acting sulfamethazine 3x every other day; treat group with preventive, if necessary, such as Corid™, Bovatec™, Decox™, and/or Rumensin™	
Abcesses	Drain/Flush/Betadine™ or Nolvasan™ topical/Fly-repellant/PPG if necessary	
Foot Rot	Clean Wound/Nulfor™ (florfenicol),2nd treatment LA200™ (sustained release oxytetracyline)	
	The herd manager, in consultation with the clinical veterinarian, has the discretion to alter timing of vaccinations and other health management activities if conditions are such that using the current protocol may adversely impact animal health. The clinical veterinarian is to be contacted if above treatments do not work.	
	Epinephrine Rx should be on hand for anaphalactic shock.	
	Mineral supplements (including selenium) will be provided year round to ALL animals.	
	Diagnosis of unknown causes of death or illness: Cause of disease to be determined if possible with assistance of Center veterinarian and UCD VMTH and California Animal Health and Food Safety Laboratory	

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CALVES (weanlings and yearlings):		**Age**
Number Brand:	Freeze Brand (Alcohol and dry ice)	**12-24 mos.**
General:	Provide supplemental selenium to all calves by injection, boluses or supplemental feeding	
Prophylaxis:		
Booster	Modified Live Vaccine - BVD,IBR, PI_3, BRSV (+ Vibrio/Lepto 5-way)	**PostWean**
	Feedlot Steers and heifers - Mannheimia hemolytica vaccination	**PostWean**
	Clostridial 8-way (can be given at same time as Brucellosis for heifers)	**PostWean**
	Histophilus somnii vaccine (e.g. somnus)	**PostWean**
Pink Eye Vaccine	Administer timely - 2nd dose to be administered 3 weeks prior to peak fly season	**Prior to Peak Fly Season**
Anaplasmosis	Anaplasma marginale vaccination - After selection of replacements (for cattle < 11 months of age)	**5-10 months of age**
Brucellosis	Brucella abortus (RB-51) vaccine by veterinarian	**5-10 mos.**
Anthelmintics	Dectomax™, Ivomec™, Valbazen™ (Rotation suggested)	**Wean/PostWean**
Coccidiosis	Decox™, Bovatec™, Rumensin™,	**PostWean**
	Medicated Block (free choice)	
Shipping	Prefer 45 days preconditioning after weaning before shipping.	
Treatments:		
Scours	Oral fluids/electroytes and If needed, LA-200™ & long acting sulfamethazine 1 - 3 x every other day; Banamine™ daily for up to 3 days	
Pneumonia	LA-200™ (Sustained release oxytetracycline) & long acting sulfamethazine 1 - 3 x every other day; Banamine™ daily as necessary. 2nd treatment -Baytril™ (enrofloxacin) 1x, 3rd treatment Nuflor™ (florfenicol) 1x, 4th treatment Excede™ (ceftiofur)	
Anaplasmosis	LA-200™ (Sustained release oxytetracycline) 1-3x every other day, or Oxytetracycline 3x daily	
Pinkeye	Nuflor™ (florfenicol), 2nd treatment LA-200™ (sustained release oxytetracycline) & Patch 1x	
Coccidiosis	Long acting sulfamethazine 3x every other day; treat group with preventive, if necessary, such as Corid™, Bovatec™, Decox™, and/or Rumensin™	
Abcesses	Drain/Flush/Betadine™ or Nolvasan™ topical/Fly-repellant/PPG if necessary	
Foot Rot	Nuflor™(Florfenicol), 2nd treatment LA-200™ (sustained release oxyteracycline), or 3rd treatment Oral Sulfa/PPPG	
	The herd manager, in consultation with the clinical veterinarian, has the discretion to alter timing of vaccinations and other health management activities if conditions are such that using the current protocol may adversely impact animal health. The clinical veterinarian is to be contacted if above treatments do not work.	
	Epinephrine Rx should be on hand for anaphalactic shock.	
	Mineral supplements (including selenium) will be provided year round to ALL animals.	
	Diagnosis of unknown causes of death or illness:	
	Cause of disease to be determined if possible with assistance of Center veterinarian and UCD VMTH and California Animal Health and Food Safety Laboratory	

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COWS (Fall Calvers):		**Month**
General:	Provide supplemental selenium to all cows by injection, boluses or supplemental feeding	
Prophylaxis:		
PreBreeding vaccinati	If open - Modified Live vaccine - BVD, IBR, PI3, BRSV + Vibrio/Lepto 5-way + (Spirovac™)	**Nov.**
Annual Re-vaccination	Clostridial 8-way	**Nov/Dec**
Anthelmintics	Dectomax™, Ivomec™ +, or Valbazen™ (Rotation suggested)	**May**
Calf Scours	If pregnant - E. coli, Rota virus, Corona virus vaccine (Scourguard™)	**Prior to calving**
	(Only in years following high incidence of calf scours)	
Treatments:		
Anaplasmosis	LA-200™ (Sustained release oxytetracycline) 1-3x every other day, or Oxytetracycline 3x daily (Only if cattle can be restrained without overexciting them)	
Foot Rot	Clean Wound/Oral Sulfa/PPG,Nuflor™ (IM), Banamine™	
Pinkeye	Eye patch 1x/Florfenicol (Nuflor™) if needed, Banamine™	
Pneumonia	Florfenicol (Nuflor™), Ceftiofur (Excede™)	
Calving:		
Dystocia	Assist as necessary; most difficult cases to VMTH in Davis	
Prolapse	1. "Shrink" Tissue w/powdered sugar - IF NEEDED	
	2. Re-place tissue	
	2a. Epidural - Lidocaine™	
	3. Pin vulva if necessary	
	4. Administer systemic penicillin	
	Retreat as needed	
	5. Remove pins	
	6. Cull cow and, where possible, offspring	
Metritis &		
Retained Placenta	1. Systemic Penicllin	
	2. Administer Lutalyse™	
	Repeat steps 1 & 2 as needed	
Pregancy Exam:		
	45 days after bulls removed from cows	**Post Wean**
	If substantial abortion problem, boost w/modified live vaccine - BVD,IBR,Pis,BRSV + Vibrio/Lepto 5 way + (Spirovac ™)	
	The herd manager, in consultation with the clinical veterinarian, has the discretion to alter timing of vaccinations and other health management activities if conditions are such that using the current protocol may adversely impact animal health. The clinical veterinarian is to be contacted if above treatments do not work.	
	Epinephrine Rx should be on hand for anaphalactic shock.	
	Mineral supplements (including selenium) will be provided year round to ALL animals.	
	Diagnosis of unknown causes of death or illness:	
	Cause of disease to be determined if possible with assistance of Center veterinarian and UCD VMTH and California Animal Health and Food Safety Laboratory	

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COWS (Spring Calvers):		**Month**
General:	Provide supplemental selenium to all cows by injection, boluses or supplemental feeding	
Prophylaxis:		
PreBreeding vaccinati	If open - Modified Live vaccine - BVD, IBR, PI3, BRSV + Vibrio/Lepto 5-way + (Spirovac™)	**May**
Annual Re-vaccination	Clostridial 8-way	**May/June**
Anthelmintics	Dectomax™, Ivomec™ +, or Valbazen™ (Rotation suggested)	**Nov.**
Calf Scours	If pregnant - E. coli, Rota virus, Corona virus vaccine (Scourguard™) (Only in years following high incidence of calf scours)	**Prior to calving**
Treatments:		
Anaplasmosis	LA-200™ (Sustained release oxytetracycline) 1-3x every other day, or Oxytetracycline 3x daily (Only if cattle can be restrained without overexciting them)	
Foot Rot	Clean Wound/Oral Sulfa/PPG,Nuflor™ (IM), Banamine™	
Pinkeye	Eye patch 1x/Florfenicol (Nuflor™) if needed, Banamine™	
Pneumonia	Florfenicol (Nuflor™), Ceftiofur (Excede™)	
Calving:		
Dystocia	Assist as necessary; most difficult cases to VMTH in Davis	
Prolapse	1. "Shrink" Tissue w/powdered sugar - IF NEEDED	
	2. Re-place tissue	
	2a. Epidural - Lidocaine™	
	3. Pin vulva if necessary	
	4. Administer systemic penicillin	
	Retreat as needed	
	5. Remove pins	
	6. Cull cow and, where possible, offspring	
Metritis &		
Retained Placenta	1. Systemic Penicllin	
	2. Administer Lutalyse™	
	Repeat steps 1 & 2 as needed	
Pregancy Exam:		
	45 days after bulls removed from cows	**Post Wean**
	If substantial abortion problem, boost w/modified live vaccine - BVD,IBR,Pis,BRSV + Vibrio/Lepto 5 way + (Spirovac ™)	
	The herd manager, in consultation with the clinical veterinarian, has the discretion to alter timing of vaccinations and other health management activities if conditions are such that using the current protocol may adversely impact animal health. The clinical veterinarian is to be contacted if above treatments do not work.	
	Epinephrine Rx should be on hand for anaphalactic shock.	
	Mineral supplements (including selenium) will be provided year round to ALL animals.	
	Diagnosis of unknown causes of death or illness:	
	Cause of disease to be determined if possible with assistance of Center veterinarian and UCD VMTH and California Animal Health and Food Safety Laboratory	

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Bulls		**Month**
General:	Provide supplemental selenium to all bulls by injection, boluses or supplemental feeding	
Prophylaxis:		
PreBreeding	Modified Live Virus vaccine - BVD, IBR, PI3, BRSV + Vibrio/Lepto 5-way + Spirovac™	**Dec.**
	Clostridial 8-way	**Dec.**
Anthelmintics	Valbazan™/Ivomec Plus™/Dectomax™ (Rotation suggested)	**May**
Reproductive	Tract exam and semen test (BSE),	**Nov.-Dec.**
	Tritrichomonas testing	
Treatment:		
Anaplasmosis	LA-200™ (Sustained release oxytetracycline) 1-3x every other day, or Oxytetracycline 3x daily (Only if cattle can be restrained without over exciting them)	
Foot Rot	Clean Wound/Oral Sulfa/PPG,Nuflor™ (IM), Banamine™	
Pinkeye	Eye patch 1x/Florfenicol (Nuflor™) if needed, Banamine™	
REPLACEMENT HEIFERS (Fall Calvers):		
General:	Provide supplemental selenium to all heifers by injection, boluses or supplemental feeding	
Prophylaxis:		
PreBreeding	Modified Live Vaccine - BVD, IBR, PI3, BRSV + Vibrio/Lepto 5-way + Spirovac™	**Oct.**
Booster	Modified Live Vaccine - BVD, IBR, PI3, BRSV + Vibrio/Lepto 5-way + Spirovac™	**Nov.**
Booster	Clostridial 8- way	**Oct/Nov.**
Reproductive	Tract score - DVM	**Dec.**
REPLACEMENT HEIFERS (Spring Calvers):		
General:	Provide supplemental selenium to all heifers by injection, boluses or supplemental feeding	
Prophylaxis:		
PreBreeding	Modified Live Vaccine - BVD, IBR, PI3, BRSV + Vibrio/Lepto 5-way + Spirovac™	**April**
Booster	Modified Live Vaccine - BVD, IBR, PI3, BRSV + Vibrio/Lepto 5-way + Spirovac™	**May**
Booster	Clostridial 8- way	**April/May**
Reproductive	Tract score - DVM	**June**
	The herd manager, in consultation with the clinical veterinarian, has the discretion to alter timing of vaccinations and other health management activities if conditions are such that using the current protocol may adversely impact animal health. The clinical veterinarian is to be contacted if above treatments do not work.	
	Epinephrine Rx should be on hand for anaphalactic shock.	
	Mineral supplements (including selenium) will be provided year round to ALL animals.	
	Diagnosis of unknown causes of death or illness:	
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HORSES		**Month**
Vaccinations	E/W/V Encephalomyelitis, Tetanus, Influenza, West Nile and Rabies	**February - 1x a year**
	Rhinopneumontis EHV1 & EHV4 - as needed for newly introduced, young (< 5 years old)	**As Needed**
	and pregnant mares	
	(Need booster 3-4 weeks after 1st vaccine on all)	
Anthelmintics	Pyrantel Pamoate	**August and February**
	Ivermectin	**October and April**
	Benzimindazole	**December and June**
	The herd manager, in consultation with the clinical veterinarian, has the discretion to alter timing of vaccinations and other health management activities if conditions are such that using the current protocol may adversely impact animal health.	
	Epinephrine Rx should be on hand for anaphalactic shock.	
	Free choice salt provided.	
	Mineral supplements will be provided year round to ALL animals.	
	Diagnosis of unknown causes of death or illness:	
	Cause of disease to be determined if possible with assistance of Center veterinarian and UCD VMTH and California Animal Health and Food Safety Laboratory	

VET VIEWS, *from the University of California, Davis*

Are You Certified?
Beef Quality Assurance Program reminds of good practices

By John Maas, DVM, MS, DACVN, DACVIM, Extension Veterinarian, University of California, Davis

Over the past three years, we have certified nearly 500 producers in California's Cow-Calf Beef Quality Assurance (BQA) Program. These have been put on to certify producers for the first time as well as for recertification to comply with national guidelines to recertify on a three-year basis.

So, if you were one of the nearly 200 producers who attended a BQA program in 2008, you will need to certify again this year to stay current in the program.

As we start another round of BQA certifications in 2011, and as many California producers are re-examining their cattle health regimen before brandings this spring, I'd like to discuss some factors that affect the immune response of the calves when we vaccinate them as part of the BQA practices we all put in place.

A large part of BQA is prevention of disease so the animals do not have to be treated after weaning, shipping or in the feedlot. Vaccination against the common diseases is an important part of disease prevention. For vaccination and the animal's immune response to work appropriately a number of steps (links in the chain) have to all work together.

First, we need to present an appropriate antigen (vaccine) to the animal via injection or intranasal inoculation. The vaccine antigen has to have been properly stored and mixed before presentation.

Secondly, the animal's immune response has to respond to the vaccine to produce protective immunity. Many factors are important in the proper working of the immune response; but, two of the most important are trace mineral nutrition and parasitism. So what are some of the weak links in the chain of protecting cattle from disease and what can we do about them?

Can storage conditions affect vaccines?

Definitely! Most vaccines should be stored at 35-45º F. The recommended storage conditions are on the vaccine label. Vaccines should be stored at the recommended temperatures from the time they are manufactured until the time you use them chute side.

Overheating vaccines can cause obvious problems, as the proteins in the vaccine will breakdown (denature) and will not produce the desired immune response. Worse than overheating, freezing vaccines will decrease their effectiveness even faster. So the recommended storage conditions of 35-45º F is a strict range on both ends.

Both modified live vaccines and killed vaccines are affected by improper storage temperatures. Nearly all killed vaccines contain an adjuvant that aids in the immune response, as do some live vaccines. High or low storage temperatures cause these mixtures to separate and lose their effectiveness to prevent disease.

What about refrigerators and their effectiveness?

It turns out that many of the refrigerators we use for storing cattle vaccines and drugs are cast-offs from some other use and may not be functioning properly. A survey by David Thain, DVM, at the University of Nevada, Reno, Reno, Nev., found 25 percent of ranch refrigerators failed to maintain temperatures to keep vaccines in the safe range and most of the failures had to do with freezing the vaccines (temperatures as low as 10º F for extended periods).

Many of the old refrigerators we use tend to freeze items stored in the back near the coils and overheat items stored near the front or in the door because the rubber seals no longer work. Additionally, some of the older refrigerators cannot insulate well enough when placed outside in the winter and the vaccines simply freeze inside the refrigerator.

How can I tell if my refrigerator is working correctly?

You can buy a thermometer which records minimum and maximum temperatures and place it in your refrigerator for several days. Put it in different locations to be sure you don't have cold spots or hot spots. These thermometers can be purchased for less than $20. They are made in both electronic form and magnetic form (the high/low thermometers we use to record daily temperatures).

Simply go on the Internet and type in recording thermometers and browse the various offerings until you find one that suits your needs. Leave a thermometer in your storage refrigerator and monitor it from time to time.

What affects the immune response of the calves?

Many things will affect the calves' ability to respond to vaccines or disease challenges. These include the presence of the BVD virus in the herd, stress, previous vaccination history, the products used, parasites, nutrition and vaccine handling. The storage of vaccines was discussed above, as an important part of vaccine handling.

Cattle that are parasitized do not respond normally to vaccines. Also, calves deficient in trace minerals such as selenium (Se) or copper (Cu) respond poorly to vaccines and infectious diseases.

Why do parasites affect the immune system?

Most parasites have complex life cycles and depend on evading the host's defenses to be able to survive. Part of this evasion is to affect the host's immune system, so there is not a large reaction against the parasites. Therefore, most parasites make chemicals that decrease the animal's ability to make a full immune response. While this helps the parasite, it harms the host in terms of handling other infections.

How do you minimize the parasites' damage?

Having a comprehensive parasite control program for the entire herd is the first step. Deworming the cow herd before they enter clean pastures will help keep the parasite load low in the herd and keep the number of parasite eggs on the pastures to a minimum. The use of effective products will also have a positive impact. Your veterinarian can advise you on the use of appropriate products and the timing of use in your herd.

What are the best dewormers to use?

Again, your veterinarian can best advise you on this topic for your herd's particular situation. However, I recommend using the brand name products at this time. There have been a number of situations in the recent past where generic ivermectin products have been associated with significant parasitism, i.e., the generic ivermectin were used and clinical problems remained.

If you have any indication that a dewormer did not work have your veterinarian investigate the problem and analyze fecal samples to determine if patent parasitic infections are present.

How do trace minerals affect the animal's immune system?

Many trace minerals and vitamins are now referred to as "antioxidants". As part of their antioxidant function they are very important in the immune system. In California, most of the beef cattle are deficient in either Se or Cu (or often both) unless they are supplemented. These minerals are very important in the calves' immune system for a normal response to vaccines and to ward off diseases such as pneumonia. Also, neither Se nor Cu are well transferred to the calf via milk, so near weaning the calves are often at their lowest level in terms of Se and Cu – at greatest risk for deficiency.

How do I know if my calves are deficient?

A few blood samples taken from your calves at or near weaning will tell the story. They can be analyzed for the trace minerals and will reflect how well your supplementation program is working. There are a number of effective ways to supplement Se and Cu and your veterinarian can help you work through the options that will work best for you.

What's the bottom line?

With regard to handling vaccines, make sure your refrigerator and vaccine storage system is working. Be sure to store your vaccines and other animal health materials according to label instructions. For vaccines this is usually between 35 and 45º F.
For parasite control, use brand name products recommended by your veterinarian at an appropriate time to make sure calves to be vaccinated are not heavily parasitized.

With regard to trace minerals, have a good supplementation program that includes occasional monitoring of calves' blood levels to be sure the program is working efficiently. Strengthen these weak links and combine them with good BQA practices and your calves will be healthier with minimal disease problems.

If you are due to recertify in BQA or you need to certify for the first time, contact Stevie Ipsen in the CCA office at (916) 444-0845 or by e-mail at stevie@calcattlemen.org to find out about an upcoming certification program near you.

Making a Difference for California
University of California Agriculture and Natural Resources
UC PEER REVIEWED
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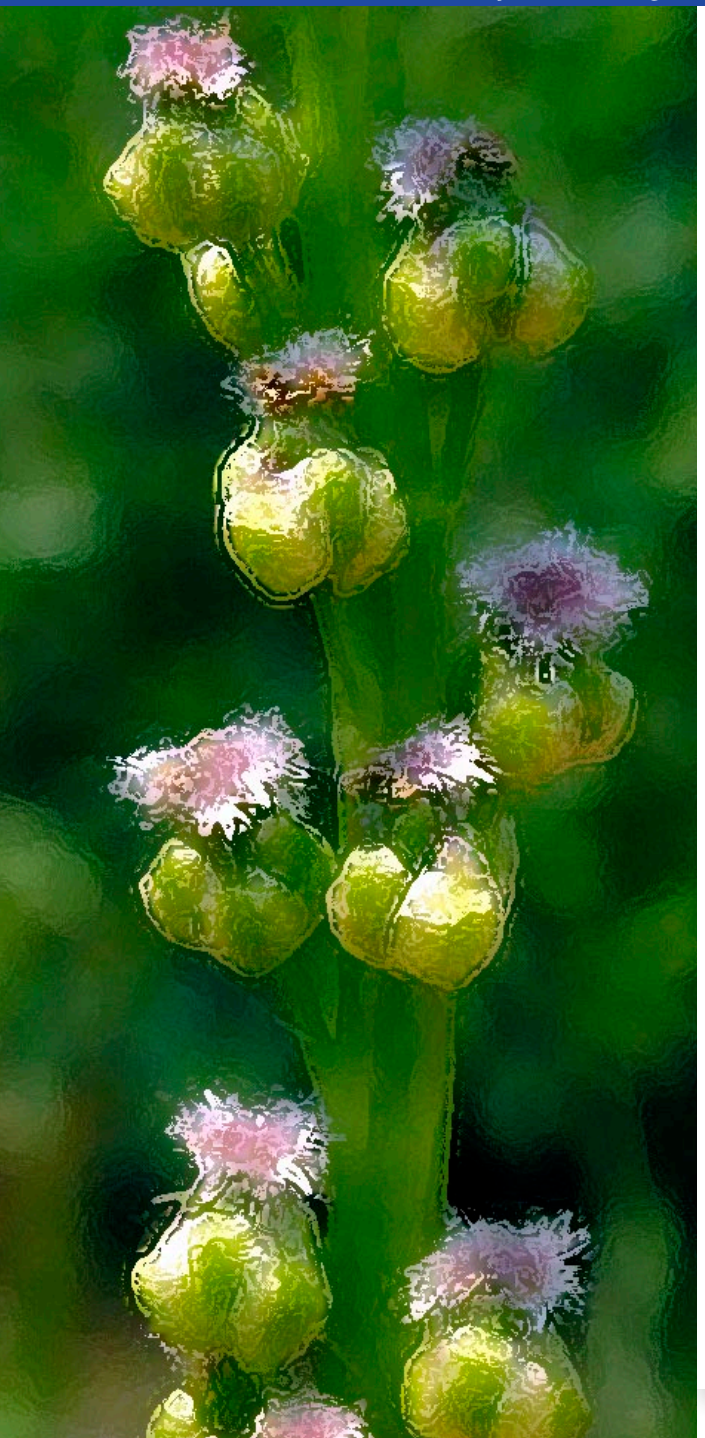

Livestock-Poisoning Plants of California

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Poisonous plants cause significant losses of livestock every year. A successful livestock operator must know which poisonous plants occur on a given range or pasture and how they can be controlled or avoided. This publication shows which plants are poisonous, tells how they affect stock, and suggests ways to reduce losses from poisoning.

Undesirable effects may result from a single ingestion of a large amount of a poisonous plant, but some plants are so toxic that very small amounts may result in severe disease or death. Other plants cause chronic poisoning only after ingestion over weeks or months. The later situation may result in clinical signs long after the exposure to the toxic plant material, and treatment may no longer be possible.

With few exceptions, livestock will not eat poisonous plants unless forced to by hunger. The single most important way to prevent poisoning is to use proper range and pasture management practices to provide ample forage, encouraging consumption of nontoxic plants. Areas infested with poisonous plants should be avoided when trailing, holding, or unloading animals. Supplemental feed may protect stock if these conditions cannot be avoided, but there are circumstances (for example, herbicide applications) that may change palatability or increase toxicity in some plants. If toxic weeds are embedded in alfalfa cubes or included in total mixed rations, animals may not be able to avoid ingestion of them.

Many poisonous plants may be controlled with herbicides. Often, however, the uneven distribution

What to do if you suspect that a plant in your hay or feed is poisoning your animals

- Work with your veterinarian to determine whether the animals' clinical sign may be a result of plant exposure.
- Collect the whole plant (if practical) or representative parts of the plant, including leaves, flowers, stems, roots, and fruit, and have it identified by a trained individual (local nursery, county agricultural commissioner, UC Cooperative Extension Advisor, veterinarian, diagnostic laboratory, etc.).
- Save suspect plant material or feed for possible future evaluation.
- Once plants are identified, ask your veterinarian to contact a veterinary toxicologist for detailed consultation.
- If the animal dies, submit it to a veterinary diagnostic laboratory.

of poisonous plants on a range or in a pasture makes large-scale chemical control uneconomical. However, small patches of poisonous plants can and should be eradicated to prevent them from spreading to other areas. The specifics of chemical control of poisonous plants are beyond the scope of this publication; see your county Farm Advisor or a UC Cooperative Extension Specialist, or contact a pest control adviser (PCA) for specific recommendations for your area.

Diagnosing a plant poisoning can be difficult and must be done quickly. In many cases, clinical signs are nonspecific (such as diarrhea), and postmortem lesions are not characteristic. The assistance of your veterinarian is crucial. This is especially important in insurance or legal investigations. Producers and farm managers, along with veterinarians and diagnosticians, play important roles, and all contribute information that may be important to diagnose a poisoning case. Once all the information is available, all evidence is collected, and proper sampling of specimens has occurred, a summary of findings will be instrumental in preventing recurrences.

Accurate plant identification is critical if you suspect that a particular plant is causing problems for your livestock. Producers can be most helpful by providing a grazing history, carefully observing what plants have been grazed, especially suspicious plants. The best approach is to collect the whole plant (if practical) or representative parts of the plant, including leaves, flowers, stems, roots, and fruit, and press them dry in a folded newspaper between two sheets of cardboard, or roll them up between the pages of a newspaper or magazine. The plant sample can then be taken or sent to your local UC Cooperative Extension advisor or county agricultural commissioner office for identification. Local nurseries may also be a good source for plant identification. To determine whether a particular plant is found in your area, see the Calflora Web site, http://www.calflora.org/.

Livestock Poisoning by Plants in California

The California Animal Health and Food Safety (CAHFS) Laboratory System toxicology laboratory investigated numerous cases of suspected plant poisonings from 1990 to 2007. CAHFS veterinary toxicologists have diagnosed plants as the cause of toxicosis in more than 600 submitted cases, most of these in livestock. The largest number of submissions was for cattle, followed by horses, pigs, goats, and sheep.

The most commonly diagnosed cause of plant poisoning is ornamental oleander, a nonnative species (fig. 1). The data from CAFHS are from submitted samples, so they do not necessarily represent the overall occurrence of plant poisonings in California.

What to do if you suspect that a plant in your pasture is poisoning your animals

1. Work with your veterinarian to determine whether the animal's clinical sign may be a result of plant exposure.
2. Determine whether a plant is found in your area. Visit the Calflora Web site, http://www.calflora.org/.
3. Walk the field, collecting any unusual or toxic plants, and have them identified.
4. If you cannot identify a suspect plant, take it to your county agricultural commissioner or UC Cooperative Extension office for identification.
5. If an animal dies, submit it to a veterinary diagnostic lab.

Although most of the plants discussed in this publication are not among those that have confirmed diagnoses, it is important to know that they can be poisonous. Awareness of poisonous plants growing in a certain geographical region, the season when they are most available, and their associated clinical signs are instrumental in making a diagnosis and initiating treatment. Most important, recognition of poisonous plants in hay or forage may help prevent plant poisonings in animals.

The following tables give summary information about livestock-poisoning plants in California. For full information on plants listed, refer to the descriptions in the text.

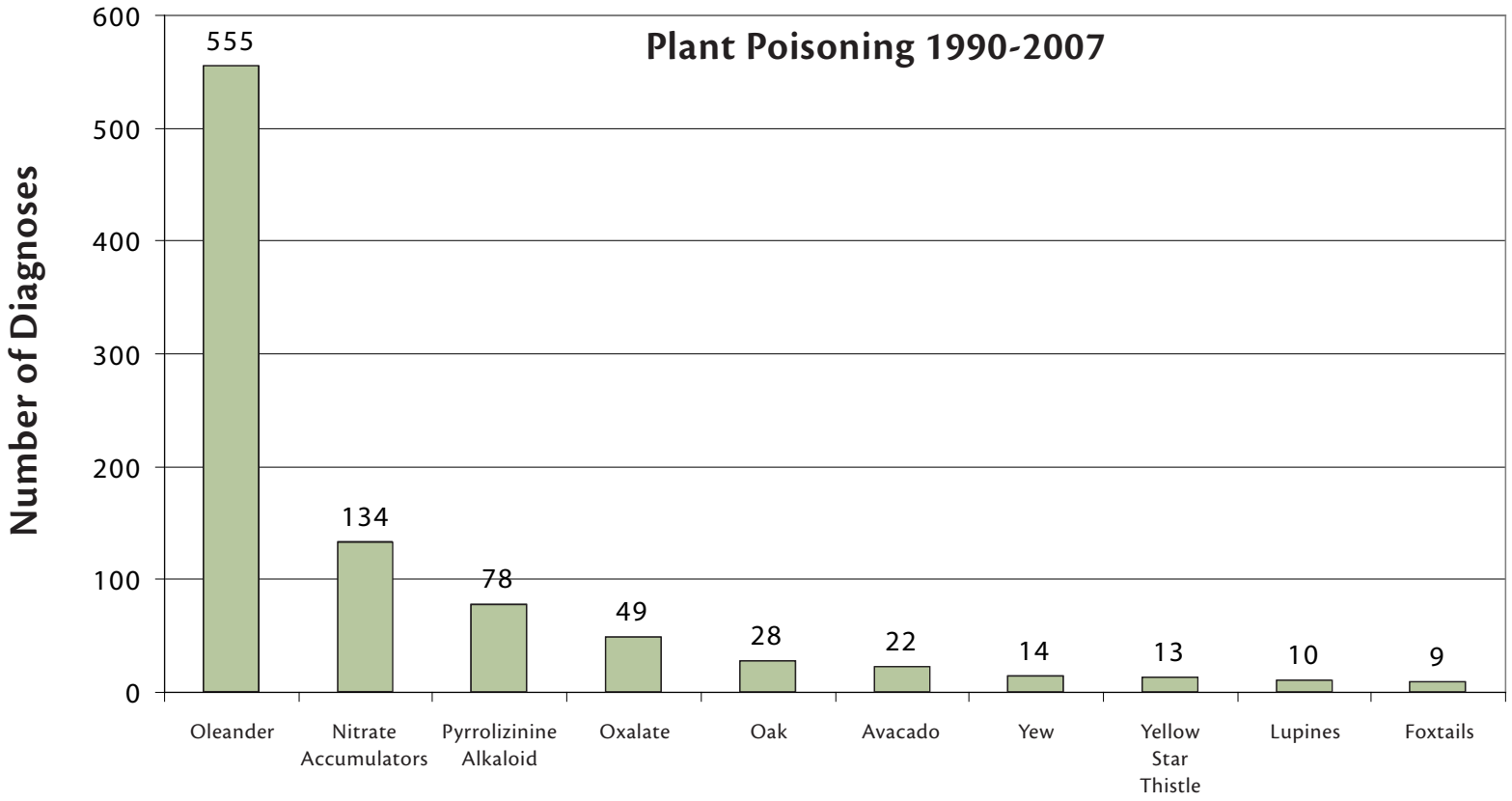

Figure 1. Sources of plant poisoning in livestock, 1990–2007. *Source:* CAHFS.

Table 1. Livestock-poisoning plants commonly found in selected regions of California

Common name	Scientific name
North Coast	
arrowgrass	*Triglochin* spp.
azalea	*Rhododendron* spp.
chokecherry	*Prunus virginiana*
dogbane	*Apocynum* spp.
fiddleneck	*Amsinckia* spp.
milkweed	*Asclepias* spp.
ragweed or ragwort	*Senecio* spp.
rhododendrum	*Rhododendron* spp.
ryegrass, perennial	*Lolium* spp.
tansy ragwort	*Senecio* spp.
veratrum (false hellebore)	*Veratrum californicum*
water hemlock	*Cicuta douglasii, C. maculata*
South Coast	
arrowgrass	*Triglochin* spp.
chokecherry	*Prunus virginiana*
dogbane	*Apocynum* spp.
fiddleneck	*Amsinckia* spp.
locoweed	*Astragalus* spp.
milkweed	*Asclepias* spp.
tree tobacco	*Nicotiana* spp.
Sacramento Valley	
cocklebur	*Xanthium spinosum, X. strumarium*
curly dock	*Rumex crispus*
dogbane	*Apocynum* spp.
fiddleneck	*Amsinckia* spp.
foxtail	*Hordeum* spp. and *Setaria* spp.
groundsel	*Senecio* spp.
milkweed	*Asclepias* spp.
oak	*Quercus* spp.
oleander	*Nerium* spp.
ragweed or ragwort	*Senecio* spp.
starthistle	*Centaurea* spp.
tree tobacco	*Nicotiana* spp.
water hemlock	*Cicuta douglasii, C. maculata*
San Joaquin Valley	
cocklebur	*Xanthium spinosum, X. strumarium*
curly dock	*Rumex crispus*
fiddleneck	*Amsinckia* spp.
foxtail	*Hordeum* spp. and *Setaria* spp.
groundsel	*Senecio* spp.
locoweed	*Astragalus* spp.
milkweed	*Asclepias* spp.
oak	*Quercus* spp.
oleander	*Nerium* spp.
ragweed or ragwort	*Senecio* spp.
starthistle	*Centaurea* spp.
tree tobacco	*Nicotiana* spp.
water hemlock	*Cicuta douglasii, C. maculata*
Sierra Foothills	
cocklebur	*Xanthium spinosum, X. strumarium*
deathcamas	*Zigadenus* spp.
dogbane	*Apocynum* spp.
foxtail	*Hordeum* spp. and *Setaria* spp.
klamathweed	*Hypericum perforatum*
larkspur (low)	*Delphinium* spp.
milkweed	*Asclepias* spp.
oak	*Quercus* spp.
ragweed or ragwort	*Senecio* spp.
toyon	*Heteromeles arbutifolia*
tree tobacco	*Nicotiana* spp.
western azalea	*Rhododendron* spp.
Sierra Nevada Northern Range	
chokecherry	*Prunus virginiana*
dogbane	*Apocynum* spp.
lupine	*Lupinus* spp.
Ponderosa pine	*Pinus ponderosa*
Sierra Nevada Central Range	
chokecherry	*Prunus virginiana*
deathcamas	*Zigadenus* spp.
dogbane	*Apocynum* spp.
larkspur (tall)	*Delphinium* spp.
Ponderosa pine	*Pinus ponderosa*
ragwort or ragwort	*Senecio* spp.
toyon	*Heteromeles arbutifolia*
veratrum (false hellebore)	*Veratrum californicum*
water hemlock	*Cicuta douglasii, C. maculata*
western azalea	*Rhododendron* spp.
western bracken fern	*Pteridium aquilinum*
East of the Sierra Nevada	
deathcamas	*Zigadenus* spp.
greasewood	*Sarcobatus vermiculatus*
horsetail and scouring rushes	*Equisetum* spp.
Ponderosa pine	*Pinus ponderosa*
ragweed or ragwort	*Senecio* spp.
summer pheasant's eye	*Adonis aestivalis*
Southern California	
avocado	*Persea americana*
chokecherry	*Prunus virginiana*
locoweed	*Astragalus* spp.
ragweed or ragwort	*Senecio* spp.
tree tobacco	*Nicotiana* spp.

Table 2. Most commonly diagnosed plant poisonings for selected livestock, in descending order of occurrence

Cattle	Sheep	Horses	Goats
oleander	oleander	oleander	avocado
nitrate/nitrite*	oxalate‡	pyrrolizidine alkaloids†	nitrate/nitrite
oxalate	nitrate/nitrite	yellow starthistle	oxalate
pyrrolizidine alkaloids	lupine	dogbane	lupine
oak	perennial ryegrass	foxtail	oleander

Source: CAHFS.

Notes:

*Nitrate/nitrite: Present in johnsongrass *(Sorghum halapense)* and sudangrass (*S.bicolor*); oat hay and other grass hays; lambsquarters and goosefoot (*Chenopodium* spp.); and pigweed (*Amaranthus* spp.).

†Pyrrolizidine alkaloids: Present in fiddleneck (*Amsinckia* spp.), tansy ragwort *(Senecio jacobaea)*, and groundsel (*Senecio* spp.).

‡Oxalate: Present in greasewood *(Sarcobatus vermiculatus)*; sorrel (*Oxalis* spp.); dock (*Rumex* spp.); pigweed (*Amaranthus* spp.); and lambsquarter and goosefoot (*Chenopodium* spp.).

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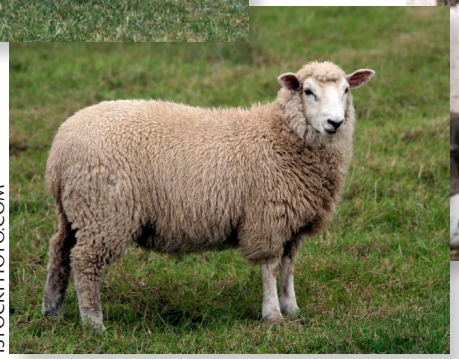

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Table 3. Toxic dosage for selected livestock-poisoning plants in California

Plant	Toxic dosage			
	Cattle	Sheep	Horses	Comments
arrowgrass	1.5% bw	5–2% bw	—	Flowering spikes have twice the amount of toxin and require one-half the dosage.
avocado	5.5 g/kg bw			
cocklebur	1% bw	1.5% bw	—	Consumption of fresh green sprouts.
	0.75% bw	—	—	Consumption of germinating plants (lethal).
deathcamas	1% bw	—	—	Consumption of green plant (lethal).
dogbane	NA	NA	NA	No conclusive toxic dosage information available.
foxtail	NA	NA	NA	
greasewood	1.5% bw	—	—	Toxic if eaten quickly.
groundsel and ragwort	5–10% bw‡	—	3–7% bw	Requires a total intake of 25 to 50% bw over several months
klamathweed	1% bw*	2.5 kg/45	—	—
locoweed	30–50% bw in dw	—	81 lb†	Symptoms appear 2 to 9 weeks after consumption.
lupine	NA	NA	NA	Depends on lupine species, plant phenology, time of consumption, and species of animal.
oak	NA	NA	NA	Depends on oak species, plant phenology, time of consumption, and species of animal.
oleander	5 leaves	5 leaves	—	—
pine needles	2.2 to 2.7 kg	—	—	Consumption per day for more than 3 days (abortion).
poison hemlock	2 to 0.5% bw	0.6–0.8% bw	—	Seed heads are more toxic than other plant parts.
tobacco	2% bw	—	—	Consumption of this level of fresh plant is lethal.
water hemlock	1–12 oz	2 oz	8 oz	Consumption of roots.
western bracken fern	—	—	3–5%	Consumption for at least 30 days.
yellow starthistle	—	—	600 lb dm	Consumption over 1 to 3 months.
Milkweed common name	***Asclepias* species name**		**Estimated toxic dosage of bw for sheep, cattle, and horses**	
antelope horns	*A. asperula*		>1%	
broadleaf	*A. latifolia*		0.2–0.4%	
Indian	*A. eriocarpa*		0.2%	
narrow-leaved	*A. fascicularis*		0.5–1%	
showy	*A. speciosa*		1–2%	

Source: Burrows and Tyrl 2001.
Notes:
bw = body weight
dm = dry matter
*500- to 700-pound calves.
†Consumed over 4 to 6 weeks.
‡This level consumed in a few days or weeks causes acute liver disease.

Table 4. Signs of poisoning and plants commonly responsible

CATTLE			
Plant	**Toxin**	**Organ or systems affected**	**Signs**
arrowgrass	cyanide	blood, all organs	**sudden death**, salivation, heavy breathing
chokecherry	cyanide	cell poison, all organs	**sudden death,** salivation, heavy breathing
cocklebur	glycosides	cell poison, all organs	**sudden death**, weakness, inappetance, incoordination, muscle spasms, convulsions, coma
curly dock	oxalates, nitrate	kidney, blood	**sudden death**, salivation, depression, incoordination, muscle tremors, weakness, coma
deathcamas	alkaloids	nervous	**sudden death**, excessive salivation, lowered temperature, staggering, weakness, coma
dogbane/Indian hemp	cardiac glycosides	heart	**sudden death**, diarrhea, weakness, irregular heartbeat
fiddleneck	pyrrolizidine	liver	chronic: appetite loss, **weight loss**, appetite loss, rectal straining
foxtails	physical	mouth, eyes, skin	**ulceration** and lesions in mouth, tongue, gums, eyes
greasewood	oxalates	kidney	**sudden death**, depression, incoordination, weakness, coma
groundsel	pyrrolizidine	liver	chronic: appetite loss, **weight loss**, rectal straining
horsetail	thiaminase	nervous	**thiamine deficiency**, depression, unsteady gait, incoordination, weakness, seizures, and diarrhea
klamathweed	hypericin	skin	**photosensitization;** sunburn of lips, mouth, and unpigmented skin; blistering, loss of condition
larkspur (tall)	alkaloids	nervous	**sudden death**, tremors, weakness, **incoordination**, staggering, slobbering, bloat
locoweed	alkaloids	nervous	difficulty eating, exaggerated mouth movement, **excitability;** depression, weakness, weight loss, incoordination
lupine	alkaloids	nervous, reproductive	**birth defects**, abortion, tremors, incoordination, head pressing, seizures
milkweed	cardiac glycosides	gastrointestinal, heart, nervous	**depression, diarrhea**, colic, irregular respiration
nightshades	alkaloids	gastrointestinal	**gastrointestinal upset**, constipation or diarrhea, drowsiness, salivation, weakness
oak	tannins	liver, kidney	**sudden death**, bloody diarrhea, kidney failure
oleander	cardiac glycosides	heart	**sudden death**, depression, salivation, weakness, irregular heartbeat, diarrhea
poison hemlock	coniine (alkaloids)	nervous, reproductive	nervousness, trembling, weakness, coma, **birth defects**
Ponderosa pine	isocupressic acid	reproductive	**abortion**, incoodrination, aimless wander, congenital defects
ryegrass	alkaloids (ergot)	nervous	**incoordination**, staggering
summer pheasant's eye	glycosides	gastrointestinal	**gastrointestinal disturbance**, diarrhea, decreased gut motility, sudden death
tobacco	alkaloids (nicotine)	nervous, reproductive	**birth defects**, abortion, weakness, staggering
toyon	cyanide	cell poison, all organs	sudden death
veratrum	alkaloids	nervous, reproductive	**birth defects**, abortion; labored breathing, salivation, convulsions
water hemlock	alcohols	nervous	**sudden death**, muscle spasms, teeth grinding, severe convulsions
western bracken fern	glycosides	bone marrow, bladder	**bone marrow depression** signs; hemmorhage, bloody feces, bloody nasal secretions; **bladder tumors**; pink, red, brown urine

Note: The signs listed in this table are commonly seen after the specific plant intoxications. However, other signs may predominate depending on the dosage, duration of exposure, and environmental factors. Diagnosis must be done by a licensed veterinarian and may require toxiciological analysis by a diagnostic laboratory to confirm the plant involved. Primary or usual signs are given in bold.

Table 4. *Continued*

SHEEP			
Plant	**Toxin**	**Organ or systems affected**	**Signs**
arrowgrass	cyanide	blood, all organs	**sudden death**, salivation, heavy breathing
chokecherry	cyanide	cell poison, all organs	**sudden death,** salivation, heavy breathing
cocklebur	glycosides	cell poison, all organs	**sudden death**, weakness, inappetance, incoordination, muscle spasms, convulsions, coma
curly dock	oxalates, nitrate	kidney, blood	**sudden death**, salivation, depression, incoordination, muscle tremors, weakness, coma
deathcamas	alkaloids	nervous	**sudden death**, excessive salivation, lowered temperature, staggering, weakness, coma
dogbane/Indian hemp	cardiac glycosides	heart	**sudden death**, diarrhea, weakness, irregular heartbeat
fiddleneck	pyrrolizidine	liver	**weight loss**, appetite loss [sheep are more resistant to poisoning by the pyrrolizidine alkaloids than cattle or horses, but may be affected by large amounts]
foxtails	physical	mouth, eyes, skin	**ulceration** and lesions in mouth, tongue, gums, eyes
greasewood	oxalates	kidney	**sudden death**, depression, incoordination, weakness, coma
groundsel	pyrrolizidine	liver	**weight loss**, appetite loss
horsetail	thiaminase	nervous	**thiamine deficiency**, depression, unsteady gait, incoordination, weakness, seizures; diarrhea in cattle
klamathweed	hypericin	skin	**photosensitization**, sunburn of lips, mouth and unpigmented skin, blistering, loss of condition
larkspur (tall)	alkaloids	nervous	tremors, weakness, **incoordination**, staggering, slobbering, bloat
locoweed	alkaloids	nervous	**depression**, weakness, weight loss, incoordination
lupine	alkaloids	nervous, reproductive	**birth defects**, abortion, tremors, incoordination, head pressing, seizures
milkweed	cardiac glycosides	gastrointestinal, heart, nervous	**depression, diarrhea**, colic, irregular respiration
nightshades	alkaloids	gastrointestinal	**gastrointestinal upset**, constipation or diarrhea, drowsiness, salivation, weakness
oak	tannins	liver, kidney	**sudden death**, bloody diarrhea, kidney failure
oleander	cardiac glycosides	heart	**sudden death**, depression, salivation, weakness, irregular heartbeat, diarrhea
poison hemlock	coniine (alkaloids)	nervous, reproductive	nervousness, trembling, weakness, coma, **birth defects**
Ponderosa pine	isocupressic acid	reproductive	**abortion**, incoodrination, aimless wander, congenital defects
ryegrass	alkaloids (ergot)	nervous	**incoordination**, staggering
starthistle	lactones	nervous	**weight loss**, abnormal curling of lips (chewing disease), yawning
summer pheasant's eye	glycosides	gastrointestinal	**gastrointestinal disturbance**, diarrhea, decreased gut motility, sudden death
tobacco	alkaloids (nicotine)	nervous, reproductive	**birth defects**, abortion, weakness, staggering
toyon	cyanide	cell poison, all organs	**sudden death**, salivation, heavy breathing
veratrum	alkaloids	nervous, reproductive	**birth defects**, abortion; labored breathing, salivation, convulsions
water hemlock	alcohols	nervous	**sudden death**, muscle spasms, teeth grinding, severe convulsions

Note: The signs listed in this table are commonly seen after the specific plant intoxications. However, other signs may predominate depending on the dosage, duration of exposure, and environmental factors. Diagnosis must be done by a licensed veterinarian and may require toxiciological analysis by a diagnostic laboratory to confirm the plant involved. Primary or usual signs are given in bold.

Table 4. *Continued*

HORSES			
Plant	**Toxin**	**Organ or systems affected**	**Signs**
arrowgrass	cyanide	blood, all organs	**sudden death**, salivation, heavy breathing
avocado	persin	vascular	**mastitis** (abrupt cessation of milk flow), heart failure
chokecherry	cyanide	cell poison, all organs	**sudden death,** salivation, heavy breathing
cocklebur	glycosides	cell poison, all organs	**sudden death**, weakness, inappetance, incoordination, muscle spasms, convulsions, coma
curly dock	oxalates, nitrate	kidney, blood	**sudden death**, salivation, depression, incoordination, muscle tremors, weakness, coma
deathcamas	alkaloids	nervous	**sudden death**, excessive salivation, lowered temperature, staggering, weakness, coma
dogbane/Indian hemp	cardiac glycosides	heart	**sudden death**, diarrhea, weakness, irregular heartbeat
fiddleneck	pyrrolizidine	liver	chronic: appetite loss, **weight loss;** walkabout disease, head pressing, rectal straining
foxtails	physical	mouth, eyes, skin	**ulceration** and lesions in mouth, tongue, gums, eyes
greasewood	oxalates	kidney	**sudden death**, depression, incoordination, weakness, coma
groundsel	pyrrolizidine	liver	chronic: appetite loss, **weight loss;** walkabout disease, head pressing, rectal straining
horsetail	thiaminase	nervous	**thiamine deficiency**, depression, unsteady gait, incoordination, weakness, seizures
locoweed	alkaloids	nervous	**depression**, stiff gait, incoordination, staring, excitability; difficulty eating, exaggerated mouth movement
milkweed	cardiac glycosides	gastrointestinal, heart, nervous	**depression,** incoordination, trembling, seizures
nightshade	alkaloids	gastrointestinal	**gastrointestinal upset**, constipation or diarrhea, drowsiness, salivation, weakness
oleander	cardiac glycosides	heart	**sudden death**, depression, salivation, weakness, irregular heartbeat, diarrhea
poison hemlock	coniine (alkaloids)	nervous, reproductive	**sudden death**, nervousness, trembling, weakness, coma, **birth defects**
starthistle	lactones	nervous	**weight loss**, abnormal curling of lips (chewing disease), yawning
summer pheasant's eye	glycosides	gastrointestinal	**gastrointestinal disturbance**, diarrhea, decreased gut motility, sudden death
toyon	cyanide	cell poison, all organs	sudden death
water hemlock	alcohols	nervous	**sudden death**, muscle spasms, teeth grinding, severe convulsions
western bracken fern	glycosides	bone marrow, bladder	**thiamine deficiency**, depression, unsteady gait, incoordination, weakness, seizures

Note: The signs listed in this table are commonly seen after the specific plant intoxications. However, other signs may predominate depending on the dosage, duration of exposure, and environmental factors. Diagnosis must be done by a licensed veterinarian and may require toxicological analysis by a diagnostic laboratory to confirm the plant involved. Primary or usual signs are given in bold.

Table 4. *Continued*

GOATS			
Plant	**Toxin**	**Organ or systems affected**	**Signs**
arrowgrass	cyanide	blood, all organs	**sudden death**, salivation, heavy breathing
avocado	persin	vascular	**mastitis** (abrupt cessation of milk flow), heart failure
chokecherry	cyanide	cell poison, all organs	**sudden death,** salivation, heavy breathing
cocklebur	glycosides	cell poison, all organs	**sudden death**, weakness, inappetance, incoordination, muscle spasms, convulsions, coma
deathcamas	alkaloids	nervous	**sudden death**, excessive salivation, lowered temperature, staggering, weakness, coma
fiddleneck	pyrrolizidine	liver	chronic: appetite loss, **weight loss**
foxtails	physical	mouth, eyes, skin	**ulceration** and lesions in mouth, tongue, gums, eyes
greasewood	oxalates	kidney	**sudden death**, depression, incoordination, weakness, coma
groundsel	pyrrolizidine	liver	chronic: appetite loss, **weight loss**
dogbane/Indian hemp	cardiac glycosides	heart	**sudden death**, diarrhea, weakness, irregular heartbeat
klamathweed	hypericin	skin	**photosensitization**, sunburn of lips, mouth and unpigmented skin, blistering, loss of condition
locoweed	alkaloids	nervous	difficulty eating, exaggerated mouth movement, **excitability**
lupine	alkaloids	nervous, reproductive	**birth defects**, abortion, tremors, incoordination, head pressing, seizures
milkweed	cardiac glycosides	gastrointestinal, heart, nervous	Profound **depression and weakness** accompanied by staggering; labored respiration, elevated temperatures, dilation of pupils, seizures
nightshades	alkaloids	gastrointestinal	**gastrointestinal upset**, constipation or diarrhea, drowsiness, salivation, weakness
oak	tannins	liver, kidney	**sudden death**, bloody diarrhea, kidney failure
oleander	cardiac glycosides	heart	**sudden death**, depression, salivation, weakness, irregular heartbeat, diarrhea
poison hemlock	coniine (alkaloids)	nervous, reproductive	nervousness, trembling, weakness, coma, **birth defects**
ryegrass	alkaloids (ergot)	nervous	**incoordination**, staggering
larkspur (tall)	alkaloids	nervous	tremors, weakness, **incoordination**, staggering, slobbering, bloat
toyon	cyanide	cell poison, all organs	**sudden death**, salivation, heavy breathing
water hemlock	alcohols	nervous	**sudden death**, muscle spasms, teeth grinding, severe convulsions

Note: The signs listed in this table are commonly seen after the specific plant intoxications. However, other signs may predominate depending on the dosage, duration of exposure, and environmental factors. Diagnosis must be done by a licensed veterinarian and may require toxiciological analysis by a diagnostic laboratory to confirm the plant involved. Primary or usual signs are given in bold.

Update on Controlling Smutgrass in Irrigated Pastures

Glenn Nader, Josh Davy, Larry Forero – UC Farm Advisors

Last summer UC Farm Advisors and Specialists researched ways to control the invasion of smutgrass that is occurring in Sacramento Valley irrigated pastures. The control of 95% of mature plants was obtained with the application of 33% glyphosate by a rotary wiper in early July. Note that a rotary wiper is different than a traditional rope wick. Research demonstrated that a rate of at least one part glyphosate to two parts water (33%) and a speed of 5 mph or less were necessary for adequate control. Pastures that will be treated should be grazed just prior to wiper application. The smutgrass plants will then be higher than the desirable vegetation that has been grazed down. This allows the operator to set the wiper height just above the desirable species, yet still make as much contact as possible with the smutgrass plants. A retreatment is likely necessary due to the large seed bank.

More UC research and demonstration is being conducted this spring and summer. Application timings will be evaluated throughout the summer to determine optimal treatment times. Additionally, a preemergence herbicide, pendimethalin (Prowl), has been applied this spring to determine if the seed bank can be depleted. The intent, if successful, is that control would be possible with the combination of both treatments in a single year. The long term goal is to develop a rigorous management program that will give long-term suppression of smutgrass in the timeliest manner.

Figure 1. Thirty days after treatment –note the application skip spot at the end of the check.

A publication was developed that covers the biology of smutgrass and how that relates to control or management, herbicides for smutgrass control, grazing management to encourage consumption of smutgrass, and the plants nutritional content. For a complete report go to *http://wric.ucdavis.edu/*, or contact the Cooperative Extension Farm Advisors office for a copy.

Figure 2. The wiper units are easily pulled with an ATV or UTV

Wiper units similar to the ones used in the summer of 2010 research are available for purchase price at (>$3,000). Another option is that one of the wipers used for this research was purchased by the Tehama County Resource Conservation District (TRCD) and is available for rent to treat smutgrass pastures. The unit rents for $50 a day, $75 a weekend, or $150 a week if the renter does the herbicide application. The RCD can also be contracted to do the application on a daily or weekly basis. There are added charges if delivery is necessary. The TRCD can be contacted at 530-527-3013 x5.

Rotating Wipers for Weed Control

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Rotating weed wipers have been used in other states successfully in weed control. They come in tow behind units (Figure 1) for ATV or UTV with 12 volt auxiliary connection or a battery direct connection and three point attachments for tractor mount (Figure 2). This equipment allows the operator to set the elevation of the rotating wiper above the desirable plants and actively "wipe" the herbicide on the taller target weeds. On the tow behind units, the wiper carpet-like material rotates on a drum that is driven by a belt that is connected to the tires. Spray nozzles are mounted inside a hood above the drum and the operator periodically sprays the herbicide as needed on the carpet material. The rotating drum allows the carpeted material to be very saturated and not drip herbicide due to centrifugal pressure. As the drum rotates forward while being pulled, it presses the carpet material against the underside of the leaf as it comes in contact.

Figure 1. Tow behind rotating wiper

It is critical that the desirable plants be lower in stature than the target weeds for this technology to work effectively. In pastures, grazing animals can be used to reduce the height of the desirable plants, making the ungrazed target weeds vulnerable to treatment. In cases where the weeds in crop systems grow erect or taller than the production plants, the rotating wiper can be another method of applying herbicide as seen in figure 2.

Figure 2. Three point rotating wiper

There are several companies that sell this style of rotating wipers. Two that provided wiper units for demonstration in a University of California project were Grassworks and Rotowiper. The tow behind units starts at six to fifteen feet in width and costs range from $3,000 to $5,000. Three point units are made in widths of six to fifty feet and costs range from $4,000 to $23,000.

There are some significant differences related to this type of application when compared to traditional broadcast spraying. Some are:

1. Herbicide drift is not an issue because the product is applied directly to the target plant.
2. The application solution is significantly more concentrated than broadcast mixes.
3. The pull behind rotating wipers we tested had a push-button which activates the pump that sprays herbicide on the rotating carpet. Depending on the ground speed, the button needs to be depressed for 2 to 5 seconds to spray the complete surface of the carpet as it rotates. The time interval that each time the button needs to be depressed will depend on the amount of weeds being wiped. The tendency is to keep hitting the button and overfilling the carpet which can result in material dripping onto the desirable vegetation.
4. Applicators are use to applying a certain volume per acre. With the rotating wipers the amount used will depend on the density of the weeds that are contacted.
5. The Roundup Original label notes that application speed should not exceed 5 mph.
6. Desirable plants in the pasture should be grazed as close (low) as practical so more of ungrazed target weeds are exposed to the wiper. It may be necessary to heavily graze the area more than normal prior to treatment so that the wiper can be dropped low enough on undesirable plants to adequately contact more of the treated plant.

A Case Study of the Rotating Wipers Use

Smutgrass is a perennial grass plant that is now invading irrigated pastures in the Sacramento Valley. Perennial plants translocate sugars to the roots after flowering. If applied during the flowering, the glyphosate is carried with the sugars to the roots and kills the plant. A study by Kyser and Nader in 2010 with wiper applied Roundup Weather Max (4.5 lbs/gal. concentration) at two rates (16 & 33%), with 3 timings (July 12, August 18, September 15), indicated the July 33% treatment was the most effective (Figure 4). It was estimated based on plant physiology the most effective time of wiper application should have been the late summer treatments. The August and September application injured the smutgrass, but did not kill them. It is assumed that later in the season the upper seed stock, which is the main portion of the plant contacted by the wiper, is less active or green did not absorb enough of the glyphosate to provide a killing amount to the root system. Below is a picture of smutgrass 56 days post 33% glyphosate treatment of July 12 (figure 3). After the crown has turned brown and appears dead the plant initiated a green shoot. It is yellow in color, where the shoots in the 10% plot had a darker green color. Some of these shoots later died.

Figure 3. Green shoot emerging 56 days after July glyphosate wiper application

October evaluation of the July 12 wiper application estimated control at 95% in the 33% glyphosate and 90% in the 10% glyphosate plots. Figure 4 shows the progression of smutgrass death with an untreated green area around an irrigation value in the front of the field. Two more years of control will be needed to reduce the seed bank.

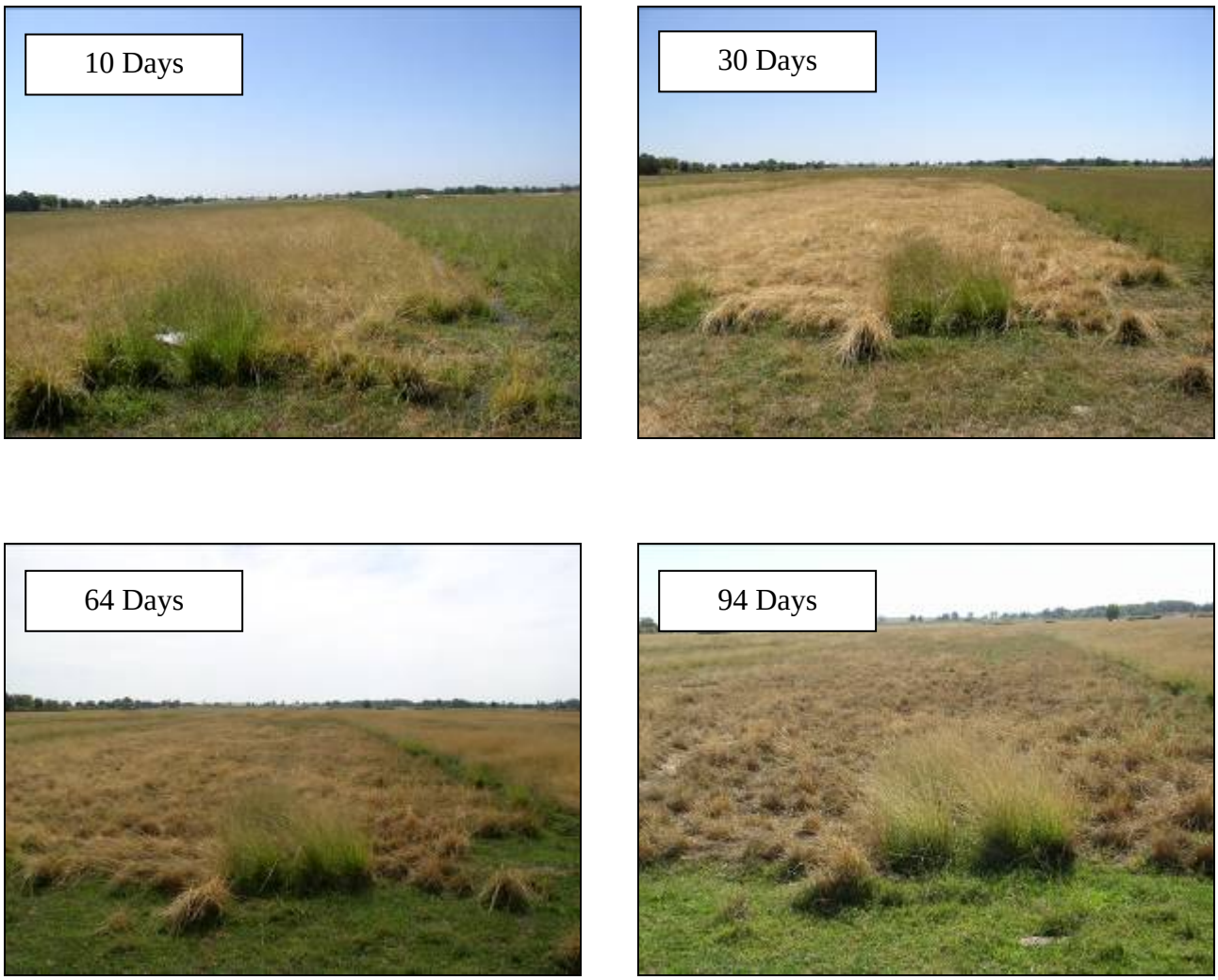

Figure 4 – Control of smutgrass at 10, 30, 64 and 94 days after a 33% glyphosate treatment in July.

Figure 5 shows the application rates and the percent green smutgrass 20 days after wiper treatment with Roundup Power Max (4.5 lbs/gallon) in a study conducted by Davy and Karle. The application was made September 13, 2010. The graph shows a clear rate response, with 33% percent providing far better control compared to 10%, but nearly the same control as 68%. Plants that survived treatments with higher rates tended to be lower growing, thus escaping contact by the wiper.

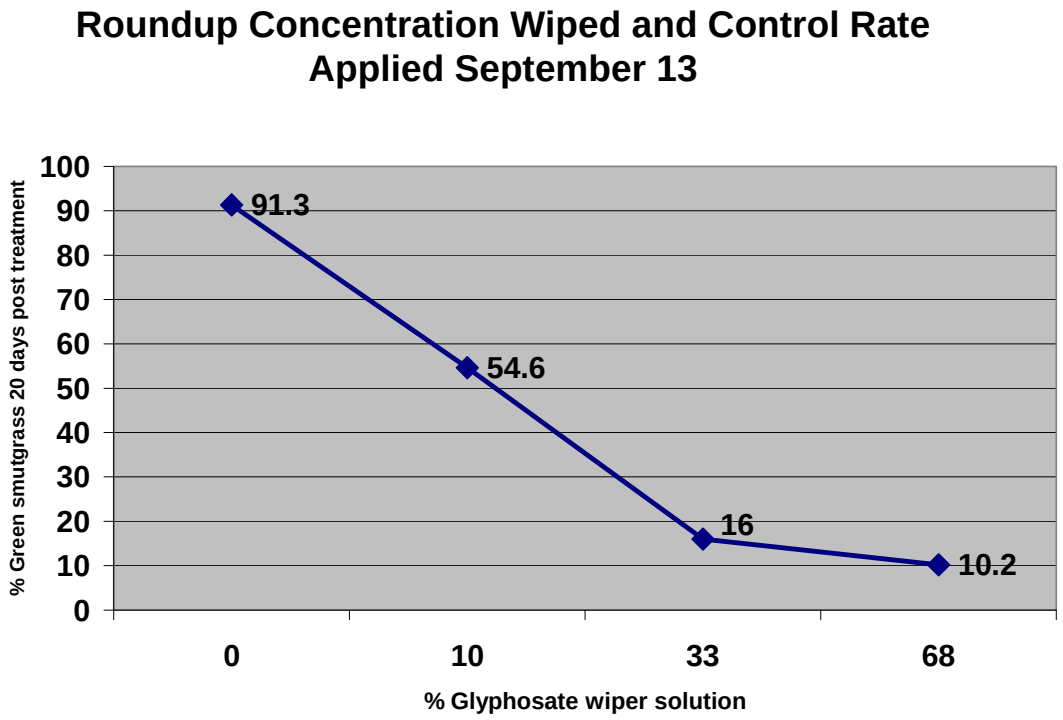

Figure 5 – Impact of Concentration of Glyphosate on Control

Rotating wipers offer a different way to effectively control weeds. Low amounts of higher concentration application of glyphosate have proven effective in control of weeds. The timing of the glyphosate application may be different than spray applications, but can provide many advantages when height differential of weeds and cultured plants occurs. This is especially true in pastures where controlling unpalatable grass is truly limited to this method.

Figure 6 - Control of Pigweed.