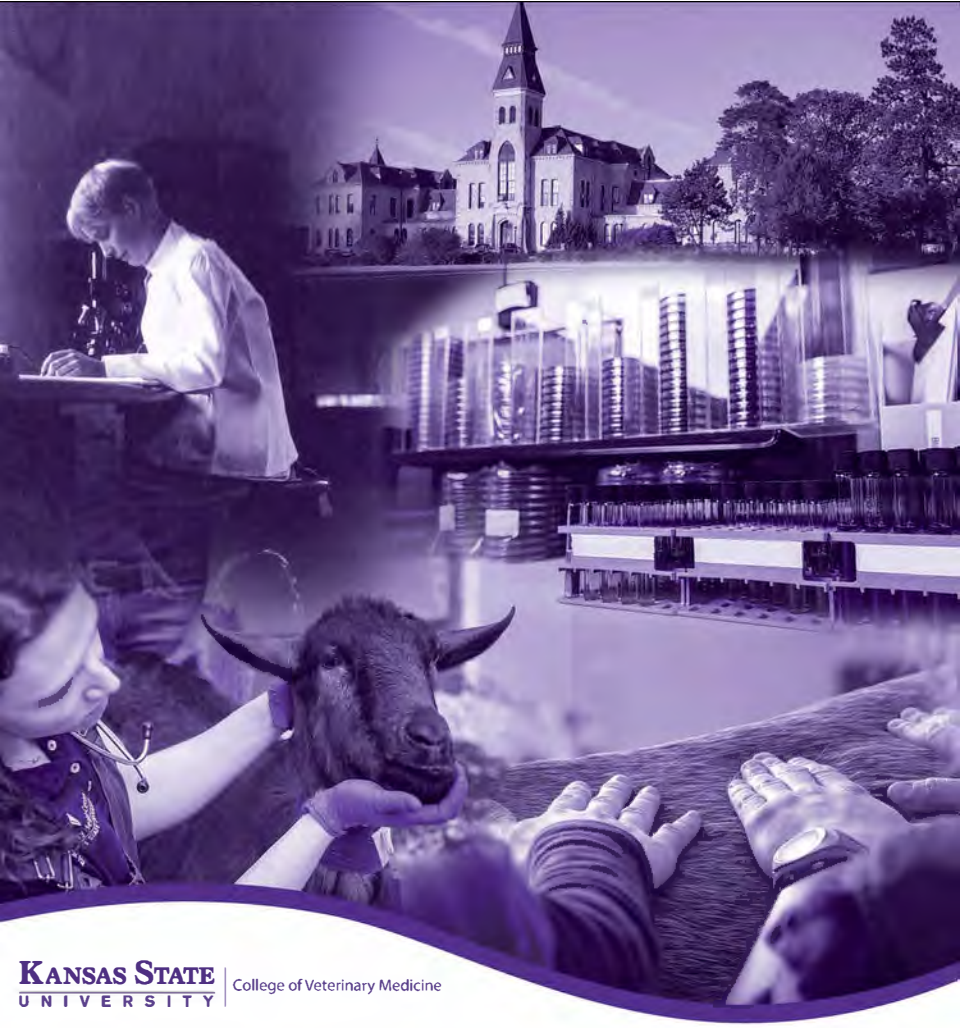

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Trichomoniasis: Herd Management and Prevention Strategies

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Bovine Trichomoniasis:

Observations on pathogenesis, sample collection, diagnostic testing, and herd management.

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Abstract:

Bovine Trichomoniasis is an important sexually transmitted reproductive disease of cattle. The increasing awareness of the financial impact of this reproductive pathogen has resulted in state regulations affecting the movement of bulls both within and between states. Discoveries regarding the pathogen and the host, as well as improvements in diagnostic testing strategies to identify infected herds, have allowed for the development of more successful control measures. This paper discusses the biology, prevalence, economic impact, management and control techniques for this important reproductive pathogen.

Introduction:

Trichomonads are unicellular protozoa that have a global distribution in livestock, poultry, companion animals, and people. The significance of these protozoal organisms goes back to the discovery of light microscopy. In the late 17th century, Antoni van Leeuwenhoek demonstrated several free-living protozoa and later an intestinal parasite with the aid of light microscopy. The first report of a venereal trichomonad (in humans) was described by Donne' in 1837 [Richardson et al 1963]. This organism later became known as *Trichomonas vaginalis*. *T. vaginalis* is the number one, non-viral sexually transmitted disease (STD) that affects more than 250 million people worldwide and causes an estimated 7.4 million cases in the United States [Mundodi et al 2006]. The original description of the disease process caused by *Tritrichomonas foetus* is credited to Kunstler in 1888. Credit for the discovery of the organism in infertile cattle is given to the Italian scientist Mazzanti in 1900. The name *Trichomonas foetus* was proposed around the work in Europe by Riedmuller in 1928. Later in 1932, Emmerson reported the disease in the United States after demonstrating the organism in infertile cows [Emmerson 1932].

Morphology:

The pear shaped organism is slightly larger than a polymorphonuclear leukocyte (measuring up to 24 microns) and is classified by the number of anterior flagellae, which range from 3 to 8 microns. Though most organisms of the order Polymastigina inhabit the gastrointestinal tract, some may cause disease in the urogenital tract. *Tritrichomonas foetus* exhibits a polar and flagellated trophozoitic form during its simple life cycle. During stress and unfavorable growth conditions, these trophozoites round up and

internalize their flagella forming pseudocysts [Pereira-Neves et al 2009]. Reproduction of the pseudocyst stage occurs by mitosis, which includes nuclear division but differs in that it lacks division of the cytoplasm resulting in giant multinucleated cells. When environmental conditions become favorable, the flagella are externalized and new flagellated trophozoites bud off the multinucleated cell. In a 2011 study, Pereira-Neves et al utilized video microscopy, fluorescence microscopy, scanning and transmission electron microscopy to evaluate fresh preputial samples from naturally infected bulls [Pereira-Neves et al 2011]. This work revealed that approximately 55% of the *T. foetus* organisms were in pseudocyst form and approximately 25% exhibited pear-shaped form.

Effects on Reproduction in Beef Cattle:

The following discussion describes the potential reproductive effects of *T. foetus* infection.

Bulls:

The bull is the long-term carrier of the organism without ill effects or visible lesions [Honigburg 1978]. BonDurant monitored infected bulls for nearly a year by weekly examination of smegma cultures and found that cultures were consistently positive for virtually the entire period [Schonmann et al 1994]. Older reports on the clinical presentation indicated that (though not always apparent) lesions included slight swelling of the prepuce, painful micturition, preputial discharges, and small red nodules on the mucosal surfaces of the prepuce can accompany infection [Richardson et al 1963]. For decades, it was believed that, as a bull matured the epithelial crypts of the glans penis and the distal prepuce became deeper to allow the *T. foetus* organism to establish a long-term infection [BonDurant 1997]. Strickland et al utilized electron microscopy and reported no difference in the depth of preputial skin folds between young and mature bulls noting no structures comparable to intestinal crypts were identified [Strickland et al 2014]. Self-clearance with sexual rest is reported in the literature. A February 2008 herd investigation by the author in east Texas revealed 25% (3 /12) of the bulls were infected (both culture + and rtPCR +) without any apparent decline in pregnancy rate. At the request of the owner, a valuable four Y.O. Black Angus bull was kept on the ranch with sexual rest until July with hopes of self-clearance. At that time, two samples were collected and submitted to the Texas Veterinary Diagnostic Laboratory (TVMDL) for testing. The bull was culture negative (both samples) yet remained positive on real-time polymerase chain reaction (rtPCR) (both samples). This diagnostic dilemma is indicative of the limitations of testing methods as well as the need for increased understanding of the disease triad (host – pathogen – environment) interactions associated with bovine trichomoniasis.

Cows / Heifers:

The *T. foetus* organism is efficiently spread during natural breeding. Parsonson et al demonstrated a 95% single service transmission rate to 19 of 20 virgin Hereford heifers when exposed to a naturally infected 3-year-old Hereford bull [Parsonson et al 1976]. Once the organism is transmitted, the infection begins. Though the organism is introduced at the time of breeding, conception appears to be established in the face of infection. Subsequent development of the embryo is not affected [Abbitt 1980]. Fetal death occurs at some point after maternal recognition

of pregnancy [Bazer et al 1991]. An early sign of the presence of *T. foetus* is a vaginitis associated with granular lesions on the floor of the cranial vagina [Richardson et al 1963]. Once the organism reaches the uterus, the resulting inflammation is detectable around 49 days post exposure. This gives rise to the extended time between observed heat cycles. Earlier work has indicated that pregnancy and infection were sustained for 50-60 days [Parsonson et al 1976]. Spontaneous clearance of the infection in female cattle can occur in 2-4 months [Bondurant 1997]. Though clearance of the organism is suggested, persistent infection (female carries the infection through gestation into the next breeding) has been documented [Skirrow 1987]. Though the estimated frequency is low (<1%), it requires consideration of the female carrier state for control programs when diagnosed in the herd [Rae et al 2006].

Prevalence:

Prevalence of bovine trichomoniasis has been estimated in various cattle producing countries around the world. Perez et al (1992) reported prevalence in Costa Rica between 3.9 and 6.2%. In the United States, slaughter studies conducted at facilities in North Carolina [Fox et al 1995], Colorado and Nebraska [Grotelueschen et al 1994] revealed an extremely low prevalence of 0% and less than 1% respectively. It should be noted; these studies utilized available culture techniques only. Other studies [Ball et al 1984] reported a prevalence of 7.5% in bulls from Arizona, Utah, Colorado, Idaho, and Wyoming. Prevalence in predominantly range cattle of the western United States has been reported at rates of 5-8% [Fitzgerald 1986 and Johnson 1965]. Szonyi et al used 2010 data from the Texas Veterinary Medical Diagnostic Laboratory (TVMDL) to estimate the occurrence and spatio-temporal distribution of bovine trichomoniasis in Texas. This work was the first large-scale epidemiological study of bovine trichomoniasis that utilized rtPCR generated data to identify spatial disease clusters within the state of Texas [Szonyi et al 2012]. This study indicated 3.7% positives out of 31,202 tests and identified a spatial cluster of disease in southeastern Texas. Though this does not provide an estimate of the true prevalence in Texas, it does provide meaningful information about the risk of introduction into susceptible herds.

Treatment:

Successful systemic treatment of affected bulls with imidazoles (e.g. ipronidazole) was reported in 1985 [Skirrow et al 1985]. More recently, a naturally infected bull was reported to have cleared the *Tritrichomonas foetus* infection after a 2-dose intravenous administration of 60 mg/kg metronidazole, 24 h apart [Love et al 2017]. The reader is reminded that the Animal Medicinal Drug Use Clarification Act (AMDUCA) of 1994 banned imidazoles, such as metronidazole, for use in food animals. These compounds are prohibited for extra-labeled use in food producing animals in the United States [Guest 1988].

Sample Collection:

Despite proven control procedures and diagnostic testing through PCR, *T. foetus* has become endemic in several US states in recent years. This has led to increased interest in *T. foetus* diagnostics. According to Mukhufhi, "while much emphasis has fallen on primer selection and the optimization of laboratory protocols to render satisfactory results, little attention has been given to sample collection and handling procedures" [Mukhufhi et al 2003].

Studies have examined the influence of transport media [Appell et al 1993, Parker et al 2003], time delay and media [Mukhufhi et al 2003, Kittel et al 1998], time delay and low holding temperatures [Cobo et al 2007], and strain, time delay, low temperatures, and media [Bryan et al 1999]. These trials have shown factors related to shipping and handling such as time, low temperatures, and transport media can influence the ability of culture and PCR to correctly classify samples from infected cattle. However, they did not address the influence of high transport temperatures on test sensitivity. It is documented that temperatures as high as 140°F (60°C) can be reached in parcel delivery vans[a]. This has obvious implications for diagnostic laboratories receiving samples for trichomoniasis testing during seasons of the year, which have high ambient temperatures.

Various methods of sample collection have been described in the literature. The most commonly used include washes and scrapes (pipettes). It has been reported that the sensitivity of scrapings (78.3%) and washings (82.1%) are not statistically different [Schonmann et al 1994]. Once the sample is collected, it is inoculated into a commercially available culture and transport media[b]. The quality of the sample is highly dependent on the skill and experience of the collector. The sensitivity of the test is affected by the field conditions (i.e. low number of organisms collected when sampled) and handling conditions (i.e. temperature and transit time to laboratory). Fortunately, the sensitivity has increased with the universal acceptance of the DNA – based PCR test.

One of the many variables (and weaknesses) of the current testing methodologies is consistent sample collection. Ensuring an adequate sample is critical for confidence in the diagnostic test results.
The author instituted a quality control process for sample collection utilizing a visual comparison between the upper and lower chambers of the commercially available culture and transport media[c]. Once the upper chamber was inoculated, it was compared to the lower chamber in the pouch before moving the contents of the upper chamber into the lower chamber and sealing. If there was no appreciable difference in opacity along with noticeable flecks of smegma in the pouch, then the collection is repeated. Care should be taken to emphasize the importance of going back to re-sample the bull if the quality control evaluation of the sample collection warrants it. Parker et al reported on the potential impact that dexterity has on diagnostic testing for Trichomoniasis [Parker et al 2003]. In this study of known *T. foetus* infected bulls (n=29), samples collected with the dominant hand were 4 times more likely to correctly classify a bull as infected when compared to samples taken with the non-dominant hand.

Sample Handling:

The foundation of a veterinarian's confidence in a test result is the knowledge that not only was an adequate sample collected, but that the sample was not compromised from the time of collection until arrival at the diagnostic laboratory. The insert that accompanies the commercially available culture and transport media[c] states that the pouch is to be maintained in the specified temperature range of 15°C (59°F) – 37°C (98.6°F) during transport to the laboratory. Poor handling of inoculated pouches can (and does) have a negative impact on the test result particularly when the culture test is selected. All the benefit of careful collection can be lost with a poorly handled specimen post-collection. Temperature effects on test results have been reported. The effect of cold on the test result was reported previously [Bryan et al 1999]. Briefly, tubes containing thioglycollate transport medium or commercially available pouches were inoculated with 4,000 to 5,000 organisms and kept for up to seven days at 37°C (98.6°F), 22°C (71.6°F), 4°C (39.2°F), or -20°C (-4°F). The culture results are as follows: 37°C (98.6°F): positive out to 4 days, 22°C (71.6°F): positive out to 4 days, 4°C (39.2°F): negative after 5 days, and -20°C (-4°F): negative after 3 Hours.

The effect of high temperatures on the ability of the laboratory to correctly classify inoculated pouches was evaluated [Davidson et al 2009]. The objective of this study was to evaluate the effect of high temperatures over simulated transport times on the results of culture and rtPCR for *T. foetus* in experimentally inoculated commercially available culture media. The results were as follows: Culture results of pouches exposed to 37°C (98.6°F) for all treatment times were positive while those subjected to 46.1°C (115°F) for 1, 3, 6, and 24 hours were positive, positive, negative, and negative, respectively. Pouches subjected to 54.4°C (130°F) for 1, 3, 6, and 24 hours were positive, negative, negative, and negative, respectively. All 72 inoculated samples were positive by rtPCR. Positive rtPCR test results for all 72 inoculated pouches were expected based on the stability of deoxyribonucleic acid at high temperatures [Wang 1985] and suggest molecular assays may offer results that are more reliable from specimens potentially exposed to high temperatures than microscopic examination. However, it is important to note that as temperature and time increased to 46.1°C (115°F) for 6 and 24 hours and 54.4°C (130°F) for 3, 6, and 24 hours so did cycle threshold (ct) values. While high temperatures may affect the growth rate of the organism, they do not appear to negatively influence the ability of rtPCR to identify the organism. Therefore, samples submitted to diagnostic laboratories for *T. foetus* testing during warm seasons should be protected from high temperatures and submitted for rtPCR testing.

Test Selection (Culture, PCR, and Pooled PCR):

Procedures for the diagnosis of Trichomoniasis have evolved over time. Current testing strategies for *T. foetus* include culture and PCR. Work by Corbeil has shown consistent results when comparing PCR to indirect immunofluorescence assay (IFA) [Corbeil et al 2008]. A better understanding of the sensitivity and specificity of these diagnostic tests has also come through focused studies. Sensitivity measures the proportion of actual positives which are correctly identified as such (i.e. the percentage of sick animals who are identified as having the condition); and specificity measures the proportion of negatives which are correctly identified (i.e. the percentage of non-diseased animals that are identified as not having the

condition). Negative predictive value (proportion of animals with negative test results that are correctly diagnosed) will decrease as the prevalence increases. In other words, there is less confidence in the negative test result on a given test as the prevalence or pre-test odds of disease increases. The literature has seen a wide range of values for sensitivity and specificity of *T. foetus* cultures. It was reported that the sensitivity of culture was as high as 99% [Tedesco et al 1979]. Moreover, before DNA typing of the enteric trichomonads, the specificity of the *T. foetus* culture was reported to be 100% specific [BonDurant et al 1990]. PCR testing has improved detection of *T. foetus* (sensitivity) and proper classification of non-*T. foetus* trichomonads (specificity). These non-*T. foetus* organisms are actually enteric species (*Tetratrichomonas spp*. or *Pentatrichomonas hominis*). This brought about awareness of the potential for a false positive culture test. In other words, bulls or female cattle might be culled based on having a non-pathogenic trichomonad organism present in the culture. As the PCR tests came into use in diagnostic laboratories, a test was developed that could differentiate the type or species of trichomonad present in the culture as well as detect as few as 1 trichomonad [Ho et al 1994] under bench top lab conditions. This sensitivity is lowered when the smegma from the bull is added to the testing specimen. Further, it can be concluded that the enteric forms (non-*T. foetus* trichomonads) can be found in the prepuces of bulls (virgin and non-virgin) and that non-*T. foetus* trichomonads can be transmitted to cattle at breeding [Corbeil et al 2008]. Though non-*T. foetus* trichomonads have been recovered from female tracts, studies [Agnew et al 2008 and Cobo et al 2007b] suggest that the infection is likely transient and non-pathogenic.

Cobo et al evaluated the sensitivity (Se) and specificity (Sp) of culture and gel PCR in artificially infected bulls [Cobo et al 2007a]. In *T. foetus*-inoculated bulls, both tests (culture and gel PCR) combined in parallel on a single sample had a Se (78.3%) and Sp (98.5%) similar to two cultures (Se 76.0%, Sp 98.5%) or two PCR (Se 78.0%, Sp 96.7%) sampled on consecutive weeks. The gel PCR on three consecutive weekly samples (Se 85.0%, Sp 95.4%) and both tests applied in parallel on three consecutive weekly samples (Se 87.5%, Sp 95.6%) were similar to the current gold-standard of six weekly cultures (Se 86.7% and Sp 97.5%). Both tests used in parallel six times had the highest Se (93.3%), with similar Sp (92.5%). For a herd with infertility problems, the pretest probability (prevalence) is estimated to be in the range of 20-60%. The effect of increasing prevalence on negative predictive value (NPV) is demonstrated in the Chart 1.

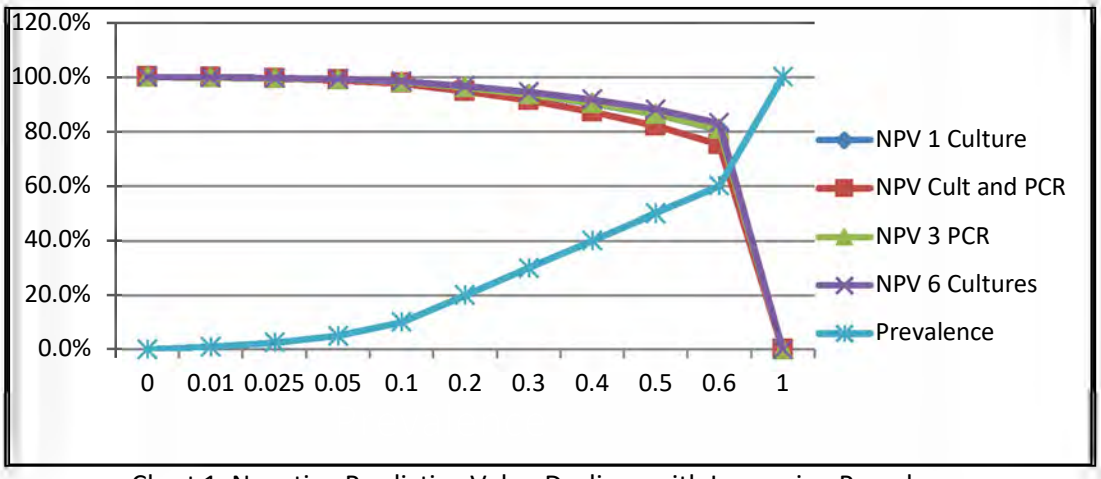

Chart 1: Negative Predictive Value Declines with Increasing Prevalence

Under adverse conditions, the PCR test has significant advantages over the culture. Recent studies have indicated that the diagnostic sensitivity is very similar [Ondrak et al 2010[a]]. Ondrak et al compared culture, gel PCR, and rtPCR during sequential samplings in a Nebraska herd with 2960 cows and 121 Angus and Hereford bulls. The analysis of 1, 2, or 3 preputial specimens from bulls on the study ranch resulted in properly identifying 21 (87%), 23 (95%), and 24 (100%) bulls infected with *T. foetus* respectively. According to Ondrak, "it is our recommendation that at least 3 preputial specimens be collected from each non-virgin bull on an infected premise." In the same study, Ondrak also correlated within herd bull prevalence with pregnancy rates as shown in Chart 2 below.

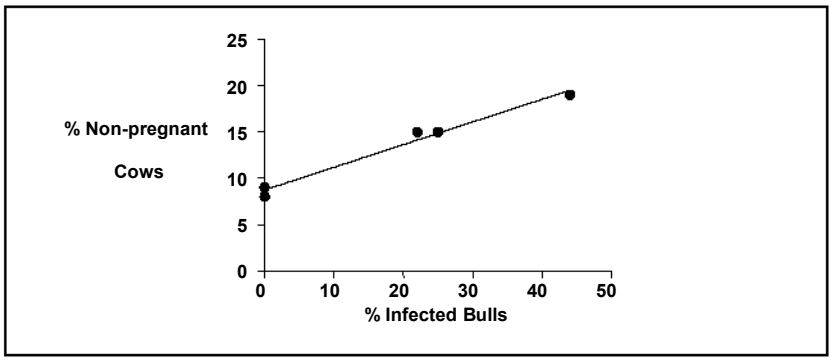

Chart 2: Correlation of Herd Pregnancy Rate with Increasing Bull *T. foetus* Prevalence

Pooled PCR Testing for *T. foetus*:

The Texas Animal Health Commission (TAHC) amended the Bovine Trichomoniasis Regulations in 2012 to allow pooled samples up to 5:1 for rtPCR testing at an official lab [Ellis 2012]. TVMDL submission data for 2014-2015 indicates that pooled PCR testing has been widely adopted with nearly 1 in 4 bulls tested in a pooled sample[d]. Kennedy was an advocate for pooled PCR testing for Bovine Trichomoniasis. His 2007 study indicated that conventional PCR testing on 61 pools of 5 samples (305 pouches total) did not negatively impact the Sensitivity (Se) or Specificity (Sp) of individual PCR tests and exceeded the Se of a single culture [Kennedy et al 2007]. Further, the Se and Sp was 100% when compared to individual PCR tests. Kennedy further stated that another potential application of pooled testing is monitoring herd status after herds or states have been declared free or at low risk of *T. foetus* infection. The utilization of pooling and PCR could minimize the number of collections per bull, as well as provide Se that would approach or surpass multiple cultures. The use of PCR relies on DNA, not viable organisms, and is capable of detecting the presence of the organisms when improper transport or collection technique has resulted in the loss of viable organisms. Kennedy summarized the work of Cowling and Sergeant [Cowling et al 1999, and Sergeant et al 2004] in developing the list of advantages and disadvantages of pooled testing listed below:

Advantages:

1. Cost advantages related to laboratory fees
2. Improved accuracy over repeating a test each time
3. More precise estimates of prevalence when Se and Sp are less than 1
4. Less bias when assumed Se and Sp are not equal to true values

Disadvantages:

1. Potential loss of Se to dilution
2. Costs to pool samples
3. Retesting costs on positive pools
4. Failing to evaluate adequate numbers of pools before predicting prevalence yielding incorrect results

Vaccination as a Component of a Herd Management Program:

The immunologic basis for reduction in the shedding resulting from *T. foetus* whole cell immunization was reported by Kvasnicka [Kvasnicka et al 1989]. This unknown mechanism was theorized to "interfere with the ability of the organism to prevent conception and/or fetal development." Kvasnicka et al later reported an approximate ten-fold increase in serum neutralizing antibodies resulting from whole cell *T. foetus* vaccination [Kvasnicka et al 1992]. More recently, Palomares and colleagues reported that a commercial vaccine containing killed whole *T. foetus* antigen provided a significant induction of IgG antibodies to both *T foetus* whole-cell and TF1.17 surface antigens in bovine serum, vaginal secretions, and uterine flush samples [Palomares et al 2017].

Cows / Heifers:

A commercially available vaccine[e] is available as an aid in the reduction of shedding of *T. foetus*. The vaccine label indicates an initial dosing regimen of two 2mL doses 2-4 weeks apart with the last injection occurring 4 weeks before breeding season. This is followed by an annual revaccination of a single 2mL dose. The benefits of the properly timed administration of this *T. foetus* vaccine in female cattle have been previously described in the veterinary literature. A 1992 study by Kvasnicka et al revealed that more pregnancies are maintained in the vaccinated group (62.5% of 65 exposed heifers) than the control group (31.5% of 64 exposed heifers) when challenged with *T. foetus* infected bulls under simulated breeding conditions [Kvasnicka et al 1992]. The authors concluded that the vaccine used in this study was effective in reducing losses in calf production attributable to early fetal death and abortion caused by *T. foetus* infection. For some less familiar with the severe challenge model reported in the Kvasnicka study, the efficacy of the protozoal vaccine is suspect. The calculated preventive fraction of 45% is well below the standards held for other vaccines such as modified live viral vaccines with fetal protection claims for bovine viral diarrhea virus (BVDV). It should be noted that the calving percentage in cattle receiving the protozoal vaccine against *T. foetus* increased 97% compared to the unvaccinated controls.

More recently, Auburn University researchers conducted a rigorous challenge study to evaluate the benefit of vaccination[e] against *T. foetus* on organism clearance and subsequent fertility in heifers [Edmondson et al 2017]. The rigorous challenge consisted of intravaginal inoculation at estrus with 10^6 *T. foetus* organisms. This resulted in 100% of the challenged heifers having positive cultures for *T. foetus*. Only 20% (4 of 20 heifers) of the non-vaccinated heifers delivered a live calf compared to 50% (10 of 20 heifers) in the vaccinated group. Based upon the 150% increase in live calves born, the authors concluded that the killed, whole cell vaccine used in this study was effective in improving reproductive health evidenced by significantly reducing losses associated with T. foetus infections.

When the economics of a near 100% to 150% increase in calf crop are considered in the infected herd, the added cost inputs (labor and vaccine) are justifiable. Villarroel et al used a simulated model to evaluate the

effect of the vaccine[e] on reproductive efficiency in *T. foetus* infected beef herds [Villarroel et al 2004]. This study concluded that vaccination resulted in a significantly higher calving incidence. Further, the authors noted that when not all risk factors could be avoided, proper vaccination decreased the economic losses attributable to *T. foetus* abortions [Villarroel et al 2004].

Bulls:

Cobo et al reported on the preputial cellular and antibody responses to a commercially available *T. foetus* vaccine[e] in dairy bulls challenged with live *T. foetus* organisms [Cobo et al 2010]. This study demonstrated that bulls vaccinated with 2 doses according to the timeline specified on the product label and subsequently challenged with 1.5×10^6 *T. foetus* organisms resisted infection (the preventative fraction was 100%), developed a humoral immune response (IgG1 and IgG2) against *T. foetus* in both serum and preputial secretions. Further, non-vaccinated bulls challenged with *T. foetus* organisms at both low (1.6×10^6) and high (1×10^7 and 3.4×10^7) doses of *T. foetus* were persistently infected and had no detectable antibodies to *T. foetus* in either preputial secretions or serum for 6 weeks post-challenge. Cobo et al concluded that "genital and serum IgG antibodies to *T. foetus* accounts for resistance of vaccinated bulls to *T. foetus* infection and that the lack of an antibody response in infected bulls accounts for persistent infection." Other researchers have concluded that vaccination of bulls along with breeding age females is a component of the control and prevention of *T. foetus* [Edmondson 2013].

Breeding Ratios:

Barth suggested that up to 80% of cows in multi-sire breeding groups might be serviced by two or more bulls during a single estrus period [Barth 2007]. The over-mating that results is a significant factor in the transmission of venereal diseases like bovine trichomoniasis. Proper male to female ratios are essential to reducing the over-mating that can exacerbate this disease. The absence of animal identification and pasture records of bull movements hampers the successful management of the infected herd.

Strategies to eliminate *T. foetus* from an infected herd: [Ondrak 2016 (adapted)]

1. Sample and test all herd bulls 3 times regardless of the test used and cull all test positive bulls, or

2. Cull all herd bulls to eliminate the time and money spent on testing while eliminating any risk of misclassifying a bull as *T. foetus*–negative, which would allow the organism to remain in the herd.

3. Cull all nonproductive cows (i.e., cows not pregnant at the end of the breeding season or that fail to deliver a live calf before the next breeding season), or

4. Establish 2 distinct female management groups based on their potential for *T. foetus* infection with virgin heifers and cows delivering live calves in one group and nonproductive cows in the other.

5. Consider vaccinating all females with an approved trichomoniasis vaccine, which will not prevent infection, but seems to reduce fetal loss associated with infection and the duration of infection.

Strategies for prevention in uninfected herds: [Ondrak 2016 (adapted)]

Low-Risk Herds:

1. Develop communication networks with neighbors to ensure rapid notification if trichomoniasis is diagnosed in a neighboring herd.

2. Monitor fences and cattle to rapidly identify when unplanned commingling with another herd has occurred so immediate steps can be taken to address the problem and reduce the risk of introducing trichomoniasis.

3. Maintain herd records to monitor herd reproductive performance and identify animals within management groups for early detection of a potential trichomoniasis incursion and efficient management of the outbreak.

4. Observe interstate and intrastate animal health regulations regarding trichomoniasis and other diseases. Although regulations are in place to protect the livestock industries of each state, they should be considered a barrier to trichomoniasis introduction and not a complete prevention program at the herd level.

5. Purchase replacement animals, preferably virgin bulls and heifers, from a reputable source. The purchase of nonvirgin bulls and cows, especially from herds with unknown reproductive performance, increases the risk of trichomoniasis introduction.

High-Risk Herds

1. Implement prevention strategies for low-risk herds as described in Low-risk Herds.

2. Plan a pasture use program to minimize contact with neighboring cattle.

3. Use proper artificial insemination protocols with semen from a reputable source in specific management groups or the entire herd to greatly reduce the risk of *T. foetus* transmission.

4. Maintain a young bull battery to reduce the rate of transmission and the potential development for chronic carrier bulls.

5. Isolate and/or test cattle if unplanned commingling with neighboring herds has occurred. Females should be isolated from the rest of the herd until after the breeding season and their pregnancy status can be confirmed. Bulls should be isolated and tested to ensure they are *T. foetus*-negative, which may require 3 tests at weekly intervals.

6. Restrict the duration of the breeding season to less than 120 days to reduce the opportunity for transmission of the disease within the herd and to more easily

monitor reproductive performance.

7. Institute a surveillance testing program.

Risk Based Vaccine Protocol Development:

Careful consideration of specific pathogens must be given when developing a risk based approach to herd health program design and development. When the prevalence, efficiency of transmission, and economic impact of *T. foetus* is evaluated, the incorporation of a commercially available *T. foetus* vaccine[e] in known infected or at risk herds should be carefully considered.

Producer Education:

Recently a new web-based producer education tool[f] was introduced by a team of veterinarians from the United States and Canada. TrichCONSULT *(Collaborative, Online, Novel, Science-based, User-friendly, Learning, Tool)* was created for the benefit of the beef cattle industry to enhance the control of Trichomoniasis in beef cow-calf herds. TrichCONSULT was designed to aid cattle producers and their veterinarians in creating Trichomoniasis control, prevention and eradication strategies that are specific to individual herds. Reference articles and additional resources related to Bovine Trichomoniasis can be accessed from the TrichCONSULT website. The project was funded by Kansas State University Veterinary Diagnostic Laboratory, the Coleman Foundation for Food Animal Production Medicine at Kansas State University and by USDA grant 2014-09684.

Client Communication:

The economic impact of Trichomoniasis has been reported previously [Clark et al 1982, Rae 1989, and Villarroel et al 2004]. Weaknesses in testing (pre-analytical, analytical, and post-analytical), prevention (vaccine timing and duration of immunity or DOI), and herd level biosecurity create challenges and potential (medico-legal) liability exposure for the attending veterinarian. Documentation of herd level recommendations regarding limitations of diagnostic testing and prevention (vaccination), as well as the impact of female carrier cattle should be incorporated into the written recommendations presented to the appropriate parties during the testing processes. The absence of a complete written communication of the aforementioned limitations in the medical record should be a cause of concern for the practitioner today.

Conclusion:

Trichomoniasis can have significant impacts in the reproductive performance of beef and dairy cattle operations. It is becoming more apparent that sample collection and handling are just as important as test selection. Vaccination of high-risk cattle, including bulls, continues to be an important component of the management of the *T. foetus* infected herd. As is increasingly the case, documentation of the management and diagnostic recommendations provided to the herd owner from the herd veterinarian is essential to preventing misunderstandings and ensure compliance.

Endnotes

a. McDavid PM. Personal communication. June 2008.
b. InPouch TF®, Biomed Diagnostics, Inc., White City, OR.
c. Package Insert, InPouch TF®, Biomed Diagnostics, Inc., White City, OR.
d. Cochran, M. Associate Director, Texas Veterinary Medical Diagnostic Laboratory, College Station, TX, Personal Communication.
e. TrichGuard®, Boehringer Ingelheim Animal Health USA Inc., Duluth, GA.
f. TrichCONSULT (www.trichconsult.org)

References

Abbitt B. Trichomoniasis in cattle. Current Therapy in Theriogenology, 1st ed 1980. 482-488.

Agnew DW, Munson L, Cobo ER, Olesen D, Corbeil LB, BonDurant RH. Comparative histopathology and antibody responses of non *Tritrichomonas foetus* and *Tritrichomonas foetus* genital infections in virgin heifers. *Vet. Parasitol;* 2008. 151:170–180.

Appell LH, Mickelsen WD, Thomas MW, Harmon WM. A comparison of techniques used for the diagnosis of *Tritrichomonas foetus* infections in beef bulls. *Agric Pract* 1993; 14:30-34.

Ball L, Mortimer RG, Cheney JM, Olsen JD. Trichomoniasis: diagnosis, pathogenesis, treatment and control. *Bovine Pract*; 1984. 16-163-165.

Barth A. D. Evaluation of Potential Breeding Soundness of the Bull In: *Current Therapy in Large Animal Theriogenology (Second Edition)*. Saint Louis: W.B. Saunders, 2007;228-240.

Bazer FW, Thatcher WW, Hansen PJ, et al. Physiological mechanisms of pregnancy recognition in ruminants. *J Reprod Fertil* 1991; 43:39-47.

BonDurant RH, Anderson ML, Blanchard P, et al. Prevalence of trichomoniasis among California beef herds. *J Am Vet Med Assoc* 1990; 196:1590-1593.

BonDurant R. Pathogenesis, diagnosis, and management of trichomoniasis in cattle. Vet Clin North Food Anim 1997; 13:345-61.

Bryan LA, Campbell JR, Gajadhar AA. Effects of temperature on the survival of *Tritrichomonas foetus* in transport, Diamond's and InPouch TF media. *Vet Rec* 1999; 144:227-232.

Clark BL, Dufty JH, Parsonson IM. The effect of *Tritrichomonas foetus* infection on calving rates in beef cattle. *Aust Vet Jour* 1982; 60:71-4.

Clark BL, Dufty JH, Parsonson IM. Immunisation of bulls against trichomoniasis. *Aust Vet J* 1983; 60:178–9.

Clark BL, Emery DL, Dufty JH. Therapeutic immunisation of bulls with the membranes and glycoproteins of *Tritrichomonas foetus* var. brisbane. *Aust Vet J* 1984; 6:65–6.

[a]Cobo ER, Favetto PH, Lane VM, Friend A, VanHooser K, Mitchell J, BonDurant RH. Sensitivity and specificity of culture and PCR of smegma samples of bulls experimentally infected with *Tritrichomonas foetus*. *Theriogenology* 2007; 68:853–860.

[b]Cobo ER, Corbeil LB, Agnew DW, VanHoosear K, Friend A, Olesen DR, BonDurant RH. *Tetratrichomonas* spp. and *Pentatrichomonas hominis* are not persistently detectable after intravaginal inoculation of estrous heifers, *Vet. Parasitol* 2007; 150:18–26.

Cobo ER, Corbeil LB, Gershwin LJ, BonDurant RH. Preputial cellular and antibody responses of bulls vaccinated and/or challenged with *Tritrichomonas foetus*. *Vaccine* 2010; 28:361–370.

Corbeil LB, Campero CM, VanHoosear K, BonDurant RH. Detection of trichomonad species in the reproductive tracts of breeding and virgin bulls. *Veterinary Parasitology* 2008; 154:226-232.

Cowling DW, Gardner IA, Johnson WO. Comparison of methods for estimation of individual level prevalence based on pooled samples. Prev Vet Med 1999; 39:211-225.

Davidson JM, Ondrak JD, Anderson A, et al. Evaluation of effects of high incubation temperatures on results of protozoal culture and real-time PCR testing for *Tritrichomonas foetus* inoculated in a commercially available self-contained culture media system. *J Am Vet Med Assoc* 2011; 239: 1589-1593.

Edmondson MA. Managing bovine trichomoniasis in the female. *Clin Ther* 2013; 5: 225-230.

Edmondson MA, Joiner KS, Spencer JA, et al. Impact of a killed *Tritrichomonas foetus* vaccine on clearance of the organism and subsequent fertility of heifers following experimental inoculation. *Therio* 2017; 90: 245-251.

Ellis, D. (2012, September 19). *TAHC Proposes Changes to Current Texas Cattle Trichomoniasis Regulations*. Retrieved from http://www.tahc.state.tx.us/news/pr/2012/2012-09-19_CattleTrichRegulations.pdf

Emmerson M. Trichomoniasis in cattle. *J Am Vet Med Assoc* 1932; 34:636-40.

Fitzgerald PR. Bovine trichomoniasis. *Vet Clin North Am Food Anim Pract* 1986; 2:277-282.

Fox EW, Hobbs D, Stinson J, Rogers GM. A Preliminary Survey of North Carolina Slaughterhouse Bulls for *Tritrichomonas foetus.* Animal Husbandry Newsletter June/July 1995. North Carolina State University.

Grotelueschen DM, Cheney J, Hudson DB, Schweitzer DJ, Kimberling CV, Taton-Allen GF, Nielson KA, Marsh DJ. Bovine trichomoniasis: results of a slaughter survey in Colorado and Nebraska. *Therio* 1994; 42:165-171.

Guest GB. Extra label policy update. FDA Vet 1988; 3:5–6.

Ho MSY, Conrad PA, Conrad PJ, LeFebvre RB, Perez E, BonDurant RH. Detection of Bovine Trichomoniasis with a specific dna probe and pcr amplification system. *Jour Clin Micr* 1994; 32:98-104.

Honigburg BM. *Tritrichomonas foetus.* Parasitic Protozoa 1978; 207-273.

Johnson AE. Incidence and diagnosis of trichomoniasis in western beef bulls. *J Am Vet Med Assoc* 1964; 145:1007-1010.

Kennedy JA, Pearl D, Tomky L, Carman J. Pooled polymerase chain reaction to detect *Tritrichomonas foetus* in beef bulls. *J Vet Diagn Invest 2008*; 20:97-99.

Kittel DR, Campero C, Van Hoosear KA, Rhyan JC, BonDurant RH. Comparison of diagnostic methods for detection of active infection with *Tritrichomonas foetus* in beef heifers. *J Am Vet Med Assoc* 1998; 213:519-522.

Kvasnicka WG, Taylor REL, Huang JC, Hanks D, Tronstad RJ, Bosomworth A, et al. Investigations of the incidence of bovine trichomoniasis in Nevada and of the efficacy of immunizing cattle with vaccines containing *Tritrichomonas foetus*. *Therio* 1989; 31:963–71.

Kvasnicka WG, Hanks D, Huang JC, Hall MR, Sandblom D, Chu HJ, Chavez L, Acree WM. Clinical evaluation of the efficacy of inoculating cattle with a vaccine containing *Tritrichomonas foetus*. *Am J Vet Res* 1992; 53:2023-27.

Love D, Fajt V, Hairgrove T, Jones M, Thompson JA. Metronidazole for the treatment of *Tritrichomonas foetus* in bulls. *BMC Vet Res* 2017; 13:107.

Mukhufhi N, Irons PC, Michel A, Peta F. Evaluation of a PCR test for the diagnosis of *Tritrichomonas foetus* infection in bulls: effects of sample collection method, storage and transport medium on the test. *Therio* 2003; 60:1269-1278.

Mundodi V, Kucknoor AS, Chang T-H, Aderete JF. A novel surface protein of *Trichomonas vaginalis* is regulated independently by low iron and contact with vaginal epithelial cells. *BMC Microbiology* 2006; 6:6.

[a]Ondrak JD, Keen JE, Rupp GP, Kennedy JA, McVey DS, Baker WD. Repeated testing of culture and PCR assay to detect *Tritrichomonas foetus* carrier bulls in an infected Nebraska herd. *J Am Vet Med Assoc* 2010; 237:1068-1073.

[b]Ondrak JD. Tactics for identifying and eliminating *Tritrichomonas foetus* from infected beef herds. Unpublished master's thesis, University of Nebraska, Lincoln, Nebraska. May 2010.

Ondrak JD. *Tritrichomonas foetus* prevention and control in cattle. *Vet Clin Food Anim* 2016; 32:411–423.

Palomares RA, Hurley DJ, Crum LT, et al. Serum, uterine, and vaginal mucosal IgG antibody responses against *Tritrichomonas foetus* after administration of a commercial killed whole T foetus vaccine in beef cows. Therio 2017; 87:235-241.

Parker S, Campbell J, Gahadhar A. Comparison of the diagnostic sensitivity of a commercially available culture kit and a diagnostic culture test using Diamond's media for diagnosing *Tritrichomonas foetus* in bulls. *J Vet Diagn Invest* 2003; 15:460-465.

Parker S, Campbell J, Ribble C. Sample collection factors affect the sensitivity of the diagnostic test for *Tritrichomonas foetus* in bulls. *Can J Vet Res* 2003; 67:138-141.

Parsonson IM, Clark BL, Dufty J. Early pathogensis and pathology of *Tritrichomonas foetus* infection in virgin heifers. *J Comp Path* 1976; 86:59-66.

Perez E, Conrad PA, Hird D, et al. Prevalence and risk factors for *Trichomonas foetus* infection in cattle in northeastern Costa Rica. *Prev Vet Med* 1992; 14:155-165.

Pereira-Neves A, Benchimol M. *Tritrichomonas foetus*: Budding from multinucleated pseudocysts. *Protist* 2009; 160:536-551.

Pereira-Neves A, Campero CM, Martinez A, et al. Identification of *Tritrichomonas foetus* pseudocysts in fresh preputial secretion samples from bulls. *Vet Parasit* 2011; 175:1-8.

Rae DO, Crews JE. *Tritrichomonas foetus*. *Vet Clin Food Anim* 2006; 22:595-611.

Rae, DO. Impact of trichomoniasis on the cow-calf producer's profitability. *J Am Vet Med Assoc* 1989 Mar 15; 194 (6):771-5.

Richardson UF, Kendall SB. Veterinary Protozoology. 1963; 3rd edition.

Schonmann MJ, BonDurant RH, Gardner IA. Comparison of the sampling and culture methods for the diagnosis of *Tritrichomonas foetus* infection in bulls. *Vet Rec* 1994; 134:620-622.

Sergeant E, Toribio J. Estimation of animal-level prevalence from testing of pooled samples. Aus Vet 2004; 1–64.

Skirrow SZ. Identification of trichomonad carrier cows. *J Am Vet Med Assoc* 1987; 191:553–4.

Skirrow SZ, BonDurant RH, Farley J, et al. Efficacy of ipronidazole against Trichomoniasis in beef bulls. *J Am Vet Med Assoc* 1985; 187:404-407.

Strickland LG, Edmondson M, Maxwell H, et al: Surface architectural anatomy of the penile and preputial epithelium of bulls. *Clin Therio* 2014; 6:445-451.

Szonyi B, Srinath I, Schwartz A, et al. Spatio-temporal epidemiology of Tritrichomonas foetus infection in Texas bulls based on state-wide diagnostic laboratory data. *Vet Parasit* 2012; 186: 450-455.

Tedesco LF, Errico F, DelBaglivi LP. Diagnosis of Tritrichomonas foetus infection in bulls using two sampling methods and a transport medium. *Aust Vet J* 1979; 55:180-191.

Trich CONSULT. Retrieved from http://www.trichconsult.org. Accessed on 6/29/2015.

Villarroel A, Carpenter TE, BonDurant RH. Development of a simulation model to evaluate the effect of vaccination against Tritrichomonas foetus on reproductive efficiency in beef herds. *Am J Vet Res* 2004; 65:770–775.

Wang AL, Wang CC. Isolation and characterization of DNA from *Tritrichomonas foetus* and *Trichomonas vaginalis*. *Mol Biochem Parasitol* 1985; 14(3):323-35.

The Changing Landscape of Ruminant Parasite Control: Principles and Approaches for Success and Sustainability

Dr. Ray Kaplan - University of Georgia

The Changing Landscape of Ruminant Parasite Control:

Principles and Approaches for Success and Sustainability

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Contemporary Management of Nematode Parasitism in Livestock

- Evolving attitudes that one can rely on anthelmintics to solve all parasite problems
  - To the neglect of common sense husbandry and pasture management practices
  - In some cases a desire to maximize animal productivity rather than seek to optimize productivity
- How did this attitude come about?
  - Amazing products – both exceptionally effective and relatively cheap
  - This is what we recommended
    - AH combined with knowledge of parasite epidemiology

This Approach Has Largely Supplanted Critical Thinking About Parasite Control

- Rote treatment has become the default strategy
  - Treating too often
  - Treating all of the herd/flock at the same time
  - Treating at times of low environmental refugia
- Little to no diagnostics usually performed
  - Is treatment needed ???
  - If yes, which animals ???
  - Does the treatment work as expected ???

Consequences of This Approach

- Has selected for populations of nematodes that are **RESISTANT** to anthelmintics
  - All major nematode species of all livestock
  - All drug classes
  - No products approved in the US for dogs or livestock containing anthelmintics from new classes since ivermectin in the 1980's
- Consequences of Anthelmintic resistance:
  - Guarantees that efficacy and benefits of treatment are greatly reduced
  - Health and production of livestock are threatened

Are New Classes of Anthelmintics the Answer ???

- They could be …….
- But this is unrealistic
- Resistance is almost certain to outpace the introduction of new anthelmintic classes
- When a new class/product comes, it will need to be used differently, or it will likely have a short useful product lifespan

Why Would We Keep Doing Things The Same Way ???

- Because it is easy ?
- Because that's what we have been doing for a long time ?
- Because this is what I am being told to do ?
- Because I am incapable of changing ?

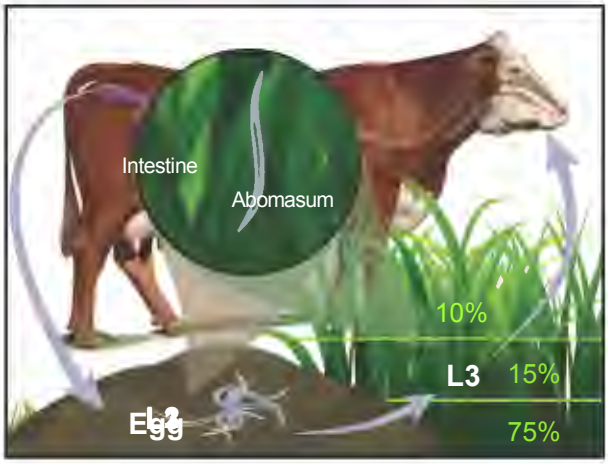
What's Your Recommended Deworming Program ???

We Need to Bring Back Critical Thinking !!!

- Issues for 2020 and beyond are not the same as they were in the 1980's and 90's
  - Drug Resistance
  - Global warming
  - Population growth – need for more animal protein
  - Increasing concerns for animal welfare
  - Public desire for reduced chemical use in food prod'n
  - Concerns regarding ecotoxicity
  - Increasingly stringent regulatory requirements
  - Extreme challenges to developing new anthelmintics for use in food animals (food safety, economical)

How Do We Address These Multiple Issues ???

- We need to re-learn the lessons of parasite biology, ecology, and epidemiology
  - We already know these things – BUT have disregarded them for too long

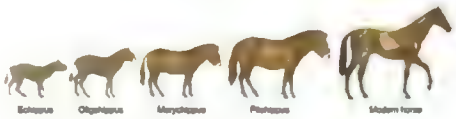


Lessons To Learn

1. Parasites are part of the ecosystem, both biologically and evolutionarily
   - Help to control herbivore populations and protect vegetation from herbivores
   - In a healthy ecosystem there is balance

Lessons To Learn

2. It is normal and natural for livestock to be infected with parasites
   - Co-evolved
   - Infection ≠ disease

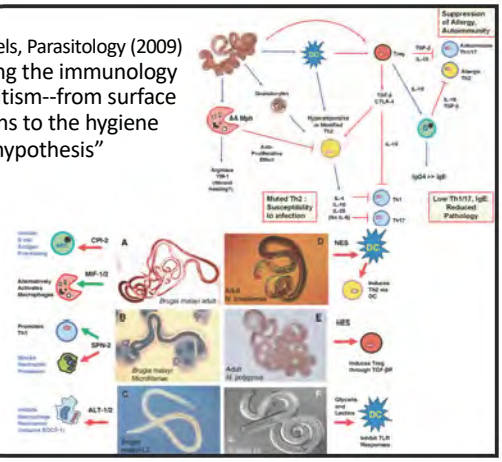


- Are Worms Good for Something ?

Are Worms Good for Something ???

- **The Hygiene Hypothesis**
  - Parasites play an important role in regulating the host immune response
  - Parasites activate Th2 arm or IR, tamps down Th1 helping to suppress exaggerated IR
    - Allergies, inflammatory Dz, autoimmune Dz
- Important for livestock to develop immunity
  - Animals without good immunity are at higher risk for disease if later exposed to high levels

R.M. Maizels, Parasitology (2009) "Exploring the immunology of parasitism--from surface antigens to the hygiene hypothesis"



Science

Helminth infection promotes colonization resistance via type 2 immunity

Deepshika Ramanan, [1,†] Rowena Bowcutt, [1,†] Soo Ching Lee, [1] Mei San Tang, [1] Zachary D. Kurtz, [1] Yi Ding, [1] Kenya Honda, [2,3] William C. Gause, [4] Martin J. Blaser, [5] Richard A. Bonneau, [6] Yvonne AL Lim [7,8] Eng Loke, [7,8] Ken Cadwell [1,9,10,‡]

Lessons To Learn

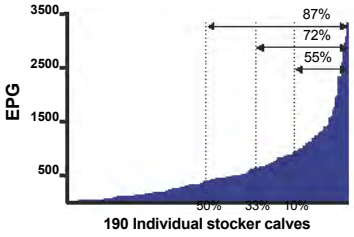
3. It is neither normal or desirable for grazing animals to be kept free of GIN infection and is impossible to achieve
   - Livestock have been raised for centuries
   - Anthelmintics only around for a few decades
   - Eradication of worms is not possible
     - Attempting to eradicate only increases the rate with which drug resistance develops

Lessons To Learn

4. Capacity to tolerate and resist parasites varies among animals in a herd
   - Over-dispersion – 20-30% of animals have 80% of the parasites
   - Except for very young immunologically naïve animals, most animals develop good protective immunity from parasites
     - Infection – Yes
     - Disease – No

Capacity to tolerate and resist parasites varies among animals in a herd

- Worm/egg counts are highly over-dispersed
  - 70 - 80% of worms in 20 - 30% of animals
  - 90% of worms in 50% of the animals



Lessons To Learn

5. Otherwise healthy livestock with low to moderate worm burdens do not require anthelmintic treatment to remain healthy
   - Deworming will not significantly improve health and welfare in many/most of the herd
     - Thus from a health standpoint there is little benefit from treatment in those animals
   - However, production efficiency may suffer
     - This has economic consequences for livestock producers

Premunition

- Immunity that is stimulated by a resident population of worms that restricts the establishment of new worms
  - Treatment removes worms and eliminates state of premunition
  - Empty niche is quickly filled
    - Animal is rapidly re-infected if in a parasite-contaminated environment

How Do We Find the Right Balance Between Productivity and Sustainability ?

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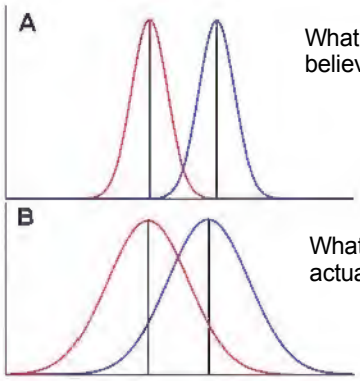
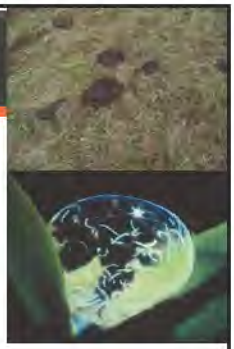
Productivity of Untreated Vs. Treated



Lessons To Learn



6. High numbers of animals per paddock = **more feces** and **more eggs** and **more larval contamination** on the pasture
   - **= more parasites** in animals

Lessons To Learn

7. High numbers of animals in paddock means fewer nutrients per animal available
   - Equals lower host resistance
   - Equals lower host resilience

➢ Must manage forage

Every Pasture Has a Tipping Point

- Lessons 6 and 7 are not separable – they go hand-in-hand
- Animals on poor nutritional plane will lose their immunity and become highly susceptible

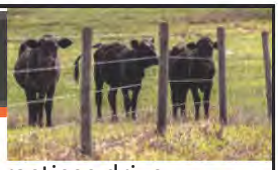
Managing Forages Well Will Help Manage Parasites

- Grass needs energy to grow !!!
- Energy comes from sunlight
- Leaf mass is required to absorb that sunlight
- Grass < 4 inches will have much slower regrowth
- THUS – the optimal approach is to manage forages well !!!
  - Side benefit is improved parasite management

Lessons To Learn

8. Anthelmintic resistant worms do not cause disease !!!
   - High worm burdens due to failed parasite control cause disease !!!
   - Often, if you have a parasite problem, then you have a management problem
     - Solve with improved management, NOT with a magic bullet

Lessons To Learn



9. Modern husbandry practices drive parasite transmission
   - Fences
   - Intensive grazing/production systems
   - Grazing pastures too low
   - Goats evolved as browsers in dry mountain environments
     - Forcing them graze is unnatural

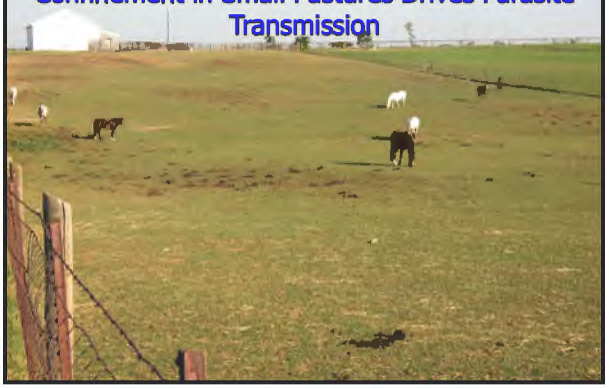
Lessons To Learn

10. It is necessary to appreciate all of these lessons & use them to develop more holistic and **sustainable integrated strategies** to control parasites **(sIPM)**
    - **Anthelmintics** should be **one, but not the only component** of this strategy
    - **Anthelmintics** must be viewed as **extremely valuable non-renewable resources**

Horses in Their Natural Environment

Horses in an Unnatural Environment

Confinement in Small Pastures Drives Parasite Transmission



90% of Strongyle Larvae are in Bottom 4 inches of Grass

75% in bottom 2 inches

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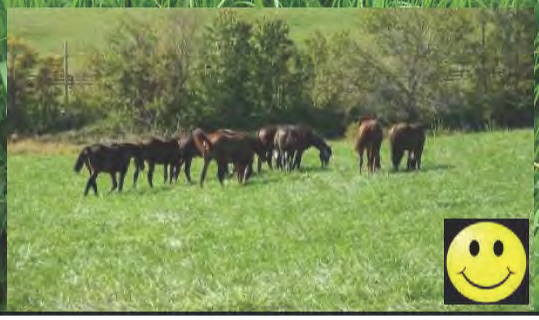

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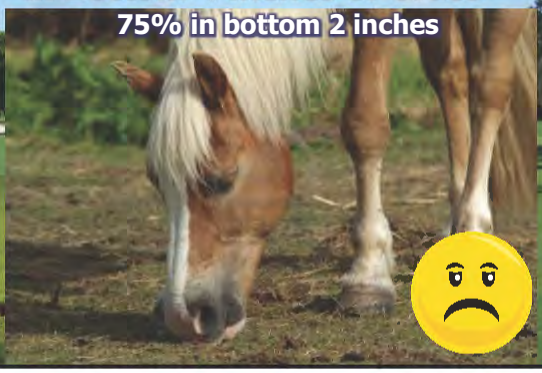


Let Mother Nature Be Your Friend worms don't have legs !!!

- Goats are natural browsers
  - Allow them to browse as much as possible
  - Animals eating with their heads up do not ingest worm L3
  - Animals eating with their heads down do ingest worm L3

What Else Besides Anthelmintics ???

- Grazing management
- Genetically determined host resistance and/or resilience
- Biological control (e.g. nematophagous fungi)
- Bioactive forages (e.g. tannins, flavonoids)
- Other natural products (Cry Proteins from Bt)
- Copper and other minerals
- Vaccines ???

Active Constituents: Each gram contains: a minimum of 30,000 chlamydospores

Daily feeding rates: 100g/100kg bodyweight

Available: Stores & End users

Withholding periods: Meat & Milk: 0 days

Packsizes: 7.5kg and 15kg pails 10kg and 20kg bags (late 2018)

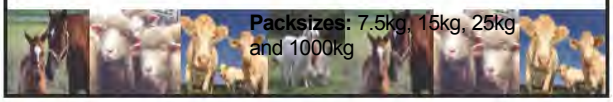
Active Constituents: Each gram contains: a minimum of 500,0000 chlamydospores

Daily feeding rates: 6g/100kg bodyweight

Available: Premixers, Feedmills and Veterinarians

Withholding periods: Meat & Milk: 0 days

Packsizes: 7.5kg, 15kg, 25kg and 1000kg



Optimal Use of Anthelmintics:

- How do achieve the right balance?
  - Route of administration
  - Reducing numbers of Treatments
    - Selective treatment and other refugia-based strategies
  - Using anthelmintic combinations

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Optimal Use of Anthelmintics: Route of Administration

- Oral is BEST – more drug gets into the worms
  - Achieves the highest efficacy
  - Especially if parasites have reduced susceptibility
- Pour-on (topical) is WORST
  - Large inter-animal variability in drug exposure and subsequent high variability in efficacy
  - Will promote the development of resistance

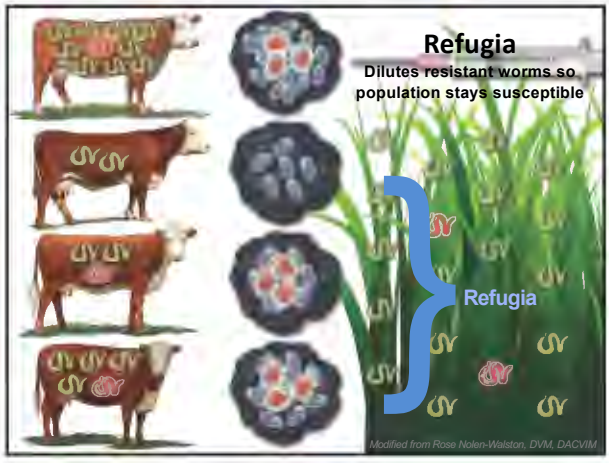
(Lifschitz et al., NZ Vet J, 2017)

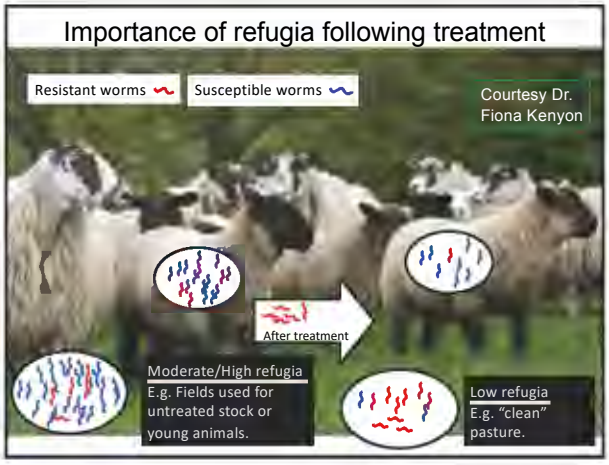
Optimal Use of Anthelmintics: Reducing Anthelmintic Treatments

- Aim of resistance control is to delay the accumulation of resistance alleles
- Reduce drug selection pressure by **managing refugia**
  - = The proportion of the worm population that is not selected by drug treatment
  - Managing refugia provides a means to maintain a majority of drug-sensitive alleles
    - But – must have a majority of S alleles, so **are we too late** ???

Refugia

- Failure to manage refugia is considered the most important factor in the development of anthelmintic resistance
  - Strongly supported by empirical observation
  - Strongly supported by computer modeling
    - Leathwick et al., NZ Vet J (2008), Gabaet al., Vet Parasitol (2010), Leathwick et al., Vet Parasitol (2012), Laurenson et al., Parasitol (2013), Cornelius et al., Vet Parasitol (2016), Berk et al., IJP-DDR (2016)
  - Strongly supported by field research
    - Leathwick et al., NZ Vet J (2008), Waghorn et al., NZ Vet J (2008, 2009), Gabaet al., Vet Parasitol (2010), Leathwick et al., Vet Parasitol (2012), Kenyon et al., Vet Parasitol (2012)





Refugia-Based Strategies for Delaying Resistance

- **Targeted selective treatment strategies used in sheep**
  - FEC (only method for horses)
  - Diarrhea score
  - Body condition score (also cattle)
  - FAMACHA (best method for SR - *Haemonchus*)
  - Milk production (also cattle)
  - Live weight gain (also cattle)
- **Selective non-treatment** (best method for cattle)
  - Treat only 80% or 90% of herd (leave heaviest or best-looking untreated)

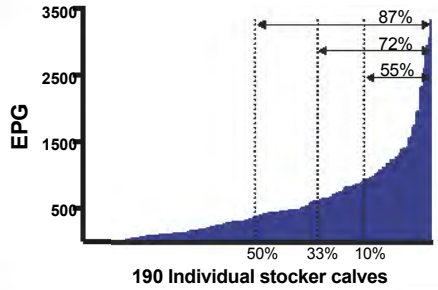
Lambs EID tagged, Treatment decisions monthly

Courtesy Dr. Fiona Kenyon, Moredun Institute, Scotland

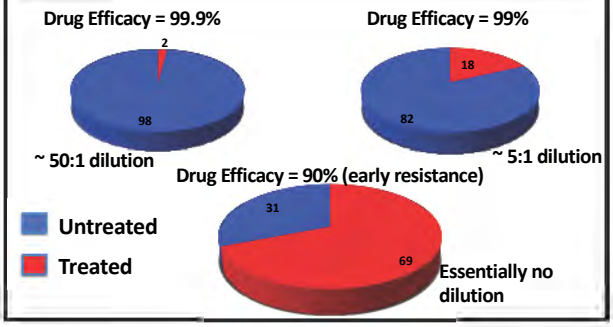
Important Requirement for Success With Refugia-Based Strategies

- **High Efficacy**
  - Refugia-based strategies must use highly efficacious drugs to be effective !!!!

Distribution of FEC in Cattle



Only Treat the 50% of Herd With High Egg Counts (80% of total eggs shed)



How to Achieve High Efficacy ???

- **Use of anthelmintic combinations**
  - Given the common state of reduced drug efficacies (resistance), use of drug combinations is essential

Why Combinations ???

- Results in fewer resistant survivors
  - Greater dilution from refugia
- The evidence tells us that combination anthelmintics are better at:
  - Slowing the development of resistance
  - Controlling worms once they become resistant
- Increases both species-specific efficacy and spectrum of efficacy (more species killed)

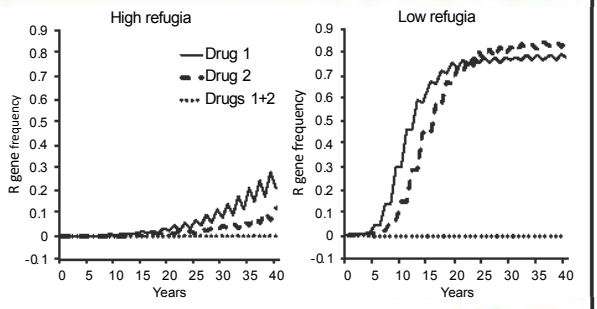
Impact of Using Multiple Drugs on Efficacy (%)

Drug 1	Drug 2	Drug 3	Combination
80	80		96
80	80	80	99.2
90	90		99
90	90	90	99.9
99	99		99.99
60	95		98
60	60	95	99.2
60	60	60	93.6
50	50	50	87.5
40	40	40	78.4

Impact of Combinations on Percent of Resistant Worms that Survive

Efficacy of Dewormer		Single Dewormer	2 Dewormers in Combination	Fold Difference
99	% Killed	99	99.99	1.01x
	% Surviving	1	0.01	100x
98	% Killed	98	99.96	1.02x
	% Surviving	2	0.04	50x
95	% Killed	95	99.75	1.05x
	% Surviving	5	0.25	20x
90	% Killed	90	99	1.1x
	% Surviving	10	1	10x
80	% Killed	80	96	1.2x
	% Surviving	20	4	5x

For example – annual rotation of single actives v continuous use of combination



No resistance initially

Courtesy of Dr. Dave Leathwick

How Does This Work ???

- Combinations slow resistance because more resistant worms are killed
- Benefits of combinations go hand-in-hand with 'Refugia'
  - The presence of 'refugia' is essential to realise the full benefit from combinations
  - **High efficacy maximizes the benefit of refugia**
- "If you accept the principle of refugia, then you **MUST** accept the principle of combinations"
  - **Dr. Dave Leathwick**

This Benefit Will Often Be Invisible to the Farmer (and Veterinarian)

- A farmer will not see a difference between a relatively efficacious single active and a combination

"I don't need a combination, my dewormer seems to work fine"

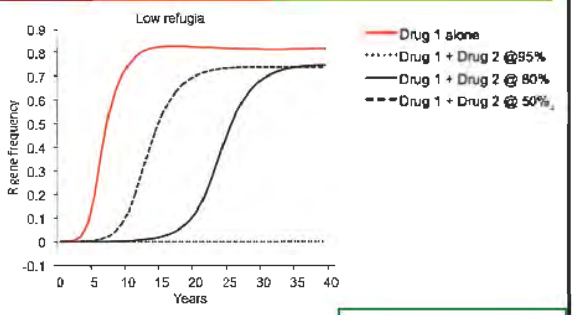
Courtesy of Dr. Dave Leathwick

Two drugs: each 98% effective		
	% killed	% surviving
Single active	98	2.0
Combination	99.96	0.04
Difference	**1.02x**	**50x**

What About On Farms That Already Have Resistance ???

- Evidence suggests that most farms already have resistance in one or more nematode species to one or more drugs
- Can use of combinations still be beneficial ???

Single vs combination with different levels of resistance

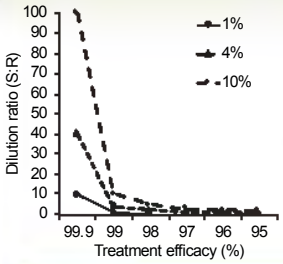


Courtesy of Dr. Dave Leathwick

How Much Refugia is Enough ???

- Maintaining refugia is widely recognized to slow the development of resistance
- BUT -- How much refugia do I need ???
- If I want to leave some animals untreated to supply refugia, what percent of cattle should be left untreated ???

Answer = It Depends



- If treatment is **99.9%** effective leaving **1%** untreated will give ~10-fold dilution
- If treatment is **95%** effective need to leave **34%** untreated to achieve a 10-fold dilution

Size of the refugia required is **highly** dependent on the efficacy of treatment !!!

Courtesy of Dr. Dave Leathwick

Selective non-treatment

Leave a proportion of herd/flock untreated

~ Fold Dilution From Refugia			
Efficacy	**90% Treated**	**80% Treated**	**67% Treated**
99.9	**100**	**250**	**500**
99.5	**20**	**50**	**100**
99	**10**	**25**	**50**
98	**5**	**12.5**	**25**
95	**2**	**5**	**10**

The Future of Parasite Control in Livestock

- Anthelmintic resistance has redefined how parasite control should be practiced
- Development of AR seems almost sure to outpace the development of new drugs
  - **Effective anthelmintics should be thought of as extremely valuable and limited resources**

Use Anthelmintics – BUT– Use Them Wisely

- **Implementation of sustainable strategies is critical -- the sooner the better !!!**
- Benefits are greatest when these strategies are used **BEFORE** there is a resistance problem
  - Too late for most of current drugs
  - **But – getting into these habits now will be critical when the next new drug comes along**

Will These Strategies Really Slow Down the Development of Resistance ???

- **All evidence (so far in sheep) says yes !!!**
- Importantly, using critical thought and evidence-based approaches should **create stronger relationships between producers and veterinarians**
- This should then **result in better overall management that benefits livestock well-being, producer profits, veterinary income and consumer confidence**

Thank You For Your Attention

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UNIVERSITY OF GEORGIA
College of Veterinary Medicine

Field Anesthesia & Surgery Techniques - Food Animal

Dr. Matt Meisner - Kansas State University

LIVESTOCK FIELD RESTRAINT METHODS
(SIMPLE, MULTIMODAL APPROACHES)

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Clinical Professor and Head- Livestock Services
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Introduction:

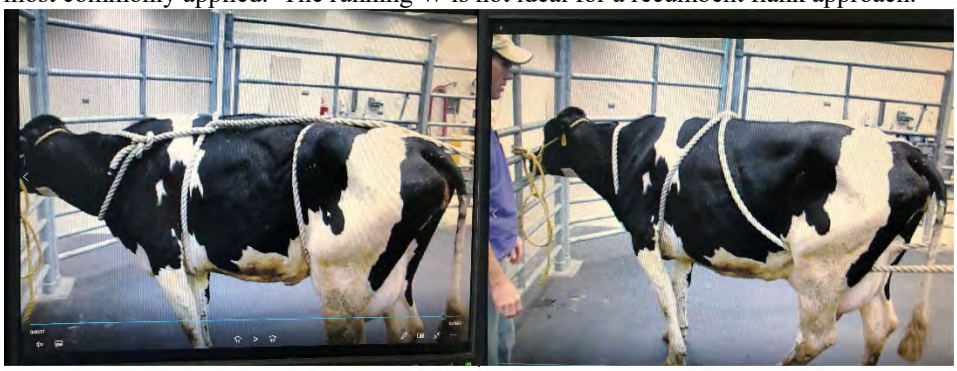
Large animal practice has many physical and mental challenges. Among those challenges, is safely and effectively restraining patients for procedures in the field. We accept the challenge as part of the job, but relish the thought of not having to hit a moving target with the suture needle, dodge the flying hoof like an inside fastball, or be forced to practice our box waltz steps while doing surgery. Mostly we want to provide the most safe, secure, and pleasant care we can while we're doing what we need to do. We have tried and true, very effective methods for physical restraint. A multimodal approach in our field includes both physical and physiologic targets. Squeeze chutes and tables as well as rope restraint methods provide the physical modes in various combinations depending on the situation. A few drugs are at our fingertips for providing the physiologic modes of dissociative sedation/anesthesia, and analgesia. Lidocaine and local anesthesia prevent the "ouch" at the focal location, but many of us have been smashed or kicked working on that totally numb flank. Sedatives, dissociative and systemic analgesics provide the mental distraction needed to reduce reflex reactions and over-ride learned behavior, but ideally want to be used at the minimally effective doses to avoid adverse side effects. To achieve this goal, consider "multimodal" anesthetic and analgesic protocols.

Restraint through low stress handling, secure physical restraint, local/regional anesthesia, and "chemical reasoning" share role. Addressing pain and stress management, reducing risk and increasing safety in both patients and handlers help us get our treatments done. We can look at this as multimodal restraint. Environment, situation, breed, etc., necessitate adjustments. This discussion will provide situations encountered by the author requiring restraint and how they are addressed.

Physical Restraint

Learn how to make a halter. Dr. Dee Griffin has a nice description in text and picture.[2,3] Knowing how to fashion your own head restraint allows you to tailor the length of lead you may need in the field or incorporate the lead in various rope casting methods. Even in squeeze chutes with various head catches, the head has enough movement to cause harm to handlers and the patient, not to mention making it really challenging to accurately incise skin during cosmetic dehorn for example. The halter should be easy to remove and not constrict the airway.

Rope casting methods are a very helpful safety net even in field anesthetized patients. The two most commonly performed are; 1) the Double half hitch, also referred to as the "reefing" method, and 2) the "running W" a.k.a "criss-cross" a.k.a "over and under". Both methods have their pros and cons, but both are safe and effective for inducing and restraining cattle in recumbency. The author prefers the "running W" for midline cesarean sections, as it allows access to the surgical field, and provides hind limb stabilization all in one. But it's harder to cast some cattle with the running W, so the double half-hitch is most commonly applied. The running W is not ideal for a recumbent flank approach.



Invest in chutes and tables that allow safe access to the ventrum and feet. Portable units are very helpful and endless possibilities exist with modifications made to adapt pulley systems and support straps for various situations. Four key knots to learn and know for this method are the 1. Bowline, 2. Tom Fool's knot, 3. Trucker's hitch, and 4. a couple quick release knots. The most complete source for learning these knots in online at www.animatedknots.com.[4]

Local/Regional/Epidural Anesthesia

Block regions when possible, versus locations. Local and regional anesthetics are commonly used in bovine practice for various procedures for both diagnostic and treatment regimes. Local infiltration of lidocaine into infected tissue or inflamed tissue can be ineffective, not to mention potentially scattering the infection with multiple injections. Therefore, when possible utilize local anesthetics at distal locations to the point of interest. Paravertebral blocks for the flank, intravenous regional limb blocks, ring blocks, and point blocks for distal limbs and feet, and epidural blocks for perineal surgery are all well described and effective.

The distal limb can be anesthetized for localizing lameness and surgery by way of intravenous administration of lidocaine after applying a tourniquet proximal to the region allowing for diffusion of anesthetic throughout the distal limb (BAER block). I have performed this procedure on standing cattle multiple times to addresses problems distal to the carpus/tarsus. After applying the tourniquet, allow several minutes to pass before attempting to insert the needle in the desired vein, usually blindly into the dorsal common digital vein. Delay after tourniquet application allows for the distal limb to desensitize to the insertion of the needle. A short extension line from the needle to the syringe is helpful to prevent perivascular injection during inevitable movement by the patient. Alternatively, a "four point" block of the abaxial pastern and interdigital region can be performed for coffin joint lesions or sole abscesses. I have found that the "four point" block is more consistent than a BAER block in small ruminants and camelids where finding a digital vein is more challenging.

Epidural anesthesia with lidocaine at high volumes (~20 to 50 ml (adult cow) in the tail head epidural space) will cause recumbency due to paralysis of the hind quarters. It is recommended that the animal be hobbled for recovery due to the extended time for the anesthetic effects to wear off. Combined with casting rope restraint, a pinch of opioid and a sprinkle of ketamine parenteral make midline cesarean sections a lot more palatable. If we want the bovine patient to remain standing, a rule of thumb for a caudal tail block is 1ml of 2% lidocaine per 200 pound of bodyweight, capping top volume in any patient at about 8cc total.

Keep ~1ml/200lb rule of thumb in mind for general small ruminant use as a caudal epidural for most procedures to remain standing, but I often give higher volumes since a recumbent small ruminant is easier to manage without compromising restraint. Higher volume caudal epidural with lidocaine 2% at 2-3 mL/adult cameid/small ruminant or increased in volume by addition of saline, provides pelvic and caudal abdominal analgesia, in addition to ataxia or motor loss to hind limbs. To provide longer duration, *bupivicaine* may be used, providing analgesia for ~4 hours. A2 agonists may also be used in epidurals, either alone or in addition to local anesthetics. *Xylazine* 20 mg + 1mL of 2% lidocaine, given to an adult alpaca, provides prolonged analgesia, with minimal motor interference. *Lumbosacral epidurals* are a little trickier than caudal epidurals and require aseptic technique, but provide analgesia to the hind limbs, caudal abdomen and pelvis. These can be done with 2% *lidocaine*, and remember, hind limb motor loss will occur. In small ruminants and camelids, 3 mg/kg of 2% lidocaine induces analgesia and motor loss that is good for C-sections, umbilical surgery and relief of urinary obstruction.[5]

Field Anesthesia and Sedation:

Chemical restraint can make procedures more pleasant for practitioners and patients, whether it be light or heavy sedation, or general anesthesia. The enhanced level of cooperation of the patient often improves efficiency to help counterbalance the cost of the drugs used. Of course, individual considerations with drug class use have to be made as to cost:benefits. Finally, food animals require food safety guidelines and drug residue avoidance decisions to be considered with use of sedatives as well as antibiotics. Little is published in this area, and frequently changes. Call FARAD and consider the T1/2 of the drug used. I am comfortable using short acting drugs where elimination is nearly always less than healing times or antibiotic withdrawals for meat. Sedation for examination only, requires an educated estimate. Milk withdrawals "should be" even shorter, but test when possible.

A few things should be taken into consideration when sedating or anesthetizing ruminants to aid in prevention of undue complications. First, ruminants produce a significant amount of saliva while under sedation or anesthesia. Thus, it is important that the patient's head be positioned so that the saliva runs out of the mouth, which is particularly important when the animal is in lateral or dorsal recumbency. As important as salivary pooling in the larynx, is rumen contents from a drug induced rumen atony and positional disruption of the rumen contents. This can be achieved simply by placing a pad under the neck just behind the ramus of the mandible or mid cervical region. The protocols that I prefer allow for some degree of "protective" laryngeal reflexes to remain intact to help prevent aspiration of saliva or rumen contents. Atropine does not necessarily reduce the amount of saliva produced in ruminants, but does make it more viscous which may be detrimental in itself. Atropine also causes reduced intestinal motility and risk of rumen atony. Try to perform as many procedures with the animal standing or in at least semi-sternal recumbency to help prevent (or more readily recognize) rumen tympany and decrease the adverse cardio-respiratory effects. Also, consider other concurrent effects of the drug used, such as xylazine's increase in uterine tone, in addition to sedation of the fetus. Xylazine has a diuretic effect, so not great choice for suspected urethral obstruction cases. Is there a dose dependent effect of the drug used? For the most part there is, therefore using low dose combinations of different drugs may provide desirable restraint without overwhelming individual mechanisms and saturating receptors.

Xylazine:

A suggested dose: Standing sedation **(0.01 to 0.015 mg/lb (.02 to .03 mg/kg) IV)**, Recumbency **0.05 mg/lb (0.1 mg/kg) IV)**[1]

This is the most common drug used in chemical restraint of ruminants, either by itself or in combination with other pharmaceuticals. I rarely use the intramuscular route of administration as I cannot as easily predict the effect. Intravenous administration provides me with a more predictable and faster onset of anesthesia and analgesia, and I can give multiple smaller doses to titrate the effect to the desired level of anesthesia or sedation. All levels of sedation from standing to recumbency can be achieved with xylazine alone. The initial demeanor of the patient does mediate the effect obtained to some extent. There are some dose dependent side effects of decreased GI motility and cardio-respiratory function and increases in uterine tone in late gestation. Use cautiously in compromised patients and/or reverse upon completion of the procedure. I will commonly reverse the effects of xylazine with tolazoline after the procedure, particularly if large amounts of xylazine were given to produce recumbency. Yohimbine has not proven to be as affective of a reversal agent as tolazoline in my hands with ruminants. I use tolazoline at a dose a lot less than the label and have had good success and smooth reversals. The dose I use for tolazoline is about 3X to 5X the milligrams of xylazine given. I also give tolazoline intramuscularly, dependent on the duration of the procedure and the time the last dose of xylazine was given. The recommended emergency reversal dose of tolazoline is 1.8mg/lb (4mg/kg) IV but that is a whopping dose to reverse routine sedation in the author's opinion and deadly complications have been reported.

In the event that your favorite Alpha-2 and reversal are backordered, consider class similarities but note concentrations does and potency. Also call FARAD with recommendations. Dexmedetomidine, medetomidine and atipamezole have been evaluated in cattle and small ruminants.[6-11] There are some inherent concerns using xylazine in sheep regarding increase cardio-respiratory mortality.[6] Combination anesthetic protocols are handy for standing and recumbent procedures and can provide the multimodal goal of anesthetic enhanced restraint. The author feels comfortable using an opiate, alpha-2, and dissociative combination for most procedures.

Some Cattle Protocols

Intramuscular Butorphanol + Xylazine + Ketamine (BXK): Butorphanol (0.005 – 0.013 mg/lb (0.01 - 0.025 mg/kg)) + Xylazine (0.01-0.025 mg/lb (0.02 – 0.05 mg/kg)) + Ketamine (0.02-0.05mb/lb (0.04 – 0.1 mg/kg)).[1] From this combination we get the benefit of a fairly potent ruminant sedative from xylazine but at low dose. Butorphanol, a mild sedative modulates some of xylazine's potency as well as providing analgesia and euphoria. Ketamine provides our dissociative limb of the combination with its affects of amnesia and catalepsy as well as analgesia.

At first glance of this dosage recommendation, it seems a little busy. But if you calculate this dose out for a 1000lb (450 kg) animal you come up with a dose of about:

5mg Butorphanol, **10**mg Xylazine, **20** mg Ketamine at the low range, and

10mg Butorphanol, **20**mg Xylazine, **40**mg Ketamine at the high range.

Notice that we are administering about 2X the amount of xylazine as butorphanol, and 2X the amount of Ketamine as Xylazine. The "5-10-20" is good starting point for tame cattle and Brahman cattle. From this starting point we estimate changes in doses administered. We don't give more than 10 mg of butorphanol, or 20 mg of Xylazine in the initial dose. I personally have given up to 80mg of Ketamine and still maintained a standing patient. If we are going to re-dose during a procedure (try to give 30-40 minutes for the initial dose to fully take effect), then you can re-dose with ½ of your initial Ketamine dose and ¼ of the initial xylazine dose.

In general we have noted up to an hour of cooperation from patients using this protocol. As with anything however, the attitude of the patient prevents blanket success, and we have had some go down, but restraint was maintained.

Other drug combinations for achieving recumbency or subduing wild patients may need to be used in certain situations. The following combinations are some suggestions that the author has used and feels comfortable with.[1]

Intramuscular Xylazine – Ketamine: Xylazine (0.05mg/lb (0.11mg/kg)) – Ketamine (2mg/lb (4.4mg/kg)) administered together in one syringe.

Extremely unruly patients may not go down in a timely fashion with this combination without some assistance. The level of anesthesia and analgesia seems to vary remarkably from patient to patient. Additional IV ketamine or "triple drip" (see below) can be administered to enhance the level of anesthesia and analgesia, if needed. Note that when using ketamine in combination with xylazine, it is important to allow sufficient time for the ketamine anesthesia to resolve (30-45 minutes post IM and 15-20 minutes post IV) before reversing the xylazine.

Intravenous Drips can be used to produce and/or sustain anesthesia.

Sometimes severely cold ambient temperatures may necessitate the use of a warm water bath to run the IV line through if used.

Triple Drip – Ruminant (GKX-Ru):

Triple Drip is 5% guaifenesin to which ketamine (1mg/ml) and xylazine (0.1 mg/ml) have been added. The resulting mixture is administered as a slow IV infusion of 0.5-0.75 ml per pound (1.1 – 1.5 ml/ kg) for induction of anesthesia and can be continued at an infusion rate of ~ 1.2 ml/lb/hr (2.6ml/kg/hr). Recovery time will be prolonged with prolonged duration of infusion during the procedure.

Double Drip:

Double drip is 5% guaifenesin to which **only** ketamine (1mg/ml) has been added. Dose at 0.75-1 ml/lb (1.5-2.2 ml/kg) of BW.

The benefit is less risk of cardiovascular compromise, but a downfall is a decreased level of analgesia. We commonly use this method for induction prior to starting the patient on inhalant anesthetics. If it were to be used in the field for a compromised patient, an analgesic such as Morphine (0.025 – 0.1 mg/lb (0.05 - .2 mg/kg) IM) or butorphanol (0.025 – 0.1 mg/lb (0.05 -.2mg/kg) IM) could be administered.

Telazol-Ketamine-Xylazine cocktail:

Instead of reconstituting the Telazol with 5ml of sterile water, reconstitute it with 250 mg (2.5 ml) of ketamine and 150 mg (1.5ml) of xylazine. Resulting total reconstituted volume is about 4.5ml. Dose 1ml per 275 lb (120kg) IM. Most adult cattle with therefore get the total volume.

This combination has been beneficial for various procedures with rodeo bulls. The cocktail can be given IM and recumbency generally occurs within 10 minutes and provides 45 to 50 minutes of procedure time. The volume of xylazine in this cocktail can become uncomfortably high especially for Brahman influence variation in sensitivity. Reconstituted dose of xylazine to 200 mg has been used by the author and others under select situations successfully, but not routinely recommended. Another option is to reduce the initial dose rate by 25% and re-dose as needed.

Swine version of this cocktail: The amount of xylazine added to the telazol is increased as well as per pound dose. Swine are very resistant to xylazine. The cocktail for swine is Telazol + 2.5 ml (250mg xylazine) + 2.5 (250mg) Ketamine. This cocktail combination is injected IM at a rate of 1ml per 75pounds of bodyweight. Expect recumbent anesthesia for about 40 minutes. Use with extreme caution in very fat marbled commercial breeds (ie Berkshire etc), as well as pet pigs (Vietnamese potbellied). Actually I would not use in VPB pet pigs.

Other Swine

Pigs are very resistant to xylazine. The above cocktail may be over-kill for some procedures or impractical or too costly for in some situations. For large Boar castration, I was taught in veterinary school that you can administer pentobarbital into the testes of a boar for anesthesia. Once the Boar is asleep, you quickly remove the testes and he can then wake up. I was never brave enough to try that, also concerns with food safety also arise in this day and age. An alternate method that has proven successful in my hands, as well as some colleagues, is 1-2mg/kg Xylazine and 3-5mg/kg ketamine administered intra-testicular divided between testicles.[17] Anesthesia restraint has been effective in boars up to 300 pounds and recovery is hastened as once the testicles are removed the boar wakes up.

Some Small Ruminant and Camelid Suggestions

Table 1. Summary of INTRAMUSCULAR Butorphanol-Xylazine-Ketamine combined anesthesia for multiple procedures requiring recumbent anesthesia for up to 30 minutes. Administering 50% of the original dose (ketmine and xylazine) can be used during anesthesia to prolong the effect up to 15 minutes.

Alpacas	0.021 mg/lb butorphanol (0.046 mg/kg)	0.21 mg/lb xylazine (0.46 mg/kg)	2.1 mg/kg ketamine (4.6 mg/kg)
Llamas	0.017 mg/lb butorphanol (0.037 mg/kg)	0.17 mg/lb xylazine (0.37mg/kg)	1.7 mg/lb ketamine (3.7 mg/kg)

If I am performing "mass castration" on 3 or more animals, I will make up a bottle of the cocktail. To a 1 gram (10ml) bottle of ketamine, add; 10mg (1ml) butorphanol and 100mg (1ml) of xylazine. This mixture is then dosed at 1mL/40# (18kg) for alpacas, or 1ml/50# (22kg) for llamas. In my experience, very few of these animals, if handled quietly and plenty of time is given before starting the incisions, will need additional local anesthetic of the scrotum or spermatic cords. Expect 20 minutes of surgical time and the patient should stand 45 min to 1 hour after injection.

I also have performed castrations standing by giving 0.4 mg/kg xylazine IM in alpacas and then infiltrating 1-1.5mL of 2% lidocaine into the median raphe of the scrotum and 2-3mL lidocaine into each spermatic cord. Many animals will lay down with this protocol when placed in a chute, likely behavioral and not related to over-sedation. Nevertheless, control over position and procedure is decreased.

Another method for standing castration utilizes intramuscular butorphanol (0.15 mg/kg IM) in combination with local lidocaine anesthesia as described above. Butorphanol alone will not cause the degree of sedation as xylazine, and the patient will appear alert. The butorphanol should be administered 10 minutes before local anesthesia and castration to allow time for it to take effect.[12]
Additionally, caudal epidural may be used for routine castrations. A clinical study reported on three different methods in alpacas. Method 1; 1.5 mL of 2% lidocaine epidural, which provided perineal analgesia in 2 minutes, but did not alleviate discomfort associated with exteriorization of emasculation of the testicles. Method 2; used 20mg of 20mg/mL xylazine IM and 1 mL lidocaine as an epidural, both 10 min before surgery. This also did not fully alleviate discomfort associated with emasculation. Method 3;

used 20mg/mL xylazine added 1:1 with lidocaine, with 0.75mL of the total solution given epidural. This also did not fully anesthetize the spermatic cord. It is believed that such low-volume caudal epidurals do not move cranially enough to block the lumbosacral plexus, which feeds the structures of the spermatic cord. So, caudal epidurals should be increased in volume, or lidocaine should be infiltrated into the spermatic cords prior to emasculation or ligation.[13]
The procedure used for castration also has some effect on pain responses. A publication reported that pre-scrotal castration, with primary closure, resulted in less incisional pain than did bilateral scrotal castration left open. Pre-scrotal castration does take longer, controlled patient positioning, and requires more attention to sterility, but may be most appropriate for some owners and during fly season.[14]

Dystocia/Cesarean/Uterine Torsion Procedures.
Dystocia is mentioned here as there are some important things to keep in mind when selecting analgesics and sedatives. Low volume lidocaine caudal epidurals (1 mL/200#) are most commonly used, but do not block the cranial vagina and cervix, and therefore may not provide analgesia sufficient to reduce straining and adverse behaviors. Higher volumes (2-3mL/adult female alpaca or ewe) provide a greater area of analgesia, but may result in some temporary loss of motor function to the hindlimbs. Butorphanol 0.05mg/kg, **IV**(not epidural) is an excellent analgesic and sedative in addition to a lidocaine caudal epidural. α_2 agonists should be used cautiously systemically or as part of an epidural when attempting to deliver live crias/kids/lambs, as, in cattle studies, they have been shown to sedate the calf and reduce uterine blood flow and oxygen delivery. In the situation where dam sedation is required, acepromazine (0.02mg/kg, IV) is a better choice, although it does not provide analgesia. I prefer to use a combination of ace and butorphanol.

An INtravenous combination of midazolam (0.2-0.4mg/kg) + butorphanol (0.1mg/kg) and ketamine (2-4mg/kg) is the author's most frequent combination protocol for cesarean-section in sheep and goats. It provides solid restraint, without the risks of xylazine.

The author has used use xylazine for management of uterine torsion and cesarean section despite the potential risks to the fetus. However, by utilizing a combination of butorphanol for these procedures, the volume of xylazine can be reduced. Recognize the potential adverse affects on the fetus and be prepared for management.

Frequently for Small ruminant Cesarean sections, tube cystostomy, perineal urethrostomy, prolapsed amputation, etc, I use <u>lumbosacral</u> lidocaine epidurals in addition to some mild sedation. Do be **cautious with Lidocaine use in Goats** and limit the dose to a maximum of **5mg/kg of bodyweight** to avoid toxicity. This may be one reason to utilize a lower volume of lidocaine with a lumbar-sacral epidural that attempting a higher volume caudal epidural to achieve recumbency and restraint.

Xylazine is our most commonly used drug, used at dosages of 0.1-0.3 mg/kg IV, IM, with the higher doses being IM generally, but effects less predictable. The analgesic properties of this drug last less time than its sedation does and animals are prone to laying down. This is particularly true for camelids, who are prone to "pout". It provides similar visceral analgesia to opioids and flunixin, but its duration is much shorter than flunixin. The preferred reversal agent for xylazine is tolazoline (1-2 mg/kg), given IM or SQ. IV administration (especially the labeled dose) of tolazoline should be avoided as adverse reactions have occurred.[15] Also remember the inherent concerns of xylazine in sheep. .[6]

Local Anesthetics include lidocaine, mepivicaine, bupivicaine and procaine. These block nerve conduction of motor, pain and proprioceptive nerve fibers. They can be used as both local and systemic analgesics, with abilities to stimulate gastrointestinal motility and counter inflammation. Given IV, they act to limit central sensitization in the pain pathway. Local anesthetic side effects depend on the location used, but can include cardiac arrhythmias and ataxia. Toxic doses for small ruminants are much lower than those for large ruminants.

Butorphanol is the drug with which we have the most success in all ruminant-types for enhanced sedation and analgesia. The dose is 0.1 mg/kg IV, IM, or SC, given q 4-6 hours. In a study of llamas, the

elimination half-life for this dose IV was 15.9 +/- 9.1 minutes, while for IM dosing it was 66.8 +/- 13.5 minutes. Therefore, if analgesia is needed for a longer duration than a quick procedure, it should be given IM or SC. We perceive that it provides excellent visceral analgesia in 8/10 animals and is probably the best drug we have for established pain.

Ketamine is a dissociative anesthetic that blocks NMDA and other receptors pre and post synaptically, which play a key role in the pain process of central sensitization. Additionally, it may have potent anti-inflammatory effects, suppressing cytokines and neutrophil chemotaxis. Ketamine is good for established pain, provides analgesia at micro doses, and can be safely used long-term. It does not last long and therefore, needs to be given constantly at 0.4-1.2 mg/kg/hr as a CRI. This is a dosage for cattle and I know of no studies using it in camelids.

Synergistic Groupings of Drugs. As stated before, it is a good idea to use drugs in combination in order to stop the pain cascade at multiple points, and to overcome the inevitable shortfalls of each drug as an individual. Some synergistic groupings include: 1) Xylazine + Opioids, 2) NSAIDs + Opioids, 3) Local anesthetics + Opioids, 4) Ketamine + Xylazine + Opioid + Local anesthetics

Epidural Techniques

Many of the painful conditions we see in small ruminants involve the pelvis, hind limbs or caudal abdomen. The use of caudal and lumbosacral epidurals can provide regional analgesia, often avoiding systemic side-effects and reducing drug costs.

Caudal epidurals, administered between the first two caudal vertebrae, provide analgesia to the perineum. 2% *lidocaine* at1mL/200# is the most commonly used drug for this purpose, providing perineal analgesia and motor loss for about 90 minutes. Higher volume caudal epidural with lidocaine 2% at 2-3 mL/adult alpaca or increased in volume by addition of saline, provides pelvic and caudal abdominal analgesia, in addition to ataxia or motor loss to hind limbs. To provide longer duration, *bupivicaine* may be used, providing analgesia for ~4 hours. A2 agonists may also be used in epidurals, either alone or in addition to local anesthetics. *Xylazine* 20 mg + 1mL of 2% lidocaine, given to an adult alpaca, provides prolonged analgesia, with minimal motor interference.

Lumbosacral epidurals are a little trickier than caudal epidurals and require aseptic technique, but provide analgesia to the hind limbs, caudal abdomen and pelvis. These can be done with 2% *lidocaine*, and remember, hind limb motor loss will occur. In small ruminants and camelids, 3 mg/kg of 2% lidocaine induces analgesia and motor loss that is good for C-sections, umbilical surgery and relief of urinary obstruction.

Cesarean Section/Uterine torsion correction

Luckily dystocia is relatively uncommon in camelids and small ruminants. However caution should be taken with rough fetal repositioning as small ruminants and camelids have more fragile. The most common causes of dystocia are fetal mal-positioning, uterine torsion, and limited cervical dilation or vaginal relaxation (possibly scarring due to previous dystocia). Critically assess the systemic stability of the dam for clinical signs of dehydration, hypotension, and shock and place IV catheter for triage with fluids and anti-inflammatories. Take precautions with vaginal delivery as the uterine and vaginal walls, and cervix in camelids and small ruminants are not as resilient to trauma as cattle. If the cervix is closed, try to obtain viability of the fetus by ultrasonography if possible. A fetal heart rate of between 80 to 120 is normal, bradycardia indicates fetal stress. Uterine fluid should normally appear clear. Rectal palpation, to detect uterine torsion, is possible in most llamas and multiparous alpaca females for clinicians comfortably wearying a 7 ½ surgery glove. In dystocia, if the uterus or fetus is not accessible or the cervix is closed, immediate c-section is indicated.

In cases of uterine torsion, external (rolling) should be attempted initially. The author and colleagues generally give the patient a "two strikes and you're out" chance. If the torsion is not corrected in two rolling attempts, laparotomy is performed. Cesarean section may or may not be performed.

Anesthesia: An IV catheter is in place at the time of surgery and the left flank is surgically prepared. The dam is given 0.05 to 0.1 mg/kg xylazine IV and 0.2mg/kg butorphanol IM. A caudal epidural, 2 to 3ml 2% Lidocaine is administered to reduce straining. A local line block of 2% lidocaine is administered in the left flank where the incision will be made. I prefer to limit my total lidocaine

administration to 10mg/kg (sheep and camelids) and 5mg/kg (goats) including the epidural. Dilute the lidocaine for the line block as needed with saline. The length of the incision will be approximately 20 cm. The dam is placed and maintained in right lateral recumbency with ropes tied over the back in a cushed position.

Procedure:

Approach to the abdomen can be through the flank or by midline celiotomy. The author prefers the left flank approach. Precautions should be taken to assure tissues will be accurately re-apposed during closure. An oblique incision following the orientation of the muscle fibers of the internal abdominal oblique (caudo-dorsal to cranio-ventral) allows accurate re-apposition in a three layer closure.

The uterus should be isolated and in cases of uterine torsion, visually assess uterine wall health. Fetal viability can be assessed by placing an ultrasound probe in a sterile rectal sleeve. Re-assess after detorsion of the uterus. If cesarean section is to be performed, exteriorize the uterine horn and remove the fetus.

Suture the uterus with size No. 0 absorbable suture material in an inverting pattern. Due to the diffuse epitheliochorial placentation, I prefer a two layer closure with the first layer only slightly inverting (nearly appositional), with a second inverting pattern (cushing). Suture the body wall in three layers (transversus and peritoneum + internal abdominal oblique + external abdominal oblique).

Post-operative care should include antibiotics (7 to 10 days) and NSAIDS (3 days). Prolong therapy as needed depending on initial presentation and surgical complications. If the placenta is retained after the first 24 hours after surgery (ie failure of cervical dilation), 250µg of cloprostenol IM (2 doses q 24 hours) can be given. Do NOT give Lutalyse® as toxicity and death has been reported.

Vasectomy in Rams and Bucks.

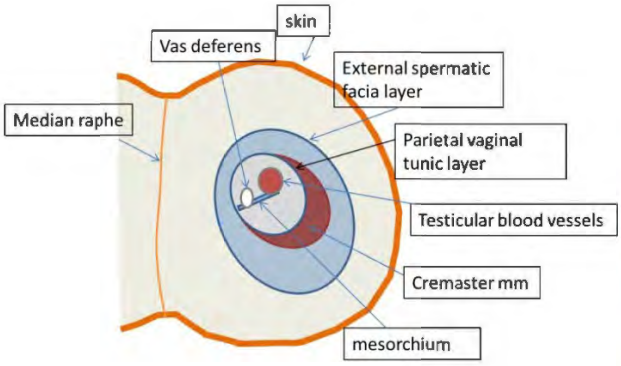
A really nice review of the procedure is described.[16] Vasectomy is preferred over epididymectomy mainly as personal preference or perceived infection and re-cannalation complications. Most commonly this procedure is performed with xylazine sedation alone, usually 0.025 to 0.05 mg/lb of bodyweight IV and local infusion of lidocaine at the neck of the scrotum approach to the testicular cord. It is nice to have assistants restrain the patient in a sitting position.(Figure 1) Clip and prep the scrotum prior to sedation then infuse lidocaine in the area to be incised and perform a final surgical prep after sedation and with the patient in position.

Figure 1: Patient restraint in sitting position. Beneficial to have assistants comfortable as well.

Grasp the neck of the scrotum and palpate the testicular cords. Isolate one cord and incise through the skin and subcutaneous tissue. Then bluntly isolate the testicular cord circumferentially so that the entire cord can be brought through the skin incision. I then stabilize the cord with the index finger to make my incision through the tunic. The Vas deferens will lie on

the medial side of the cord naturally, so try to avoid twisting the cord when exteriorizing it. (Figure 2 and 3) Often you can visualize the vas deferens before incising the tunic. Try to avoid incising the pampiniform plexus. Some prefer to use the back of the scalpel to open the tunic for that reason. Once the tunic is open Isolate the vas deferens and ligate with 2-0 absorbable suture to remove 1-2 inches of vas deferens. Then close the skin with either a monofilament absorbable or nonabsorbable suture. Repeat on the other side. I prefer to give the animal 45 days rest, but some literature would suggest 14 days is sufficient.[8]

Figure 2: Cross section of testicular cord to orient positioning of the vas deferens and other structures within the scrotum as well as layers layers of tissue encountered on approach.



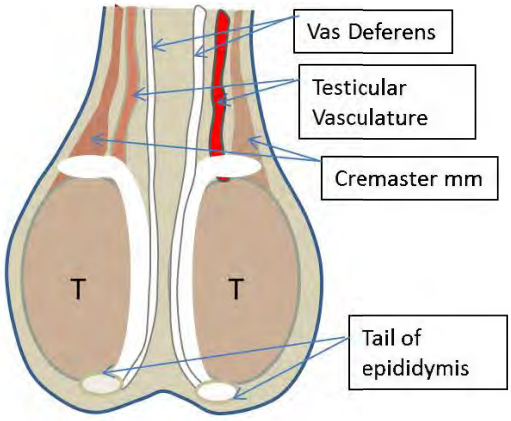


Figure 3: drawing of position of structures within the scrotum for vasectomy.

Conclusion: Multimodal overall restraint is enhanced through multimodal chemical restraint techniques. Species variations are expected as well as situational application.

References:

1. Abrahamsen, EJ. Chemical Restraint in Ruminants. In; VCNA-food animal practice, Field surgery of cattle part 1. July 2008;24(2):227-243.
2. Griffin, D. Easy halter or foot-restraint rope. *Bovine Veterinarian* supplement Jan 2009; p19.
3. http://gpvec.unl.edu/Elective_files/feedlot/KNOTS,Restraint&CattleHandling2016.pdf
4. https://www.animatedknots.com/
5. Scott PR. Extradural Analgesia for Field Surgery in Sheep. Compendium, Food Animal Supplement, March 2000 22(3);S68-S75.
6. Kastner SBR, Kull, S, Kutter APN, et al. Cardiopulmonary effects of dexmedetomidine in sevoflurane-anesthetized sheep with and without nitric oxide inhalation. 2005 AJVR 66(9):1496-1502
7. Cagnardi P, Villa R, Ravasio G, et al. Pharmacokinetics and sedative effects of dexmedetomidine in dairy calves. 2017 NZ Vet Journ. 65(1);14-18.
8. Mattos E, Flaherty D, Nishimura LT, et al. Clinical effects of epidurally administered dexmedetomidine with or without lidocaine in sheep. 2019 Vet Rec
9. Adam M, Raekallio MR, Vainio OM. Sedative effect of intramuscular medetomidine with and without vatinoxan (MK-467), and its reversal with atipamazole in sheep. 2018 Vet Anesth and Analgesia 45;788-793.

10. Roja E, Kerr CL, Enouri SS, et al. Sedative and cardiopulmonary effects of medetomidine hydrochloride and xylazine hydrochloride and their reversal with atipamezole hydrochloride in calves. 2008 AJVR 69:319-321.
11. Shah Z, Hu ML, Qiu ZY, et al. Physiologic and biochemical effects of electroacupuncture combined with intramuscular administration of dexmedetomidine to provide analgesia in goats. 2016 AJVR 77:252-259.
12. Barrington GM, Meyer TF, Parish SM. Standing castration of the llama using butorphanol tartrate and local anesthesia. Equine Pract. 1993;15:35-39
13. Padula AM. Clinical evaluation of caudal epidural anaesthesia for neutering of alpacas. Vet Rec 2005;156:616-17.
14. Baird AN, Pugh DG, Wenzel JG, et al. Comparison of two techniques for castration of llamas. JAVMA 1996;208(2):261-2
15. Read MR, Duke T, Toews AR. Suspected tolazoline toxicosis in a llama. JAVMA 2000; 216(2):227-29.
16. Boundy T, Cox J. Vasectomy in the ram. In Practice, July/Aug 1996;18:330-334
17. Newcomer B and Walz P. In: Farm Animal Anesthesia: cattle, small ruminants, camelids and pigs. 2014 Chapter 8: anesthesia for specific procedures. P 168.

Recommended Reading

Fowler ME. Restraint and handling of wild and domestic animals, 2nd Edition. Wiley-Blackwell, 1995.

Marketing Considerations for Veterinarian Practices

Dr. Doug Walker - Kansas State Univeristy, College of Business

Marketing Considerations for Veterinarian Practices

Doug Walker, PhD – Associate Professor of Marketing
Kansas State University
College of Business

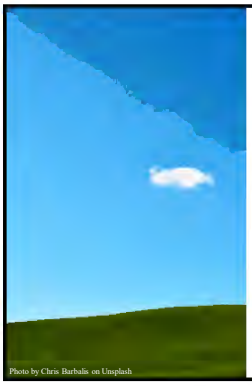
1

What would you pay to acquire a new dog patient?

First, you need to know what a new dog patient is worth to your practice.

And only then can you determine what you can spend on marketing.

2



Objectives for our session

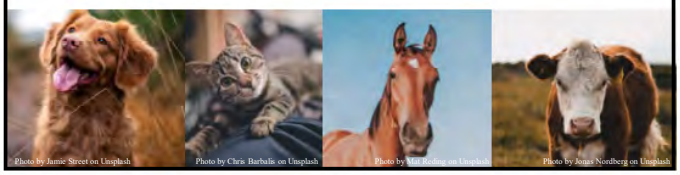
Work through an approach to determining the value of each of your patients,

that can be realistically implemented by you and your staff,

and used to guide your marketing efforts.

3

Lifetime Value (LTV)



4

Value of a particular dog patient at t=0

t=0	t=1	t=2	t=3
Exam $100 - C_E Vaccinations $100 - C_V Neutering $100 - C_N Anti-parasitics $100 – C_P Margin $400 - $100 = $300	Exam $100 - C_E Vaccinations $100 - C_V Anti-parasitics $100 – C_P Margin $300 - $75 = $225	Injury $300 - C_I Margin $300 - $75 = $225	Exam $100 - C_E Vaccinations $100 - C_V Anti-parasitics $100 – C_P Teeth cleaning $100 – C_P Margin $400 - $100 = $300

vetinfo.com
vin.com

5

Value of a particular dog patient at t=0

$$LTV = \sum_{t=0}^{3} \frac{M_t}{(1+i)^t} = \frac{M_0}{(1+i)^0} + \frac{M_1}{(1+i)^1} + \frac{M_2}{(1+i)^2} + \frac{M_3}{(1+i)^3}$$

$$= \sum_{t=0}^{3} \frac{M_t}{(1+i)^t} = \frac{300}{(1+.035)^0} + \frac{225}{(1+.035)^1} + \frac{225}{(1+.035)^2} + \frac{300}{(1+.035)^3}$$

$$= 998.01$$

t=0	t=1	t=2	t=3
Margin = $300	Margin = $225	Margin = $225	Margin = $300

6

Value of an average new dog patient?

$$LTV = \sum_{t=0}^{3} \frac{M_t}{(1+i)^t} = \frac{M_0}{(1+i)^0} + \frac{M_1}{1+i^1} + \frac{M_2}{1+i^2} + \frac{M_3}{1+i^3}$$

$$= \sum_{t=0}^{3} \frac{M_t}{(1+i)^t} = \frac{300}{(1+.035)^0} + \frac{225}{1+.035^1} + \frac{225}{1+.035^2} + \frac{300}{1+.035^3}$$

$$= 998.01$$

t=0	t=1	t=2	t=3
Margin = $300	Margin = $225	Margin = $225	Margin = $300

Probably not!

How old is your new patient?
What kind of health are they in?
Will the client be compliant?
How often will you see the patient?
Will the patient get hurt or sick?
Will the client move, and if so, when?
Will the client change vets?
When will the patient die?
On and on and on ……

7

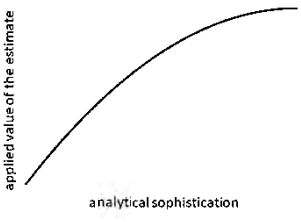
Common realities concerning analytics

Consideration of what is possible with analytics (sophistication) often leads to a complete rejection of analytics (the water gets too deep).

Software and consultants are available, but the cost of those products and services often don't make sense for a practice, especially smaller ones.

8

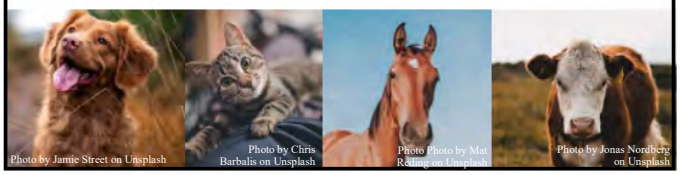
But there is hope: The best kept secret in analytics



Analytics produce educated "guesses" – and barely educated "guesses" are way better than nothing.

9

An Applied Approach to Lifetime Value (LTV)



10

Value of an average new dog patient

Ultimately, all we are going to need to estimate is:

- the average annual earnings for a dog patient in your practice
- and the likelihood of a patient sticking around from one year to the next.

11

Value of an average new dog patient

$$LTV = \sum_{t=0}^{3} \frac{M_t}{(1+i)^t}$$

We considered this for a particular dog patient with the unreasonable assumption of knowing all future cash flows

M	margin (i.e. net cash flow)
i	discount rate

12

Value of an average new dog patient

$$LTV = \sum_{t=0}^{T} \frac{M_t P_t}{(1+i)^t}$$

Here is a standard lifetime value specification that incorporates the uncertainty of future cash flows.

M	margin (i.e. net cash flow)
i	discount rate
P	probability of cash flow

13

Value of an average new dog patient

$$LTV = \sum_{t=0}^{T} \frac{M_t r^t}{(1+i)^t}$$

Often times, the probabilities of the future cash flows are represented using a retention rate.

M	margin (i.e. net cash flow)
i	discount rate
r	retention rate

14

Value of an average new dog patient

$$LTV = \sum_{t=0}^{\infty} \frac{M_t r^t}{(1+i)^t}$$

One modification of this model can be a game changer for practical application.

Since $0 < r < 1$, this infinite series will converge.

M	margin (i.e. net cash flow)
i	discount rate
r	retention rate

15

Value of an average new dog patient

$$LTV = \sum_{t=0}^{\infty} \frac{M_t r^t}{(1+i)^t}$$

$$= \frac{M}{1 - \frac{r}{(1+i)}}$$

No longer a summation – much easier to work with.

Now we just need to estimate:

- average annual margin (M)
- annual retention rate (r)
- discount rate (i)

16

Value of an average new dog patient

$$LTV = \frac{M}{1 - \frac{r}{(1+i)}}$$

$$= \frac{M}{1 - r}$$

We are in a historically low interest rate environment, so let's assume discount rate (i) = 0

Now we just need to estimate:

- average annual margin (M)
- annual retention rate (r)

17

Value of an average new dog patient: Estimating margin (M)

$$LTV = \frac{M}{1 - r}$$

How can you efficiently estimate the average annual margin (M) for a dog patient in your practice?

$$M = \frac{(total\ annual\ dog\ revenues)}{(number\ of\ active\ dog\ patients)}\ x\ (1\ -\ COGS\%)$$

1. Total up your dog revenue for the last year.
2. Count the number of active dog files.
3. Estimate the % of dog revenue that goes to cover costs.

This approach accounts for the percentage of dog patients that are puppies, older, owned by non-compliant clients, injured, sick, accessing additional services, … everything

18

Value of an average new dog patient: Estimating margin (M)

So, if you:

- had 100 active dog patients last year,
- that produced $50,000 in revenue,
- with a COGS% of 25%.

$$M = \frac{(total\ annual\ dog\ revenues)}{(number\ of\ active\ dog\ patients)}\ x\ (1\ -\ COGS\%)$$

$$= \frac{\$50{,}000}{100}\ x\ (1\ -\ 0.25) = \$375$$

19

Value of an average new dog patient: Estimating margin (M)

An assumption:

- the addition of one more dog patient will incur only variable costs (cost of goods sold, supplies, etc.)
- i.e. capacity exists so that an additional patient won't require another exam room, additional staff and equipment, etc.

Obviously, if you are considering the acquisition of 100 new dog patients, capacity may need to be increased and any additional fixed costs would need to be allocated to the new patients

20

Value of an average new dog patient: Estimating retention rate (r)

$$LTV = \frac{M}{1 - r}$$

How can you efficiently estimate the average annual retention rate (r) for a dog patient in your practice?

$$r = \frac{(\#\ of\ active\ dog\ patients\ at\ beginning\ of\ year\ still\ active\ at\ end\ of\ year)}{(\#\ of\ active\ dog\ patients\ at\ beginning\ of\ year)}$$

This approach is difficult because it takes a while to realize a patient is "gone", e.g. client moved, dog died, switched to another vet, non-compliant client, etc.

21

Value of an average new dog patient: Estimating retention rate (r)

$$LTV = \frac{M}{1-r}$$

How can you efficiently estimate the average annual retention rate (r) for a dog patient in your practice?

Incorporate a distribution of common breeds by age …..

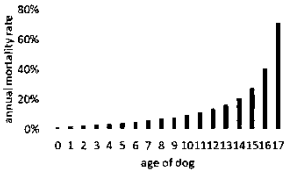
Egenvall, et al. 2005

22

Value of an average new dog patient: Estimating retention rate (r)

$$LTV = \frac{M}{1-r}$$

How can you efficiently estimate the average annual retention rate (r) for a dog patient in your practice?



… annual mortality rates for common breeds by age …

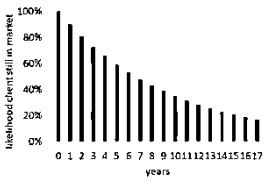
Egenvall, et al. 2005

23

Value of an average new dog patient: Estimating retention rate (r)

$$LTV = \frac{M}{1-r}$$

How can you efficiently estimate the average annual retention rate (r) for a dog patient in your practice?



… and estimates given that 10% of the population moves each year.

24

Value of an average new dog patient: Estimating retention rate (r)

$$LTV = \frac{M}{1-r}$$

How can you efficiently estimate the average annual retention rate (r) for a dog patient in your practice?

Combining each of these factors produces a weighted average retention rate (r) for dogs in a practice of 0.826 (and an average lifetime duration of 7.6 years).

25

Value of an average new dog patient: Estimating retention rate (r)

An assumption concerning the use of the r = 0.826 estimate:

- every practice is about the same in terms of client retention related to client satisfaction, competition, etc.
- if that is unreasonable, ideally go back to estimating

$$r = \frac{(\#\ of\ active\ dog\ patients\ at\ beginning\ of\ year\ still\ active\ at\ end\ of\ year)}{(\#\ of\ active\ dog\ patients\ at\ beginning\ of\ year)}$$

26

Value of an average new dog patient

$$LTV = \frac{M}{1-r}$$

$$= \frac{\frac{\$50{,}000}{100} x(1-0.25)}{1-0.826}$$

$$= \$2155.17$$

So, if you:
had 100 active dog patients last year,
that produced $50,000 in revenue,
with a COGS% of 25%,
along with our estimated retention rate of 0.826

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Some important considerations

- You may not have the software or the time to easily sum up all of the dog-related revenues for the year – maybe just randomly pull 20 dog accounts, sum the revenues for the last year, and divide by 20
- You may need to give some thought to your COGS% estimate - focus on the variable costs, i.e. the cost of the products you are selling and the supplies used to treat your patients
- These estimates will be conservative because puppies will make up a higher proportion of new patients than existing patients, so
  - margin (M) will be underestimated because of the "puppy premium"
  - retention rate (r) will be underestimated due to the low mortality of young dogs

28

Considering other patients

	Average annual retention rate
Dogs	0.826
Cats	0.832
Horses	0.838

O'Neill, et al. 2006
Koskinen 2013

29

Look for ways to increase LTV

Make margin (M) bigger:

- add available products or services
- advocate for additional products or services
- reduce discounts and giveaways
- increase fees (but beware of negative effects on retention rate (r))

Make retention rate (r) bigger:

- enhance client service and satisfaction
- implement retention marketing efforts (but beware of negative effects on margin (M))

30

Value of a marketing campaign

Acquistion cost (AC) = amount spent on campaign / number new patients acquired
Profit = LTV - AC
ROI = Profit/AC

Example – Google paid search:

- $300 campaign yields 4 new dog patients
- Acquisition cost (AC) = $300/4 = $75 ← average health/medical AC = $78.09
- LTV dog = $2155
- Profit = $2155 - $75 = $2080
- ROI = $2080/$75 = 2773% ← huge marginal ROIs due to sunk costs

Irvine 2019

31

Paid search for patient acquisition

Some examples

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Next steps

1. Estimate annual margin (M) for each type of patient.
2. Using your estimates for margin (M) and the estimated retention rates (r), calculate LTV for each type of patient.
3. Check Google search for your placement in search results.
4. Experiment with various marketing campaigns, track the results, and calculate the acquisition cost, profit, and ROI.

Photo by Kishore Ragav Ganesh Kumar on Unsplash

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Evidence-Based Parasite Control in Horses

Dr. Ray Kaplan - University of Georgia

Evidence-Based Parasite Control in Horses

Ray M. Kaplan, DVM, PhD, DipACVM, DipEVPC

Department of Infectious Diseases
College of Veterinary Medicine
University of Georgia
Athens, Georgia

Evidence – Based Veterinary Medicine

- "EBM is the conscientious, explicit and judicious use of current best evidence in making decisions about the care of individuals and populations"

- Why Not For Parasites ???

What's Your Recommended Deworming Program ???

Lessons To Learn

1. Parasites are part of the ecosystem, both biologically and evolutionarily
   - Help to control herbivore populations and protect vegetation from herbivores
   - In a healthy ecosystem there is balance

Lessons To Learn

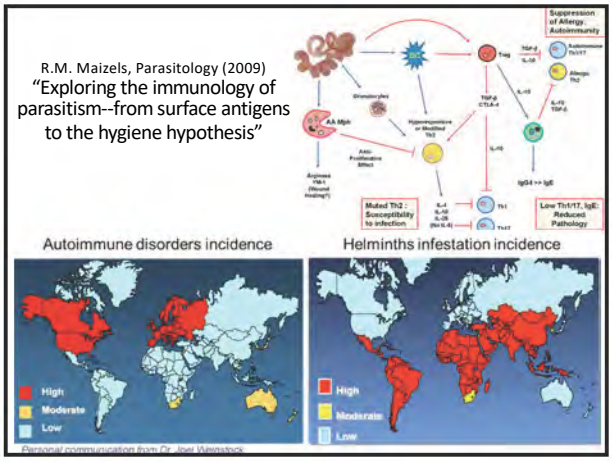
2. It is normal and natural for livestock to be infected with parasites
   - Co-evolved
   - Infection ≠ disease

- Are Worms Good for Something ?

Are Worms Good for Something ???

- **The Hygiene Hypothesis**
  - Parasites play an important role in regulating the host immune response
  - Parasites activate Th2 arm or IR, tamps down Th1 helping to suppress exaggerated IR
    - Allergies, inflammatory Dz, autoimmune Dz
- Important for livestock to develop immunity
  - Animals without good immunity are at higher risk for disease if later exposed to high levels

R.M. Maizels, Parasitology (2009)
"Exploring the immunology of parasitism--from surface antigens to the hygiene hypothesis"




International Journal for Parasitology
Invited Review
Translatability of helminth therapy in inflammatory bowel diseases
Joel V. Weinstock, David E. Elliott

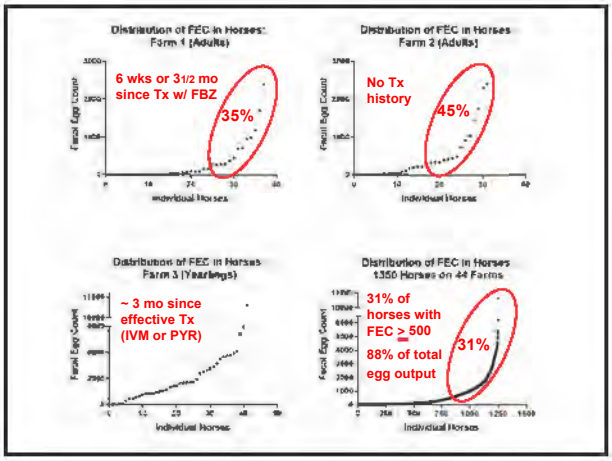

International Journal for Parasitology
Invited Review
Helminth mediated modulation of Type 1 diabetes (T1D)
Paola Zaccone, Anne Cooke

Lessons To Learn

3. It is neither normal or desirable for grazing animals to be kept free of GIN infection and is impossible to achieve
   - Livestock raised for centuries
   - Anthelmintics only around for a few decades
   - Eradication of worms is not possible
     - Attempting to eradicate only increases the rate with which drug resistance develops

Lessons To Learn

4. Capacity to tolerate and resist parasites varies among animals in a herd
   - Parasites are not equally distributed in groups of animals
   - Over-dispersion: 20-30% of animals have 80% of the parasites
     - ~95% of worms in 50% of the animals
   - Except for very young immunologically naïve animals, most animals develop good protective immunity from parasites



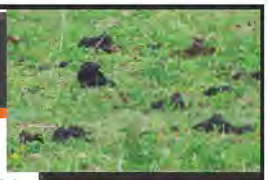
Lessons To Learn

5. Otherwise healthy horses with low to moderate worm burdens do not require deworming to remain healthy
   - ✧ Thus from a health standpoint there is little benefit from treating those animals
     - ✧ Infection – Yes
     - ✧ Disease – No !!!

Premunition

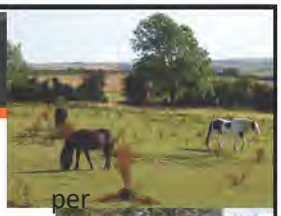
- ❑ Immunity that is stimulated by a resident population of worms that restricts the establishment of new worms
  - ■ Treatment removes worms and eliminates state of premunition
  - ■ Empty niche is quickly filled
    - ❑ Animal is rapidly re-infected if in a parasite-contaminated environment
    - ❑ **In horses, removal of adult cyathostomins stimulates emergence of encysted L4**
      - ■ **This is the process that causes disease !!!!!**

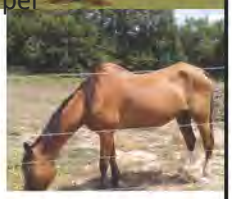
Lessons To Learn



6. High numbers of horses per paddock **= more feces** and **more worm eggs** and **more worm larvae** on the pasture
   - ◆ **= more parasites** in horses

Lessons To Learn





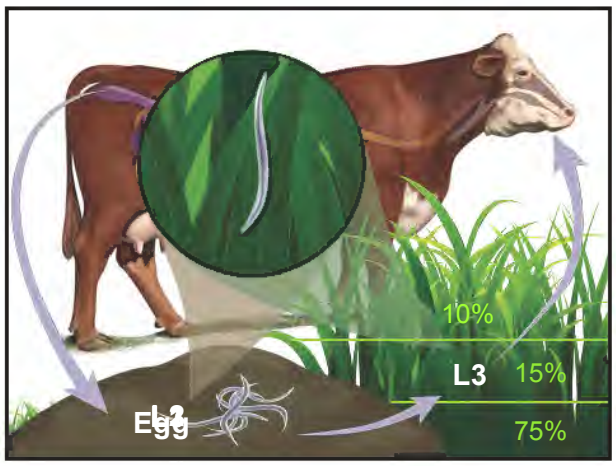
7. High numbers of horses in a paddock means fewer nutrients per horse are available
   - ◆ Equals lower host resistance
   - ◆ Equals lower host resilience
   - ◆ Equals deterioration of pasture quality

Lessons To Learn

8. High worm burdens **due to failed parasite control** cause disease !!!
   - ■ Frequent deworming won't prevent this !!!
     - ❑ Especially if using drugs that don't work because of resistance
     - ❑ Most drugs don't kill encysted stages
   - ✧ If you have a parasite problem, then you have a management problem !!!
   - ✧ Solve with improved management, NOT with a magic bullet

Lessons To Learn

9. Modern husbandry practices drive parasite transmission
   - ◆ Fences
   - ◆ Intensive grazing/production systems
   - ◆ Grazing pastures too low
   - ◆ Grazing horses segregate pastures
     - ✧ Lawns and roughs
     - ✧ roughs have 15X increase in risk



Managing Forages Well Will Help Manage Parasites

- Overgrazing damages the pasture and reduces soil fertility:
  - Compacts the soil, reducing water infiltration and moisture holding capacity
  - Stunts root growth
  - Reduces leaf mass
  - Encourages weed growth
- Grass needs energy to grow !!!
  - Energy comes from sunlight
  - Leaf mass is required to absorb that sunlight

Managing Forages Well Will Help Manage Parasites

- Grass < 4 inches will have much slower regrowth
- 90% of worm larvae are below 4 inches
- THUS – the optimal approach for parasite control is to manage forages well !!!
  - Move horses when pasture height is down to 4 inches
  - Let grass regenerate until it reaches 8 inches or more
- Get Double the benefits:
  - Better quality pastures and nutrition for your horses
  - **IMPROVED PARASITE MANAGEMENT**

Lessons To Learn

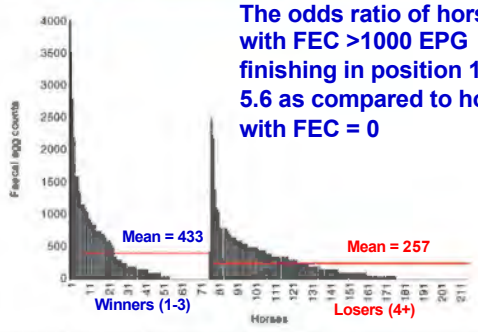
10. It is necessary to appreciate all of these lessons & use them to develop control strategies for parasites that are more holistic:
    - **sustainable Integrated Parasite Management (sIPM)**
      - **Anthelmintics** should be **one, but not the only component** of this strategy
      - **Anthelmintics** must be viewed as **extremely valuable non-renewable resources**

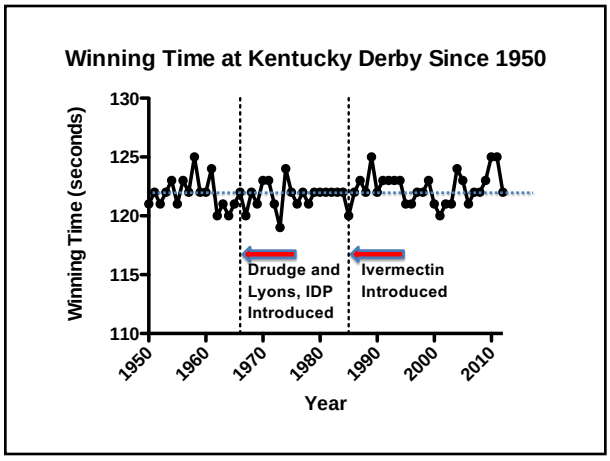
Do Worms Make Horses Run Faster ???

EQUINE VETERINARY JOURNAL

Strongyle egg counts in Standardbred trotters: Are they associated with race performance?

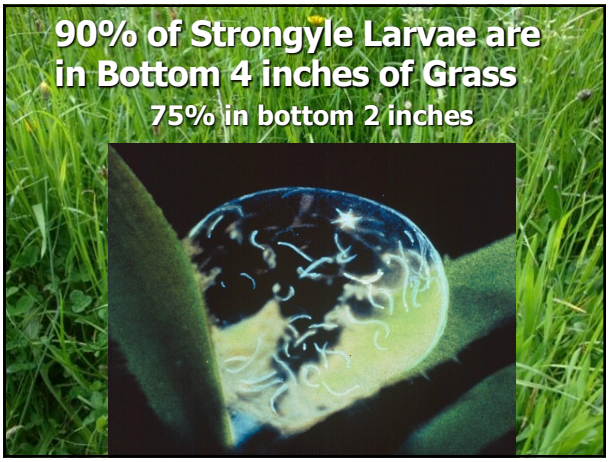
The odds ratio of horses with FEC >1000 EPG finishing in position 1-3 was 5.6 as compared to horses with FEC = 0




Winning Time at Kentucky Derby Since 1950
Winning Time (seconds)
130
125
120
115
110
1950
1960
1970
1980
1990
2000
2010
Year
Drudge and Lyons, IDP Introduced
Ivermectin Introduced


Horses in Their Natural Environment


Horses in an Unnatural Environment
Confinement in Small Pastures Drives Parasite Transmission


90% of Strongyle Larvae are in Bottom 4 inches of Grass
75% in bottom 2 inches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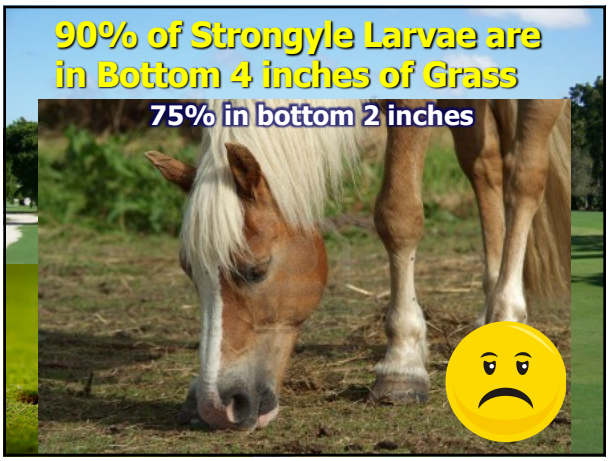
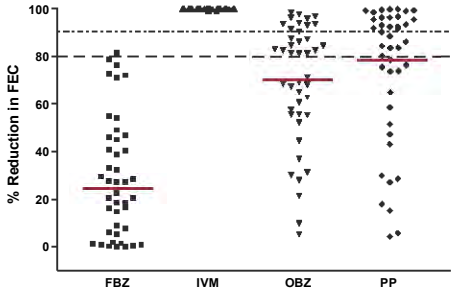

90% of Strongyle Larvae are in Bottom 4 inches of Grass
75% in bottom 2 inches

Prevalence of Resistance
44 Farms - Southern US (2001)

Result	FBZ	OBZ	PP	IVM
Sensitive >90% reduction	0 (0.0%)	10 (23%)	19 (45%)	43 (100%)
Resistant <90% reduction	43 (100%)	33 (77%)	23 (55%)	0 (0.0%)

Prevalence of anthelmintic resistant cyathostomes on horse farms
Kaplan, et al., JAVMA, 225(6): 903-910, 2004

Distribution of FECR By Farm and Drug



2014 Data: Mid-Atlantic States

- "Efficacy of major anthelmintics for reduction of fecal shedding of strongyle-type eggs in horses in the Mid-Atlantic region of the United States"
  - *Veterinary Parasitology* 2015: 214(1-2) pp.139-143
- **% of horses showing FEC reductions of greater than 90%:**
  - Fenbendazole: 6%
  - Oxibendazole: 21%
  - Pyrantel: 43%

Resistance Prevalence Data Unavailable in Other Regions of the US

- However, there is no evidence or reason to believe that resistance prevalence is much different in other region of the US
- Studies have been performed in multiple other countries
  - Results tend to be similar
    - High to very high resistance to benzimidazoles
    - Low to moderate resistance to pyrantel
    - None to low resistance to macrocyclic lactones

JAVMA Vol 245, No.8, October 15, 2014

Comparison of a single dose of moxidectin and a five-day course of fenbendazole to reduce and suppress cyathostomin fecal egg counts in a herd of embryo transfer–recipient mares

Maren E. Mason, MPH; Nathan D. Voris, DVM, MBA; Hunter A. Ortis, DVM; Amy A. Geeding, MS; Ray M. Kaplan, DVM, PhD

- We Identified a large commercial herd of embryo transfer–recipient mares from diverse geographic locations across the United States and performed a study on this farm in 2013

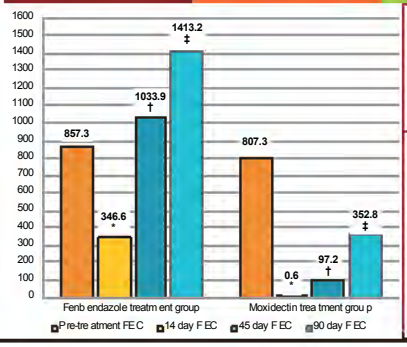
How Effective are Currently Used Larvicidal Drugs in the US ???

- The study farm had hosted transient embryo transfer–recipient mares since 2004
  - Prior to this, the property had been used as an alfalfa hay field and cattle feedlot
  - Subsequent to arrival on the farm there was no exposure to either fenbendazole or moxidectin

Results:
Fecal Egg Count Reduction Test (FECRT)

	% Reduction Compared to Day 0	
Days Post-Tx	**FBZ x 5 Days**	**MOX**
14 day	**41.9% (0-1850 epg)**	**99.9% (0-25 epg)**
45 day	**-57.7% (0-3650 epg)**	**77.5% (0-850 epg)**
90 day	**-125.0% (550-2575 epg)**	**4.9% (0-1600 epg)**

Mean FEC (EPG)



Statistically significant (P< 0.01) difference between treatment groups at 14, 45 and 90 days post-treatment

Environmental impact:

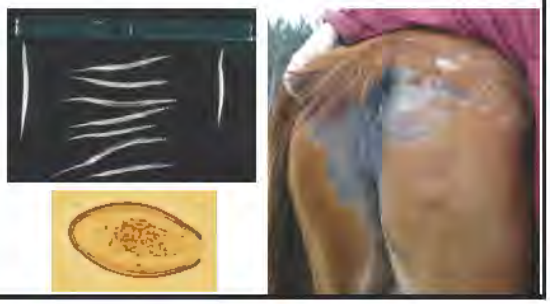
On avg at 14, 45, and 90 days, FBZ mares shedding > 500, 10, and 4x more eggs than MOX mares

Horses

- *Parascaris spp.* (roundworms)
  - Most important parasite of foals

Horses

- Oxyuris equi (pinworms)



What Drives the Rate of Development of Drug Resistance ???

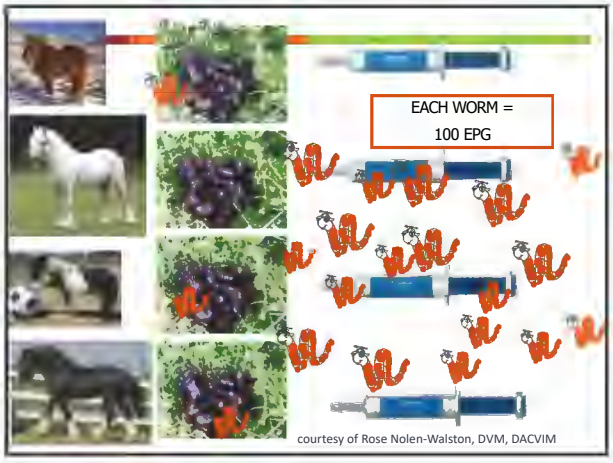
- The rate of development of drug resistance is proportional to the magnitude of the drug selection pressure placed on a given worm population
- How we use drugs directly effects this selection pressure
  - Rate of resistance can be reduced

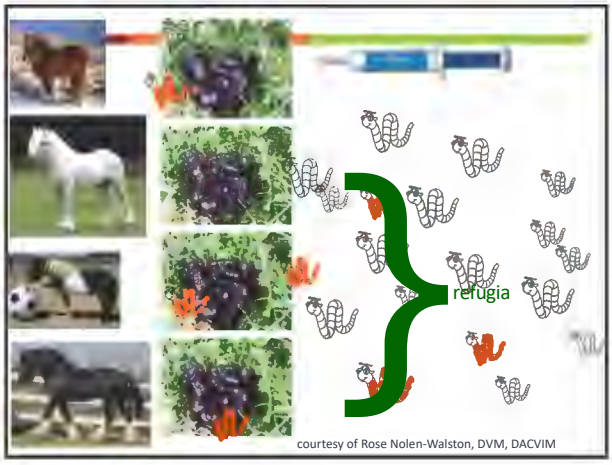
Control of Resistance
How Do We Achieve It ???

- Aim of resistance control is to delay the accumulation of drug-resistance genes in the pop'n (worms with drug-resistance)
- Reduce drug pressure by **managing refugia**
  - = The proportion of the worm population that is not selected by drug treatment
  - Managing refugia provides a means to maintain a majority of drug-sensitive worms
    - But – must have a majority of drug-susceptible worms, so **are we too late** ???

Maintaining **Refugia** is Key

- **Failure to manage refugia is the single most important factor effecting the rate of drug resistance development**
- Refugia dilute out resistant worms so that drug-sensitive worms remain in the majority
- Where do refugia come from ???
  - eggs/larvae already on pasture
  - Worms in horses that are not treated with dewormer
  - Stages of worms in treated horses that are not effected by the drug (encysted larvae)





We Are At Risk Of Having No Effective Dewormers In The Near Future

- New drug classes introduced every decade during 50's, 60's, 70's, and 80's
- No new drug classes introduced since ivermectin in 1981 - Moxidectin (Quest) in 90's, but same class
- **"We have what we have"**
  - **No** new equine dewormers expected in near future
    - >10 years, probably much longer
- **Effective dewormers must be thought of as extremely valuable and limited resources**
  - What happens when we have no effective dewormers left ???

Major Parasites of Adult Horses

- **Cyathostomins** - Small strongyles
  - primary target of worm control program
- Tapeworms - *A. perfoliata*
- Bots - *Gasterophilus spp.*

→ Secondary Targets for Control

- Large strongyles - *S. vulgaris*
- Pinworms - *Oxyuris equi*
- Others
  - *Habronema/Draschia, Onchocerca, Dictyocaulus*

→ Treat on a case by case basis if not adequately controlled by targeting 1° and 2° species

Major Parasites of Foals (up to 12 months)

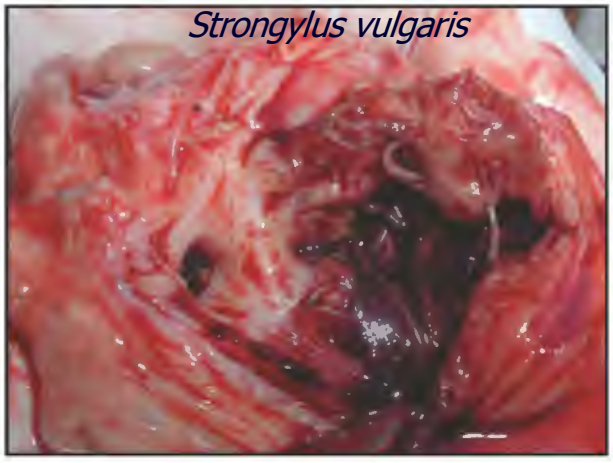
- *Parascaris spp.*
  - Roundworms
  - primary target of worm control program
- Cyathostomins (Small strongyles)
- *Strongylus vulgaris*
- *Strongyloides* - Threadworms
  - keep foaling stalls clean and dry

Strongylus vulgaris

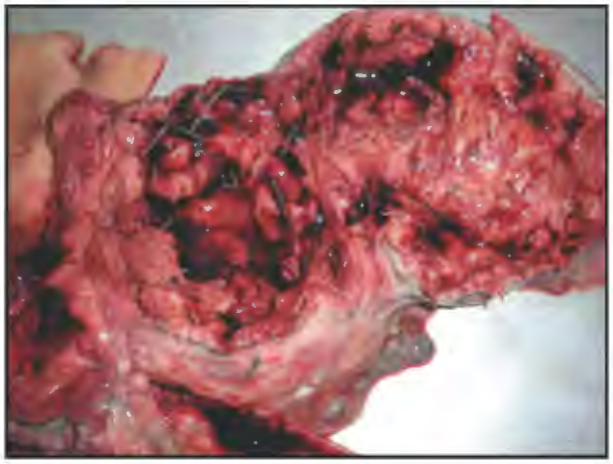
- Historically, the most important equine parasite
  - Most pathogenic helminth of horses
  - 90% of colics due to *S. vulgaris* ?
  - Now very uncommon



Strongylus vulgaris



Strongylus vulgaris



Equine Cyathostomins (Small Strongyles)


- **Now considered the principal parasitic pathogen of horses**

Importance of Cyathostomins

- Primary target of worm control programs
  - Usually mild pathogens but with serious pathogenic potential
- Encysted larvae in mucosa are the most pathogenic stage
- Prevalence = 100% – cyathostomins are ubiquitous in equids worldwide
- Disease is uncommon and usually subclinical; severe disease is rare

Host-Parasite Dynamics Cyathostomins

Larval Cyathostominosis
A Rare and Worst Case Scenario

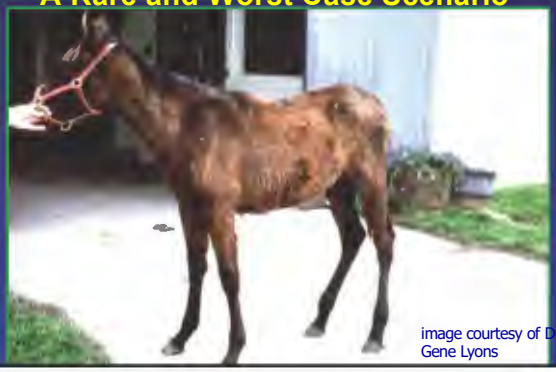
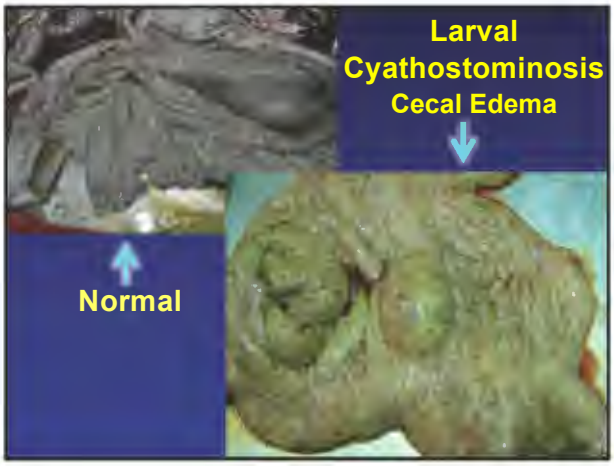
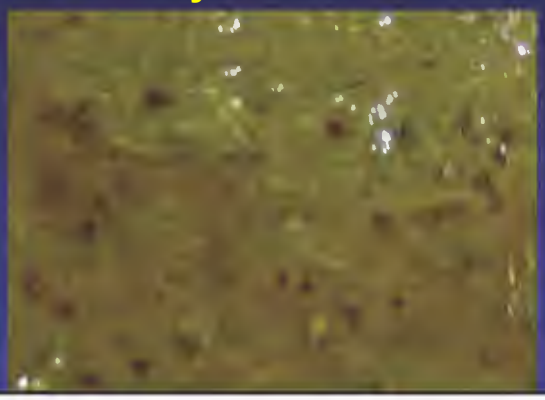


image courtesy of Dr. Gene Lyons



Larval Cyathostominosis



Larval Cyathostominosis

Larval Cyathostominosis

Fear of Parasites in Mature Horses is Highly Overrated

- Frequent mind-set problem to overcome
- Cyathostomins cause little harm unless very large numbers accumulate in the horse
- Most other parasites cause little disease unless relatively large numbers accumulate
- **Disease from worms in adult horses is a rare event**
  - Parasitism is a natural state !!!!
  - Worms are a normal part of the gut fauna of all animals – particularly grazing animals

Why Am I Deworming This Horse ???

Why Am I Treating This Horse With Anthelmintic ???

Do I really need to treat this horse ???

- Is there a clinical justification for treating this horse ???
- What parasite am I trying to kill ???
  - What stages of that parasite are likely present ???
- Why did I pick this dewormer ???
  - Will it kill the desired parasite(s) and stage(s) ???
- How confident am I that it will work as intended (no resistance to that drug) ???
- Are there better options -- is it the best choice for this horse ???

Goal of Parasite Control is NOT to Eliminate All Parasites

- Eradication of worms is not possible, nor is it desirable
  - Attempting to eradicate only increases the rate with which drug resistance develops

Then What Are We Trying To Achieve ???

- Primary goal is to keep parasite infections below levels that produce disease
  - Secondary goal is to preserve anthelmintic efficacy while maintaining good control
- Important for horses to develop immunity
  - Frequent movement of horses virtually ensures exposure to parasites
  - Horses without good immunity are at higher risk for disease

How Do We Use Anthelmintics To Achieve Control of Strongyles?

- Prevent environmental contamination with large numbers of eggs ⇒ prevent future infections
  - Larval stages are the MOST pathogenic
  - Killing adult worms is too late to greatly help
- Treatments must be timed to kill adult worms before they produce substantial numbers of eggs
  - **BUT** – only when those eggs will survive and develop into infective L_3 larvae

Traditional Approach to Worm Control in Horses

- Interval Dose Program
  - Drudge and Lyons (Kentucky, USA) **1966**
  - Tx with dewormers every 6 – 8 weeks
  - **Designed to control *Strongylus vulgaris***
    - At the time, cyathostomins considered little more than nuisance parasites
- Highly successful – revolutionized equine health and parasite control
  - Became gospel and was blindly followed

BUT --- Much Has Changed in the last 50 Years

The traditional "Drug-Based" approach to parasite control usually fails to perform as desired and only makes the drug resistance problem worse

5 Important Reasons Why The Traditional Approach Often Fails to Perform as Desired

1. *S. vulgaris* is very uncommon and is no longer the primary target
2. Cyathostomins are the primary target, BUT – these are different parasites with different biology:
   - Optimal control requires a very different approach

Traditional Approach Often Fails to Perform as Desired

3. Drug given is often not the best choice, and treatment interval is not optimal
   - Worms are resistant to the drugs !!!
   - Time of year – effects which parasites and stages are present
   - Different drugs suppress egg output for different amounts of time after treatment
   - Often there is a huge amount of egg shedding onto pasture despite frequent deworming

Traditional Approach Often Fails to Perform as Desired

4. Most anthelmintics do not kill encysted cyathostomin larvae
   - Most of the worms in the adult horse are in the encysted stages
   - Most damage is done when the larvae emerge from the cyst
   - Killing adult worms with a dewormer stimulates larvae to emerge from the cysts
5. All horses are treated the same despite large differences in need for treatment

Rotation of Dewormers Is This A Good Idea ???

- Rotation is illogical !!!!
  - On many farms, a sensible rotation is not possible because of drug resistance
  - Leads to failed worm control
- Creates a false presumption among horse owners and veterinarians that they have a bona-fide resistance prevention program
- Rotation will mask resistance
  - Drug resistance is extremely common but few realize they have resistant worms in their horses

Case Study: "Contraband"

- Mar: Anthelcide
- Apr:
- May: Panacur
- Jun:
- Jul: Ivermectin
- Aug:
- Sep: Strongid
- Oct:
- Nov: Anthelcide
- Dec:
- Jan: Panacur
- Feb:

Rather Than Calendar - Based Rotation

- Must determine which drugs are effective on a particular farm – for both strongyles and *Parascaris* spp. (if foals present)
- Better approach is to make best medical use of each drug
  - at particular times of the year to target particular parasites or stages
  - in particular animals
  - for particular program goals
  - keep egg reappearance periods in mind
  - Combination treatment is optimal

Anthelmintics "Dewormers"

- What is the **Best** dewormer?
  - There are only 3 major groups of dewormers
- What is the **Most Appropriate** dewormer?
  - Good parasite control programs use a variety of drugs
  - Any given dewormer will not work against some parasites (or parasite stages) but will on others
  - Any given dewormer will not work on some farms but will on other farms
  - Treatment programs need to change over time

"Evidence- Based" Approach to Parasite Control

- Treatment decisions based on:
  - Biology of parasites
    - that are important – NOW
  - Biology of host – parasite relationship
  - Dynamics of resistance development
  - Resistance status of worms on a farm
  - Efficacy of drugs against particular parasites and stages of development
  - Needs of individual patients

Veterinary Involvement

- In early years vets were highly involved in worm control
  - Naso-Gastric tube
- Over the past 20+ years:
  - Substantial decrease in veterinary involvement
  - Recipe approach to worm control
- Suggested evidence-based approach requires veterinary involvement
  - Significant diagnostic needs
  - Sometimes complicated interpretation of results

Important Considerations for Cyathostomin Control

But Which are Rarely Considered

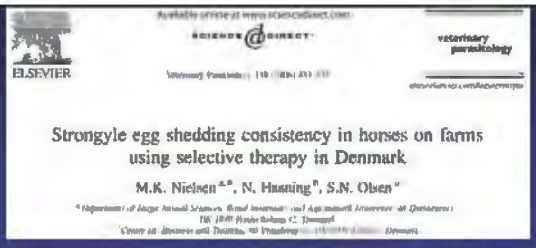
1. Majority of worms exist as encysted larvae in the mucosa
   - Can remain in mucosa for many months
2. Most anthelmintic treatments have no effect on mucosal larvae
3. Effective Tx of adults stimulates larval emergence and re-population of the lumen with adults
4. Greatest pathology occurs when larvae come out of the mucosa

Important Considerations for Cyathostomin Control
But Which are Rarely Considered

5. Many horses develop good immunity and have consistently low FEC
   - Many horses are dewormed with very low or negative FEC
   - Not treating these horses will:
     - Have little impact on horse health
     - Have little impact on overall level of control
     - Save $ and reduce selection for resistance
6. Whether FEC are high or low, there is a great degree of consistency

"The Danish Experiment"

- August 1st 1999: Legislation enacted placing all anthelmintics by prescription only
  - Treatment may be given based on:
    - diagnostic results
    - general knowledge of parasite status on farm
    - strong suspicion of infection
- Similar legislation enacted in Sweden, Netherlands and Finland. others
  - Based on an EU directive


ELSEVIER

Strongyle egg shedding consistency in horses on farms using selective therapy in Denmark

M.K. Nielsen, N. Haaning, S.N. Olsen

- 424 horses on 10 farms using a selective treatment regime over a 3-year period
- FEC were performed twice yearly
  - March-May and August-September
- Horses w/ FEC > 200 EPG treated

Strongyle egg shedding consistency in horses on farms using selective therapy in Denmark

M.K. Nielsen, N. Haaning, S.N. Olsen

Table 1
Calculated probabilities for outcomes of the faecal egg counts, when the results of the two previous egg counts were known

Results of two previous egg counts	Result of the third egg count	Probability (%)

Egg counts are given as eggs per gram (EPG) faeces.

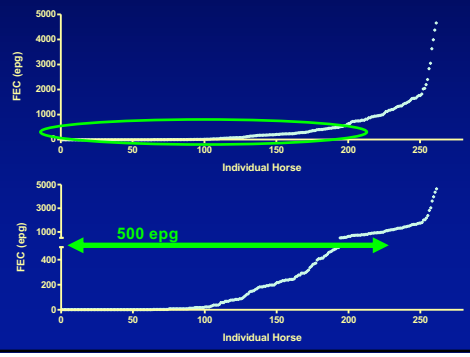
Targeted Selective Treatment Approach

- Treat only those animals that require treatment
  - A new idea that has been around for a long time
- Parasites are highly overdispersed
  - Parasites follow the 80 – 20 rule
  - 20 - 30 % of animals harbor most of the worms

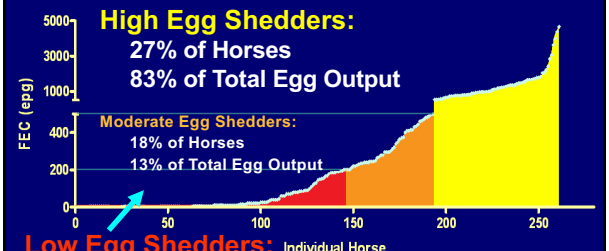
Targeted Selective Treatment Approach

- Let's look at some real data from horse farms
- Perform simulations to illustrate how to implement evidence-based worm control

Distribution of FEC on 12 Horse Farms in Georgia, USA

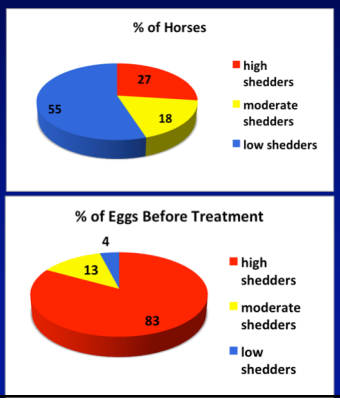


Distribution of FEC on 12 Horse Farms in Georgia, USA



Low Egg Shedders:
55% of Horses
4% of Total Egg Output

Distribution of Egg Shedding in Horses



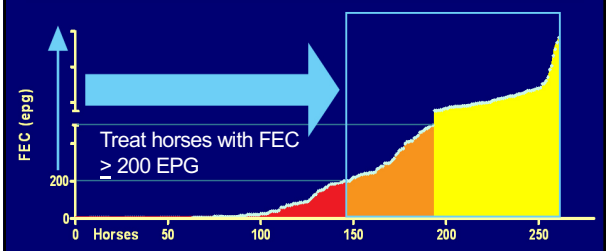
EPG Levels

LOW: <200

MOD: 200-500

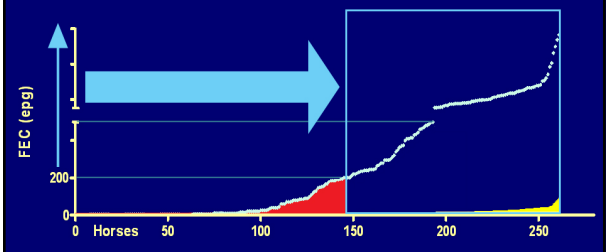
HIGH: >500

What Happens if We Apply Selective Treatment ???



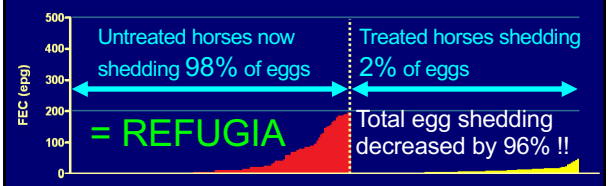
Assume Treatment Reduces FEC by 99.9% (ivermectin or moxidectin)

What Happens if We Apply Selective Treatment ? ? ?



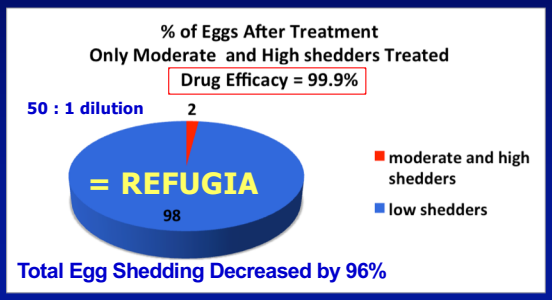
Change in Distribution Following Targeted Selective Treatment

With Ivermectin or Moxidectin



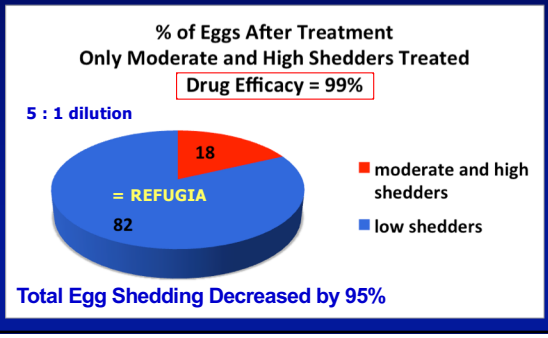
Only horses with FEC ≥ 200 EPG were treated with a drug that has 99.9% efficacy

Change in Distribution of Egg Shedding Leaving Low Shedders Untreated



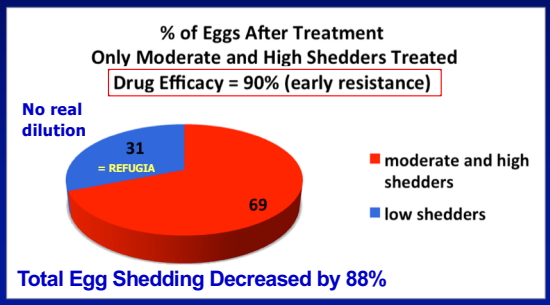
Total Egg Shedding Decreased by 96%

Change in Distribution of Egg Shedding Leaving Low Shedders Untreated



Total Egg Shedding Decreased by 95%

Change in Distribution of Egg Shedding Leaving Low Shedders Untreated



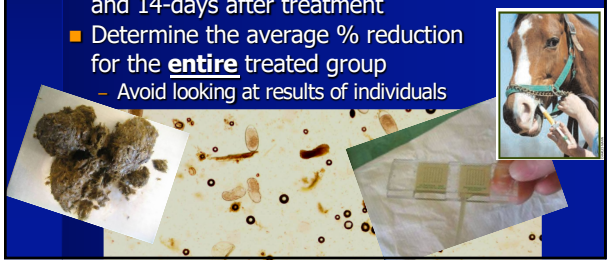
Total Egg Shedding Decreased by 88%

Change in Distribution Following Targeted Selective Treatment

Only horses with FEC ≥ 200 EPG were treated with a drug that has 90% efficacy

Diagnosis of Resistance
Fecal Egg Count Reduction Test

- Only test that we have for resistance
- Compare the FEC of each horse both before and 14-days after treatment
- Determine the average % reduction for the **entire** treated group
  - Avoid looking at results of individuals



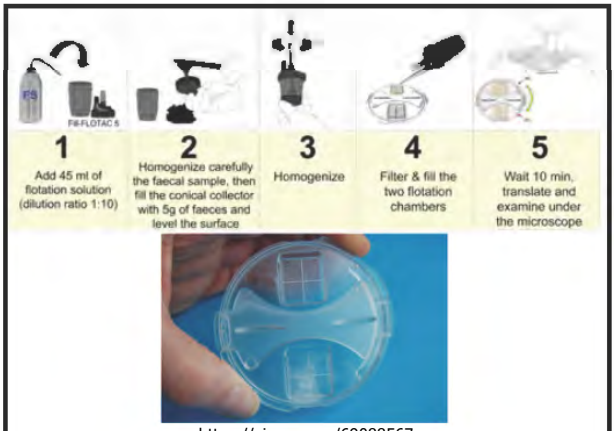
Methods for Measuring FEC

- **McMaster (https://youtu.be/HVW62c9C-vU)**
  - Egg detection sensitivity usually 25 or 50 epg
  - Quickest and easiest method
  - **Best method for routine FEC surveillance**
  - High sensitivity 3-chamber has 8 epg detection – takes longer
- **Modified Wisconsin**
  - Egg detection sensitivity can be varied from 1EPG to 10 EPG
  - Most time consuming method
  - Least accurate and least precise method
- **Mini-FLOTAC (https://vimeo.com/69088567)**
  - Egg detection sens = 5 EPG (or 10 depending on protocol used)
  - **Best method for FECRT** – highest accuracy and precision

Mini-FLOTAC

- Fecal Egg Counting Device
- Developed in Italy in 2012 by Dr. Cringoli
  - Univ of Naples, Italy
- Benefits:
  - 5 (or 10) EPG sensitivity
  - Self-contained system
- Available through Kaplan lab at UGA
  - Email to famacha@uga.edu

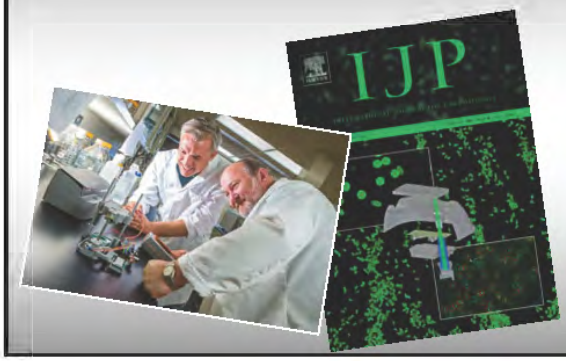
https://figshare.com/articles/_The_fill_FLOTAC_and_the_mini_FLOTAC_kit_/762104



https://vimeo.com/69088567

Poop2Proof Automated fecal egg counts

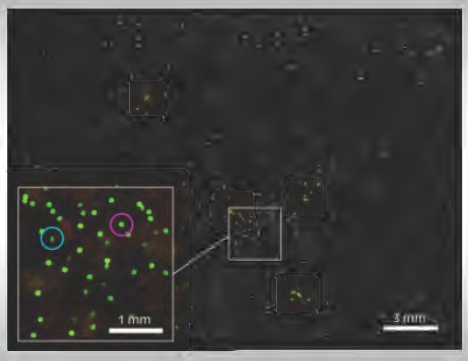
MEP EQUINE SOLUTIONS



The Technology

Sample Preparation | Sample Staining | Imaging

Sample Prep Tool (SPT) | Reagent Dispensing Units (RDUs) | Imaging Unit (IU)



https://www.youtube.com/watch?v=gncXzCbwcrQ

"A WORST CASE SCENARIO THAT ISN'T"

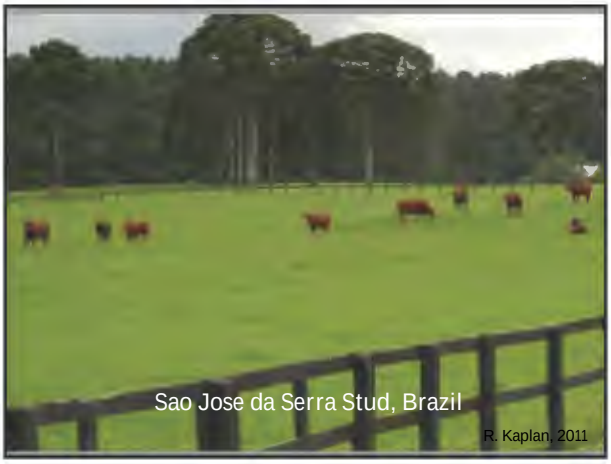
A Case Study on how to deal with multiple-drug resistance and maintain the health and productivity of a horse herd

A HORSE FARM…

With the Worst Problem of Anthelmintic Resistance in the World!

But They Have Healthy Horses And Continue To Win Ribbons And Trophies !!!

- What Are They Doing To Overcome This Problem
  - EPG count surveillance at 50-day interval
  - Selective treatment, > 500 EPG (or more)
  - Low stocking densities
  - Excellent quality pastures
  - Grazing/mixing cattle and horses



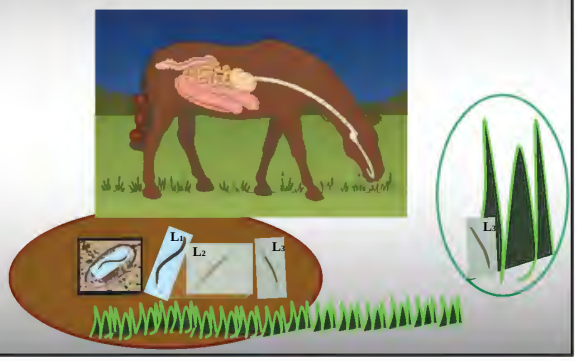
What About Prevention ??? Are There Times When All Horses Should Be Treated ???

- If 100% of worms were cyathostomins then Targeted Selective Treatment based solely on FEC would be fine
- But this is never the case
- Therefore, factors other than FEC must enter into treatment decisions

Factors To Consider in Addition to FEC

- If Tx become too infrequent:
  - Risk that parasites that have become less prevalent as a result of decades of intensive anthelmintic Tx will re-emerge
    - Particularly high risk for *S. vulgaris*
    - Monitoring for this will be essential
- Tapeworms can be difficult to Dx by fecal exam
- No Dx test for bots

Cyathostome life cycle



Climatic Factors Relevant To Strongyle Transmission

- Warm weather factors
  - Optimum temperature for development of eggs and larvae is 25 – 33 ºC (77 – 91 ºF)
    - But survival is short at these temps (few weeks)
    - The upper limit for development of eggs is 38 ºC (100 ºF)
    - at 40 ºC eggs die quickly

Climatic Factors Relevant To Strongyle Transmission

Hot Weather Factors

- Development and survival of larvae on pasture is poor in hot weather
  - In warm/hot climates it is TOO HOT for larvae to develop and persist on pasture from mid-May – mid-September (1/3 of year)
    - Horses on pasture in summer (where summers are hot) do not get infected with clinically important levels of strongyles

Climatic Factors Relevant To Strongyle Transmission

Cold weather factors

- The lower limit for egg hatching is 7.5–10 ºC (45–50 ºF)
- Eggs, L_1 and L_2 are very sensitive to freezing, but L_3 can survive in cold for long periods
  - Under snow cover L3 survival can be high
- But alternation between frost and thaw have deleterious effects on all stages of strongyles

Worm Control is a Yearly Cycle

- Worm control programs are best viewed in the context of a yearly cycle
  - Starts when risk of strongyle transmission changes from negligible to probable
  - Will change from region to region
    - In South begins late summer/early autumn
    - In north (cold winters) begins in spring
- Worm control programs should be tailored to the farm
  - Avoid a "one size fits all" approach

Treat Based on Epidemiological Factors

- Do not give preventive treatments when strongyle eggs are destined to die
  - These also are times of low refugia
- Treat when strongyle transmission is likely
  - Prevent pastures from acquiring high levels of infective larvae
    - This prevents future infections
    - Also ensures there are refugia on pasture
- Treat to kill tapeworms and bots at strategic time of the year

Treat Based on Biological Factors

- Keep the following factors in mind when selecting drugs
  - Time of year relative to expected species fauna and stage
  - Drug efficacy relative to expected species fauna and stage
  - Drug Resistance
  - ERP
  - Egg contamination potential of individual horses

Practical Approach To Targeted Worm Control in Adult Horses

- Have baseline program for all resident horses
  - 1-2 treatments per year
    - At least one of these should be with an ML
  - This is all that 'low' egg shedders will need
    - 50% or more of the adult horses
- Add in additional treatments for the 'moderate' and 'high' egg shedders
  - Timing will depend on drug(s) used and region

What Does This Mean for Kansas ???

- Too cold in winter and too hot in summer for clinically important levels of transmission of strongyles
- Focus anthelmintic treatments in the spring and fall to prevent pasture contamination
  - Feb to May and Aug to Nov
  - Especially early in those seasons
  - Especially in high egg shedders

Additional Suggestions

- Treat for bots and tapeworms in winter
  - **February** would be a good time in KA, as would also get the benefit of strongyle control in the early spring
    - Ivermectin or moxidectin + praziquantel or 2X pyrantel
    - ML drugs will kill any large strongyles present (migrating larvae and/or adults)
    - ML drugs will prevent strongyle egg shedding
    - Using moxidectin will also kill encysted cyathostomins

Don't Forget About Non-Drug Strategies For Parasite Control

- Good pasture management
  - Keep stocking rates appropriate for the pasture
    - grazing horses segregate pastures
      - Lawns and roughs
      - roughs have 15X increase in risk
    - overstocking -- under nutrition
      - pecking order
      - increased parasite transmission to "lowly critters"

Don't Forget About Non-Drug Strategies For Parasite Control

- Reduce number of deworming treatments by:
  - Picking up feces
    - fork and wheel barrel
    - Pasture vacuuming
  - Pasture cleaning alone 2X/week offered the same advantages of deworming with the added bonus of a 50% increase in grazing area
    - RP Herd, VCNA-Equine Pract (1986)

Don't Forget About Non-Drug Strategies For Parasite Control

- Create safe pastures
  - build fences, make hay
- Mixed species grazing
- Alternate species grazing
- Pasture rest and rotation

Harrowing Pastures

- Helps to break up manure and spread across pasture
  - Good for pasture health
  - Good or bad for parasite control depending on timing
- Want to do this in hot/dry periods
  - Late spring or summer
- Leave pasture ungrazed by horses for 4 weeks or more

Recommendations For Foals and Weanlings

- We are rapidly losing our ability to control parasites in foals by using only drugs
  - Picking up feces can help dramatically
- Combination drug treatments are rapidly becoming the only option
  - High chance of resistance in cyathostomins to everything but ivermectin/moxidectin
  - High chance of resistance in *Parascaris* spp. to ivermectin/moxidectin

Recommendations For Foals and Weanlings

- Give first treatment at 2 months of age
  - Repeat treatment at 5 months of age
  - Check fecals regularly
    - Add additional Tx as necessary at 7/8 months
    - If FEC high then probably have resistance problems – need to investigate
- Modeling indicates that reducing number of Tx targeting *Parascaris* spp. to only 2, administered at 2 & 5 mo. of age, is likely to slow the development of resistance
  - Tx at 3 & 4 mo. of age caused resistance to develop much faster

Recommendations For Foals and Weanlings

- Drug combination suggestions:
  - Use 2 or 3 different classes of drugs
  - Examples:
    - Ivermectin + (oxibendazole, fenbendazole or pyrantel)
    - (oxibendazole or fenbendazole) + pyrantel

Take Home Message

- Being infected with parasites is a natural and normal part of being a (grazing) animal
- Modern husbandry practices drive parasite transmission
- Parasite transmission can be greatly reduced by taking a holistic view, that accounts for:
  - the biology of the parasite
  - the host-parasite dynamics
  - and the environment

Take Home Message

- Dewormers are an important and necessary component of parasite control
  - But -- **SHOULD NOT BE THE ONLY** component of parasite control
- Less frequent deworming done in an intelligent manner combined with better management will both:
  - Result in healthier horses
  - Slow resistance and keep drugs working

Final Point

- Properly managed adult horses should never (at least very rarely) have problems with parasitic disease
- Thus, if you have a parasite problem then you have a management problem !!!
  - Must solve the MGT problem

Thank You For Your Attention

UNIVERSITY OF GEORGIA
College of Veterinary Medicine

Bovine and Equine External Parasites

Dr. Brian Herrin - Kansas State University

Insecticide Resistance

- The selection of a specific heritable trait (or traits) in a population of arthropods, due to that population's contact with a chemical, that results in a significant increase in the percentage of the population that will survive a standard dose of that chemical (or a closely related chemical in the case of cross resistance).
- Or Simply
  - As a measurable decrease in the efficacy of a compound against parasite species and stages that were previously susceptible.

Insecticide Resistance

- Individuals with genetic traits that allow them to survive exposure to an insecticide will pass genes on to the subsequent generation, thereby potentially increasing the percentage of a population that can survive subsequent exposure to the chemical.
  - There are three necessary conditions for evolution of resistance to occur:
    - Individuals in the population must differ genetically
    - Genetic differences must produce a phenotypic difference
    - The phenotypic difference must enhance survivability, transferring the resistance to the next generation

Insecticide Tolerance

- In contrast to resistance, tolerance is a natural tendency rather than a result of selection pressure.

- Tolerance is often used to describe natural differences between different species or between life stages of organisms.

–For example ticks are naturally more tolerant of imidacloprid than fleas Natural Susceptibility or Natural Variability

- Refers to differences in response by an organism or population to a toxicant relative to other individuals or populations unrelated to prior exposure or prior selection.
  - Sometimes it is difficult to differentiate true resistance from the natural range of pesticide susceptibility that exists as a bell curve in every population of pests.

Insecticide Resistance

- How does a population go from point A (a certain rate of susceptibility) to point B (a reduced rate of susceptibility).
- Example:
  - Today insecticide X at dose Y kills 98% of the population
  - Then at some later date insecticide X at dose Y kills 80% of the population

Resistance is a Problem of Evolution
often referred to as time compressed evolution

- Insecticide resistance is a change in a population in which susceptible individuals are killed by an effective, frequently, or continually used chemical, leaving the non-susceptible individuals to breed.
- Populations become resistant
  - The genetic component is within the population
  - Due to natural genetic diversity individuals with different phenotypes already occur within a population.
  - Traits that here-to-fore were not beneficial now may be beneficial.

        - Selection increases the percentage of individuals, with a particular trait, within a population.

Factors Affecting the Rate of Resistance Development

- Genetic & Biological Factors
    - Frequency of R alleles
    - Dominance of R alleles
    - Generation turn-over
    - Number of offspring per generation
    - Type of reproduction (sexual or asexual; single or multiple mates)
    - Isolation
    - Migration
    - Natural Refugia
    - Fitness (cost to population of carrying a particular trait)
- Operational
    - Relationship to chemicals used in the past (cross resistance)
    - Persistence of residues
    - Life stages targeted
    - Refugia (% of population left untreated)
        - Selection threshold

<u>Refugia</u>

- Portion of the parasite population that is not exposed to the chemical.
- A reservoir of pesticide-susceptible genes because there is no selection pressure on parasites that are unexposed to the chemical(s).
    - Managing refugia has been used strategically to help delay progression of resistance.
        - Population ("pool" of genetic material) that is susceptible and can mate with resistant population
        - Population ("pool" of genetic material) must be able to immigrate into the treated area

<u>Insecticide Resistance</u>

- If you remember nothing else remember this!
    - Insecticide and acaricide resistance (as well as anthelmintic resistance) only occurs if we are able to put enough SELECTIONAL PRESSURE on the pest population to eliminate most if not all the susceptible individuals leaving behind mostly if not exclusively the non-susceptible individuals.
    - If you can not put selectional pressure on the population (often due to large refugia populations) it is unlikely resistance will develop or develop very slowly.
    - There are exceptions such as certain insect populations with very long life cycles (months to years) and low reproductive capacity that regardless of the selection pressure they do not develop resistance.
        - Example: "Bot Flies"

<u>Practical & Theoretical Issues</u>

- Selection Pressure
    - The higher the selection pressure (the more insects that are killed) the faster the rate of resistance development
- Insecticide Persistence
    - A persistent insecticide enhances resistance development by applying selection

pressure on multiple generations

Practical & Theoretical Issues

- A variety of operational and biological factors commonly buffer (delay) insecticide resistance selection.
  - Inadequate application
  - Inappropriate dosing
  - Infrequent applications
  - Refugia
  - Immigration from refugia (non-selected population)
    - Fitness (cost to population of carrying a particular trait)

Factors Affecting the Rate of Resistance Development

- Short generation time
- Few offspring per generation
- Isolated population
- Immigration from natural refugia
- Highly effective persistent residual insecticide
  - Client applying doses irregularly

Practical & Theoretical Issues

- Often the very thing we are trying to accomplish for pet or livestock health is what forces selection (resistance)!
  - Attempted elimination, prevention and control of the entire pest population
- Whether because of true resistance, natural variability, product attributes or ecologic reasons, perceptions of performance problems do occur.
- In a private practice it is often difficult if not impossible to determine if resistance or some other factor is responsible for control failure.
- In such cases client education, additional control measures or switching to a different product may be necessary.

Common Reasons for Perceived Product Failure

- Lack of understanding of product performance attributes
  - Speed of kill, duration of adulticide activity, repellency, unrealistic efficacy expectations
- Inadequate control measures
  - Not treating all pets, visitor pets, misadministration, no treating regularly, etc.
- Lack of knowledge concerning parasite biology and epidemiology

Integrated Pest Management

- Uses a variety of systems to control pest populations & delay development of resistance.
  - Establishment of injury and treatment thresholds
    - Maintaining refugia
  - Monitoring of pest populations
  - Chemical control (rotations?, combinations?, mosaics)
    - Insecticides
    - IGRs
  - Biological control
  - Mechanical control
  - Resistance monitoring

- Ruminant Ectoparasites
    - Lice
    - Flies
    - Ticks
    - Mites
- Lice - Pediculosis - Livestock
    - Cattle
    - Sucking Lice:
        - Haematopinus eurysternus - "Short nosed cattle louse"
        - Linognathus vituli - "Long-nosed cattle louse"
        - Solenopotes capillatus - "Little blue cattle louse”
        - Haematopinus quadripertusus - "cattle tail louse"
    - Biting Lice:
        - Damalinia (Bovicola) bovis - "Biting cattle louse"

    - Sheep
    - Sucking Lice:
        - H. tuberculatus - "Buffalo louse"
        - Linognathus ovillus - "Sucking face louse"
        - L. pedalis -"Sucking foot louse”
    - Biting Lice:
        - Damalinia ovis -"Biting louse of sheep"
    - Goats
    - Sucking Lice:
        - Linognathus stenopsis - "Blue Sucking Louse"
    - Biting Lice:
        - Damalinia caprae - "Red biting louse"
    - Life Cycle
        - Lice are very host specific
        - Entire life cycle on host
        - Adult--> Egg --> Nymph --> Adult (3-4 weeks)
        - Transmission by direct contact (carriers)
    - Predisposing Causes
        - Neglect
        - Poor nutrition, poor condition, poor grooming, overcrowding, filth, cold, debilitation
        - Seasonal; worse in winter
        - Usually more a problem in young animals
    - Pathogenesis
        - Damage skin and wool (pruritus - rubbing, etc.)
        - Anemia
    - Clinical signs
        - From asymptomatic carriers to death due to anemia
        - Alopecia and crusts

        - Heavy louse infestations can result in:
            - lowered milk production, loss of weight, stunted growth, wool damage, anemia, and very rarely secondary infections & death.
    - Diagnosis
        - Physical exam; find and identify lice and eggs
    - Treatment, Management and Control
        - Correct underlying causes (Crowding, debilitation, nutrition, etc.)
        - Cattle
            - Ivermectin;
                - Injection - 200µg/kg eliminates "sucking lice" Sub-Q
                - Topical - (500µg/kg) - Biting & sucking lice
            - Doramectin;
                - Injectable - 200µg/kg sub-Q or I.M. (sucking lice) one or two injections 3 weeks apart.
                - Topical - 500µg/kg - Biting & Sucking lice
            - Moxidectin;
                - Topical - 500µg/kg – Biting & Sucking lice
            - Eprinomectin;
                - Topical - 500µg/kg – Biting & Sucking lice
            - Viable lice can be found for 1 week after treatment with avermectins - do not mix cattle for 1 week.
            - Label - Single doses of avermectins are generally 100%, however - may be best to give two treatments 3 - 4 weeks apart.
            - Insecticide sprays, pour-ons and dusts
                - Permethrin, Coumaphos, Cyfluthrin, Phosmet, Fenthion
                - Generally 2 applications at 2 - 3 weeks intervals; read and follow labels
        - Sheep
            - Insecticide sprays, pour-ons and dusts are used.
                - Coumaphos, diazinon, fenvalerate, permethrin, methoxychlor
                - Generally 2 applications at 2 - 3 weeks intervals; read and follow labels
            - Macrocyclic lactones not approved for louse control in sheep
                - (Ivomec Drench for sheep is not labeled for lice)
- “Big 4 flies”
    - House fly
    - Stable fly
    - Horn fly
    - Face fly
- Housefly
    - Musca domestica
    - Non-parasitic fly – but of significant veterinary & economic importance
    - Lapping or Sponging mouth parts (Labellum); "do not bite”
        - feed by imbibing fluids or regurgitating on feedstuffs then imbibing liquid.

- Size: 6-9 mm long
    - Black and yellow w/ four dark
    - stripes lengthwise on thoracic
    - dorsal surface
    - Eyes separated
- Lifecycle - Musca domestica
    - Oviposit (Eggs laid) in & Feed on fresh or old manure, garbage, sewage, food waste, lawn clippings, compost piles.
    - Moist (>90%) decaying organic matter
    - Overwinter as larvae (maggots) or pupa
    - Life cycle can be completed in 7 - 10 days.
    - House flies will accumulate in large numbers around confined livestock operations.
    - Due to the large number of oviposition sites (organic waste).
    - Resting sites - fences, buildings, trees, and shrubs; often in the sun.
- The major development sites in a confinement livestock operations
    - behind feeding aprons
    - under fences and gates
    - bedding in sick pens
    - along drainage areas and debris basins
    - diary calf hutches
    - old hay stacks
    - in silage and haylage drainage areas
    - along and under feed bunks
    - around leaky waterers.
- V. Veterinary/Medical Importance
    - Dispersal of flies into surrounding urban environments constitutes a major public annoyance.
    - Producers may suffer legal action (fines &/or being forced to cease operation) when failing to adequately control house fly development in organic waste produced by their livestock operations.
    - Transmits diseases - on body/also regurgitate when they feed
        - including Vibrio cholerae, Bacillus anthracis, E. coli (O157:H7), Staph spp, Salmonella spp, Mycobacterium tuberculosis, etc…
    - Transport eggs of parasitic worms
        - Ascaris lumbricoides, Trichuris trichiura, T. solium, Giardia sp.,
        - T. pisiformis, A. caninum, D caninum, Toxocara etc….
    - Intermediate host of:
        - Habronema muscae & Draschia megastoma
- Control - Musca domestica
    - Sanitation – The “KEY” to fly control
    - House fly control in confinement operations may be fruitless without proper sanitation (manure & waste management).

- Scrape pens, alleyways & fence lines to remove manure. Clean up spilled feed, decaying hay & silage around feed bunks, under fences, gates, & around waterers.
- Remove manure; spread thinly on fields or mound up in pens & allow to dry.
- Construct lots with good drainage.
- Physical - screen windows & doors in dairies
- Residual surface sprays - (only helps for a short time)
- Apply to resting sites such as ceilings, panels and walls.
    - (Fenthion, Diazinon, Permethrin, Cyfluthrin, Spinosad etc..)
- Baits
    - Insecticide impregnated material that kills flies when they land and feed on bait. Baits attract using pheromones and sugars.
    - House fly pheromone attractant (Muscalure & Muscamone) Z-9-Tricosene (Musca domestica sex & aggregation pheromone) to attract flies.
- Bait systems: examples
    - Golden Malrin® Fly Bait granular scatterbait. (Z)-9-Tricosene fly attractant. Then, as they feed on the sugar-based bait the ingredient methomyl kills flies.
    - QuickBayt® Bayer (Z-9-Tricosene and sugar + Imidacloprid) – flies die within 1 –2 minutes. – other trade names
    - QuikStrike® Fly Scatter Bait (Dinotefuran & (Z)-9-Tricosene)
    - Baits can be applied as a dry bait, sprays or mixed with water to make a spray or paste that can be applied to vertical surfaces.
- Traps –
    - Ultraviolet light traps
        - Attract and collect the flies inside an inverted cone or kill them with an electrocuting grid.
        - Attractant (sugar and pheromone) based traps
- Biological control – very tiny parasitoid wasps – larvae eat fly pupae
    - Tiny wasps (2 – 4 mm)- obligate parasites of flies.
    - Wasp seeks out fly pupae, inserts ovipositor through puparium and lays egg on pupae.
    - Wasp larva eats fly pupae and develops inside fly puparium and emerges as wasp in 3 weeks.
    - Wasp lays 20 – 45 eggs. One egg laid/fly pupae.
    - Periodic (weekly to every other wk) release of parasitoids throughout fly season is necessary to impact fly populations.
    - Wasp pupae are sprinkled onto manure or soiled feedstuff
    - Must use correct wasp species.
        - Certain parasitoid wasps only parasitize certain species of flies and will survive best in specific environments.
        - Muscidifurax zaraptor has been found to be the species that works best in Kansas Feedlots for house fly control.

- Stable fly
    - Stomoxys calcitrans
        - Looks like housefly except it has a slender, rigid, piercing - sucking proboscis projecting forward beneath head
        - Female and males feed on blood;
        - only on host when feeding
        - A fiercely biting, blood sucking fly
        - Cluster around feet and legs of cattle & horses
        - Primarily a problem in confined livestock operations
        - But has become more of a problem in pastured livestock.
    - Life History – Stomoxys calcitrans
        - Oviposit in moist decaying vegetable matter
        - Accumulated old manure (at least 2 – 3 weeks old), spilled feed, compost & grass piles, wet silage, bottoms of large round bales, decaying vegetable material, old hay (has to be moist) – material that is aged and fermenting
            - Similar to House Fly; but typically much more decay
        - Eggs to adult in about 33-36 days
        - Populations highest late May through June.
            - Can be a recurrence in the fall
        - Overwinter as larva, pupa, or adult
        - Can disperse long distances, up to 160 miles.
        - Resting sites: shaded sides of fence posts, wooden fences, feedbunks, on buildings, or lower parts of tress, shrubs or weeds - usually low to ground.
        - Over past 3 decades Stable flies have also become more of a problem in range land cattle wherever large round bales have been used to feed cattle in fall & winter.
        - Uneaten hay mixed with manure decomposes over winter and is an attractive oviposition site and excellent development site for larvae in the spring.
        - Also becoming a problem in suburban areas because flies will oviposit in grass compost piles.
    - Pathogenesis and Veterinary Importance:
        - Primarily Injury or losses due to:
            - Inflict painful bite - foot stamping, tail switching, animals reduce feed intake, lose weight and decrease milk production.
        - As few as 3 – 5 flies/leg has measurable economic loss.
        - Economic losses for feeder cattle, even at low fly population levels, are dramatic.
            - 0.1lb to 0.5 lb/head/day decreased weight gain.
        - House & stable flies are considered together in nuisance lawsuits.
            - This litigation usually cites dust, odors, and flies as a complex without identifying fly species.
            - If lawsuits are settled in favor of the claimant, the settlement requires either punitive damages or cessation of livestock production.
    - Control

- Sanitation - manure removed or mounded
    - Clean around feed bunks, feed aprons, under fences, gates, & around waterers.
    - Remove manure and spread thinly on fields or mound up in pens & allow to dry.
    - Construct lots with good drainage.
    - Note: In most instances, if sanitation is poor, chemical control may be useless.
    - Proper sanitation denies flies effective oviposition and develop sites for both stable and house flies.
- Traps - translucent or semi-translucent plastics
    - Reflect sunlight in polarized wavelengths that is attractive to stable flies.
        - Alsynite and Polyethylene terephthalate
    - Adhesive coated &/or insecticide treated – very effective
    - Several commercially available
    - Wavelength of light transmitted through these traps is specifically attractive to S. calcitrans.
- Chemical control – typically pyrethroids
    - Premise Surface sprays
    - Resting surfaces can be treated with residual sprays.
    - Area sprays
        - Mists or foggers
    - Animal sprays
        - Cover legs and underside of body where flies commonly attack. Repeat as needed; limited effectiveness because flies only on the host long enough to feed.
- Biological control - parasitoid wasps - destroy fly pupae - Gnat sized parasitic wasps that oviposit on fly pupae. Developing wasp larva eats fly pupae.
    - Species specific must use correct wasp species.
        - Spalangia nigroaenea
    - Best used May – June
    - Typically wasp pupae are deposited every 1 to 2 weeks to fly oviposition sites
    - Some companies sell up to 4 species of parasitoids in a pack
- *fly baits* have no attractiveness for Stable flies they are only effective against House flies. Contain sugar or aggregation attractants.
- Managing Stable Fly Production at Pasture Feeding Sites. http://www.ksre.ksu.edu/bookstore/pubs/mf2662.pdf
    - Continual movement of feeder location between feedings.
    - Rolling hay out in different locations throughout the pasture.
    - Avoid rolling out poor quality or rotted hay that will not be eaten.
    - Grinding hay helps decrease waste.

- Avoid overfeeding to prevent trampling of hay, which becomes habitat once mixed with manure.
- Feeding locations should have adequate drainage to keep moisture from accumulating around the feeder.
- Proper cleanup and removal of residue is necessary. Because the majority of fly production occurs in May and early June, the site must be cleaned and waste disposed of before April 15.

- Tabanids: Horse & Deer Flies
  - In U.S. two major genera:
    - Tabanus sp.,"horse fly"
    - Chrysops sp., “deer fly”
    - 1 minor - Hybomitra sp.,
  - General characteristics: Tabanids
    - Large and heavy bodied
      - Horse fly ~ 25 mm,
      - Deer fly ~ 6-10 mm
    - Strong fliers
    - Females - blood feeders
    - Males non-biters, both males and females feed on nectar
    - Larvae aquatic and predaceous;
      - Eggs oviposited in wet habitats, usually vegetation over water. Most species live along edges of ponds, lakes or streams.
  - Control
    - No satisfactory chemical control for horse and deer flies.
    - Wetland habitats that support development make it impractical and environmentally unacceptable to treat breeding sites.
    - Adults do not constantly rest on any surface, so residual insecticide treatments are not effective.
    - Daily treatment of animals with Pyrethrin (1%) or weekly treatment with synthetic pyrethroid (permethrin or cypermethrin). Repeat as needed. Control is considered poor.
    - Box, pyramid, umbrella or canopy traps – based on horse & deer fly attraction to large dark objects.
      - http://www.youtube.com/watch?v=PcTaUZBQees
    - The EPPS fly trap
      - Horse-like components are large black sheets of polypropylene tarp with open areas underneath, much like the side of a horse might appear to a horse fly.
      - Flies enter and hit clear panels and drop into soapy water.
      - https://www.youtube.com/watch?v=nIlD9h8ebyg
- Face fly
  - Musca autumnalis
  - Characteristics:
    - Looks like a housefly only little larger

        - Adults (females mostly) feed on cattle secretions: tears, saliva, nasal discharge, blood (from wounds/insects) & serum.
        - Females need protein for egg development.
        - Both female and male feed on nectar.
        - Puparium is dirty white
            - contrasted to reddish-brown in house fly, stable fly & horn fly.
    - Life cycle
        - Females lay eggs in fresh dung (5 hr to 24hr) post defecation
            - Until crust forms on fecal pad
        - Egg to adult ~12-21 days
        - Flies Overwinter in barns/homes
        - Adults emerge late April mate and disperse to pastures.
        - Found on nose, around eyes (face), and backs of cattle.
            - Flies only spend 5 - 10 minutes per day on an animal then leave to rest on shaded vegetation
            - Less than 5% of the face fly population is actually on the cattle at any one time.
        - Adult face flies are strong fliers capable of traveling several miles.
        - Face flies are also called attic flies (overwinter in the attics of houses)
        - During the fall (decreasing daylight hours) the ovaries stop developing and the flies accumulate lipid.
        - Flies stop feeding & "hibernate' or over-winter in reproductive diapause in houses.
            - Several thousand may be found in some attics.
        - In warm climates non-feeding flies remain active and utilize lipid reserves and die.
            - In the spring photoperiod changes induce ovarian development.
        - Adults emerge late April -- mate and disperse to pastures.
    - Economic and Veterinary Importance:
        - Weight reduction
        - Decrease milk production
        - Biological vector of - Thelazia sp.
        - Mechanical vector of -
            - Moraxella bovis - pink eye
            - IBR
        - Mechanical damage to conjunctiva (possess pre-stomal teeth)
    - Control
        - Sanitation - Remove dung? - not likely
        - highly impractical/impossible in a pasture
        - Ear tags impregnated with pyrethroids or organophosphates. Control up to 5 months
            - 2 tags/animal
        - Organophosphate: diazinon, diazinon + chlorpyriphos, fenthion, coumaphos + diazinon, etc
        - Pyrethroids: permethrin, cypermethrin, cyfluthrin, fenvalerate
        - Avermectins (abamectin)

        - The pyrethroid insecticide ear tags provide better face fly control than do those containing phosphate insecticides.
        - Insecticide sprays to animals face.
            - pen up cattle to apply spray during summer, often difficult to "sell" to rancher
        - Backrubbers - containing an insecticide mixed in oil
        - Dust bags - containing insecticides.
            - Oilers & bags - suspended near (not over) mineral or salting stations; water source, alley ways, or holding pens
        - Oral larvicides - insecticides or insect growth regulators in mineral mixes, feed mixes or bolus
            - Act by killing developing larvae in manure.
            - Diflubenzuron (chitin inhibitor - IGR): oral bolus (5 months)
            - Methoprene (juvenile hormone analog -IGR): premix, mineral blocks, granules
            - Rabon (tetrachlorvinphos) - premix
        - Effective as a part of an overall program, but not effective by itself due to fly immigration
- I. Horn Fly
    - Haematobia irritans
    - Characteristics
        - 1/2 size of a house fly (~ 4 mm long)
        - Resembles stable fly but more slender, labium is heavier, and palpi almost as long as the proboscis
    - Life cycle
        - Females oviposit in fresh manure (< 3 min. post-defecation)
        - older manure not attractive
        - Entire life cycle approx. 10 - 14 days;
        - In Kansas 7 - 10 generations per year
        - Overwinter as pupae & emerge at end of April
        - Adult flies spend virtually all their adult life on backs of cattle.
        - Cattle are feeding and resting site.
            - When cattle try to dislodge the flies, the flies move immediately to another animal in the herd.
        - Horn flies have a high rate of dispersal and movement.
    - Pathogenesis
        - The primary pest of range cattle
        - Irritation, annoyance & blood loss - flies inflict a painful bite:
        - Weight loss
        - Reduced milk production
        - Loss of blood
        - Irritation and blood loss can result in reduced weight gain of up to 0.5 lb./day. As few as 50 - 200 horn flies per animal can result in measurable weight loss.
    - Control
        - Effective control can yield an extra 20 to 30 lbs/head on grass during the summer grazing season.
        - Ear Tags (up to 5 months activity)

                - Pyrethroid, Organophosphate, Abamectin or Combination
                - Initially pyrethroid tags highly effective (100% control)
                - Resistance first reported in Kansas in 1982.
                - Recommendations to combat resistance to ear tags:
                    - Do not tag before fly season starts
                    - Use tags in conjunction with other control measures
                    - Rotate tags 1 year Pyrethroid – 2 years Organophosphate (OP)
            - Topical residual insecticides
                - Treat late May when fly counts reach 50 flies/animal
                - Pour-ons (cyfluthrin, permethrin, fenthion)
                - Sprays (Coumaphos, phosmet, permethrin, spinosad, etc...)
            - Backrubbers (insecticide containing)
            - Dust bags - (insecticide containing) provide excellent control of horn flies.
                - Ready to use bags are available.
                - Suspend near (not over) mineral or salting stations, water, in alley ways, in loafing pens, or in holding sheds.
            - Larvicides – effective as apart of an overall program, but not effective by itself due to fly immigration
            - Topical Avermectins
                - Short duration of action typically 7 days, may get some benefit for up to 28 days
                - Pour-on formulations of
                    - ivermectin (28 days)
                    - eprinomectin (7days)
                    - moxidectin (7 days)
                    - doramectin (7 days)
- Melophagus ovinus
    - "Sheep Ked", "Sheep tick"
    - Wingless fly: ectoparasite of sheep and goats
        - Blood sucker
        - Reddish-brown in color
        - Size (5-7 mm long)
        - Short head
        - Body leathery and spiny
        - 3 pairs of legs
    - Life Cycle
        - Entire cycle on host
        - Larvae develop within female (7 days)
        - Pupate soon after extruded from female
        - Pupation (2 - 5 weeks)
        - Female oviposits larvae one at a time
        - Average life cycle 4 months
        - Female and male suck blood
        - Have sharp mouthparts they thrust into skin
    - Control:
        - Various insecticide sprays, dips, and dust - treat in spring after shearing
        - If must treat in fall/spring select a warm day & keep outside till dry.

- I. Cattle grubs, Ox warbles,
  - (adults - heel flies)
  - Hypoderma lineatum - Heel Fly, common cattle grub
    - wide dist. (Southern U.S.)
  - Hypoderma bovis - Northern cattle grub
    - Northern regions
  - Description
    - Adult flies
      - Similar to honeybees in size & color (yellow and black)
      - 13-15 mm long
      - No biting mouthparts & can not “sting”
    - Larvae
      - Mature larva 25-28 mm long
  - Life cycle
    - Adult flies active on warm days
    - Flies emerge late spring early summer
    - Flies only live a few days (up to 1wk)
    - Females attach eggs to lower legs (heel fly) of cattle.
    - Eggs hatch in 2 to 6 days.
    - Larvae burrow through skin & migrate through connective tissues for 2 – 4 months
    - Larvae spend 5 - 7th month in tissues around spinal cord or esophagus (November to December)
      - H. bovis - epidermal tissues of spinal canal
      - H. lineatum - submucosa of esophagus
    - Larvae then move to back, cut a breathing hole and remain for 30 to 90 days.
    - Larvae emerge, fall to the ground and pupate 1 - 3 months with adults emerging April - May.
  - Signs
    - Adult flies
      - Frighten cattle - "Gad" about (Gadfly)
        - "to wander from place to place without a particular destination".
    - Injury while escaping to shade or water
    - Decreased milk production
    - Cattle standing in water can be from heat, Hypoderma, Stomoxys sp. Etc.
  - Clinical Signs due to Larvae
    - “Grubby backs”
    - Cysts, painful, soft, fluctuant, "Air holes”
    - Occasional anaphylaxis
    - CNS and esophageal damage (stagger, bloat, ataxia, especially if crushed)
  - Pathogenesis
    - Migrate in esophagus and spinal cord
    - Inflammatory reaction if die in situ or crushed (accidentally or on purpose)
  - Diagnosis
    - Observe eggs on hair

        - History - physical exam (grubs in back)
    - Treatment
        - Application of systemic insecticides during the period following heel-fly activity and before grubs reach esophagus (bloat) and spinal canal (posterior paralysis).
        - Kansas treat from June until October
        - Insecticides in spray and pour-on formulations: Coumaphos, Fenthion, and Famphur, etc…
        - Injectable Ivermectin & Doramectin
            - Usually administered at 200µg/kg for internal nematodes and ectoparasites, but doses as low as 50µg/kg are 100% effective against migrating grubs (Hypoderma sp. larvae)
        - Topical spot-ons
            - Eprinomectin, Moxidectin, Ivermectin & Doramectin 500µg/kg single dose
- Tick species of interest - Livestock
    - Amblyomma americanum - Lone Star Tick
    - Amblyomma maculatum - Gulf Coast Tick
    - Dermacentor variabilis - American Dog Tick
    - Dermacentor albipictus – Winter Tick
    - Otobius megnini - Spinose Ear Tick
    - Rhipicephalus annulatus (formerly Boophilus annulatus) "Texas Cattle Fever Tick"
    - Rhipicephalus microplus (formerly Boophilus microplus) “Southern Cattle Tick”
- Dermacentor albipictus
    - D. albipictus (var. nigrolineatus)
    - Winter tick or “Ghost Moose Tick”
    - Thousands of these ticks may infest deer, moose, cattle and horses in the fall and winter, resulting in severe anemia, alopecia and death due to exposure in winter.
        - A study from western Canada estimated an average of 33,000 ticks per moose with some individuals having over 100,000. http://www.youtube.com/watch?v=Rsd2i-qFHK4
    - 1 host tick;
    - Inornate or ornate
        - Inornate strains: only inornate forms have been found in Kansas
        - “Nigrolineatus" was originally named and described as a distinct species because of the inornate scutum and black or dark "lines" visible in dorsal view (caused by the diverticulae showing through the transparent dorsal wall usually present in this form).
        - It is unknown why there is an apparent transparency of the chitin.
    - Larvae are active late August – early October
        - Larvae that don’t find a host, do not survive the winter.
    - Larvae feed for a few weeks & molt on host to nymph
    - Nymphs feed for a few weeks or months & molt on host to adult.
    - In Kansas we find adults on horses as early as late October to mid November
        - In northern regions nymphs do not molt to adults until November or as late as January.
    - Adults drop off in winter or early spring to lay eggs.
-

- Amblyomma maculatum - "Gulf Coast Tick"
    - Historically Gulf Coast to Carolinas
        - Recent expansion into Kansas
    - Cattle; wide range of hosts. Larvae and nymphs on foxes, smaller mammals and birds.
    - Prefer to attach to the ears
    - 3-host tick
    - Larvae and nymphs
        - Many small mammals - most abundant on birds, (quail, meadowlarks and sparrows).
        - In addition, juvenile forms feed on migratory birds such as cattle egrets.
    - Adults feed on cattle, horses, deer, sheep, coyotes, and dogs.
        - In Kansas adults found in the ears of cattle in April - June.
    - Feeding is accompanied by bacterial infection.
        - Combination of immune response to the ticks' saliva and the infection causes characteristic thickening of the ear and the bending of the ear into a permanent deformity known as "gotch ear".
        - Deformation of ear cartilage, excoriation with serum and blood exudate, crusting, & alopecia
    - The literature shows that growth performance of young cattle infested with Gulf Coast ticks can be reduced by as much as 20%.
- Rhipicephalus spp.
    - Rhipicephalus annulatus - (formerly Boophilus annulatus) "Texas Cattle Fever Tick"
    - Rhipicephalus microplus - (formerly B. microplus) "Southern Cattle Tick".
    - 1-host inornate ticks
    - Officially eradicated from U.S. in 1943
        - All cattle (>1million/yr) transported into the U.S. from Mexico are inspected.
        - However ticks periodically reintroduced from cattle & wildlife crossing the border from Mexico
        - Quarantine zones are frequently in effect in counties in southern Texas due to these infestations.
    - Tick transmit Texas Cattle Fever (Babesia bigemina and Babesia bovis), Anaplasmosis
    - National Cattle Fever Tick Eradication Program
        - Today, surveillance efforts rely upon trained inspectors who evaluate livestock such as cattle and horses (both resident or imported, as well as stray or smuggled animals from Mexico) by examining suspected animals in their entirety by hand to feel for ticks, a process known as 'scratching'.
        - All cattle within the Texas permanent quarantine zone (PQZ) are scratch-inspected and vaccinated against ticks on a yearly basis.
        - Prior to removal from the quarantine zone, cattle must be scratch-inspected to certify that they are fever-tick free and are whole-body dipped in a bath of the organophosphate pesticide, coumaphos.
        - Regulations are even stricter for imported cattle. In addition to the required coumaphos bath, if scratch inspection reveals the presence of any tick of any species, the entire shipment of cattle is denied entry to the US.
        - Nilgai and deer are often able to move unimpeded between pastures and fields due to their ability to cross fencing and natural barriers such as rivers.
            - Analysis of hides submitted during the 2017- 2018 Texas hunting season showed a high level of tick infestation, among both native and exotic

game animal populations. Approximately 34% of native deer hides were infested with Cattle Fever ticks, compared to 46% of Nilgai hides submitted for inspection.

- Treating free-ranging wildlife or exotic animal hosts for fever ticks poses a particular challenge.
  - Treatment is limited to feeding ivermectin treated corn or the use of four-poster feeders with permethrin infused rubbing posts. Ivermectin treated corn has been approved to feed to white-tailed deer by the FDA and can only be done legally by USDA and TAHC personnel.
  - All ivermectin treated corn must be withdrawn no later than 60 days before the start of hunting season. White-tailed deer or exotics maintained in pens can be treated like cattle.

- Pathogenesis
  - Blood feeders
  - Tick paralysis
  - Transmit diseases
  - Cause irritation
  - Predispose to screw worm flies

- Clinical Signs
  - Anemia
  - Affected animals appear dull; off feed
  - Loss of condition
  - Decreased milk production
  - Tick paralysis
- Diagnosis
  - Physical exam
  - Remove and identify ticks
- Control of Ticks on Cattle & Horses
  - During the early life stages most tick species are dependent on rodent hosts.
  - Reducing numbers of mice, wood rats, gophers and rabbits may reduce tick populations.
  - Where possible eliminate shrubs and other woody vegetation.
    - Provides habitat for both rodents and ticks and tick climbing sites.
  - Pasture burning
    - Results have been variable. Regular prescribed burning has been shown to reduce D. variabilis and A. americanum, but not A maculatum.
  - There are no acaricides registered for treating grazing land.
  - Killing of ticks on cattle & horses can be accomplished with repeated whole body treatments at 3 to 4 week intervals
    - Permethrin (various formulations such as Ectiban, Atraban, Permectrin, Expar etc.)
    - Coumaphos: (Co-Ral: Bayer)
    - Amitraz (Taktic) not registered for use on horses – currently not available
      - Always read and follow label directions
  - Gulf Coast or Spinose Ear ticks
    - spray ears directly!!!
    - acaracide impregnated ear tags (one tag per ear)

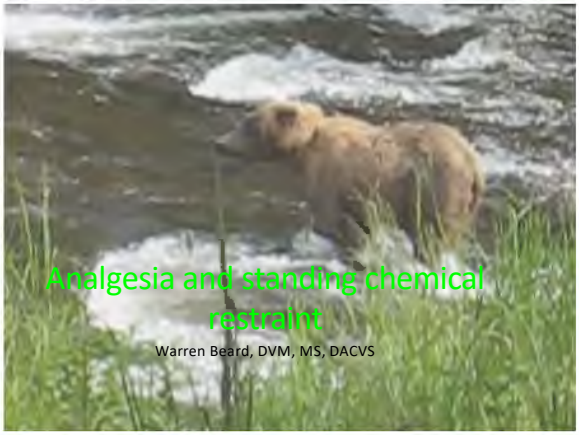
- Scabies - Livestock
    - Five genera of mites in cattle
        - Psoroptes sp.; Sarcoptes sp.; Chorioptes sp.; Psorergates sp.; Demodex sp
        - Three of these species are classified as scabies mites.
        - They are Sarcoptes scabiei, Psoroptes communis bovis and Chorioptes bovis.
    - Upon detection cases of scabies in cattle, sheep or goats should be reported to the State Veterinarian (Kansas).
        - The most important and legally reportable in all states is Psoroptes communis bovis.
        - Note many state regulations say "cattle scabies" is legally reportable without differentiating between mite species.
- Psoroptic Scabies, Psoroptic Mange
    - Psoroptes communis bovis; common Cattle Scab (rare)*
    - P. communis ovis*; common Sheep Scab)* Sheep, (eradicated in U.S.)
    - P. communis caprae; common Goat Scab
    - *Legally reportable in all states
    - Life Cycle
        - Mites on sheep and cattle are morphologically the same but they do not spread from one host species to another.
        - The life cycle is completed entirely on the host.
        - The life cycle is usually about 12 - 14 days.
        - Mites can survive up to two weeks off the host.
        - Transmission is generally by direct contact
            - but mites may be spread by contact with infected trucks or pens.
    - Pathology & Clinical Signs - Psoroptes (cattle)
        - Small papules, yellowish in color with a moist surface and later exudate, hyperkeratosis, serum oozing from wounds, crusts, alopecia and scabs will be evident.
        - Lesions are caused by the intense pruritus;
            - licking, rubbing along fences, feed bunks, etc.
        - Chronic cases may present with dry lesions on the scrotum, perineum, sternum, ears and bases of horns.
        - Affected cattle will exhibit reduced feed efficiency, weight loss, secondary infections (pneumonia) and death.
- Psoroptic "Scabies"
    - Transmission
        - Direct contact
        - Infested pens ,barns, blankets, brushes, etc.
    - Diagnosis
        - Clip hair & Skin scraping
        - mites: - elongated mouth parts
            - pedicels (leg stalks) long & jointed
- Sarcoptic scabies
    - Different subspecies for different hosts
        - Sarcoptes scabei var.bovis
        - S. scabei var. ovis
        - S. scabei var. caprae

- Sarcoptes scabiei has become extremely rare in the United States in both cattle and sheep.
- Clinical signs similar to Psoroptes sp. but not as severe.
- Transmission & Diagnosis the same as Psoroptes sp.
- Mite is morphologically identical to scabies in dogs.
    - Body rounded, short, spines on dorsum, blunt mouthparts & pedicels (leg stalks) long and unjointed, 3rd and 4th pair of legs do not extend beyond body.

- Chorioptic Scabies
    - Chorioptes bovis (cow)
        - Tail Mange
    - Chorioptes ovis (sheep)
        - Foot mange
        - Hindlegs (C. bovis will occur in sheep with no signs!)
    - Chorioptes caprae (goat)
        - Head and neck.
    - Chorioptic mites live on the surface of the skin.
        - The mites live in sloughed skin, and hair.
        - Serum exudate occurs that dries to form crusts.
    - Lesions are generally not as severe as with the other scabies mites.
        - Lesions are found inside the hind legs, under the flanks and along the legs.
        - Pruritus is common, with infested animals licking, biting and rubbing
    - Clinical signs
        - Small wounds usually
        - Skin under thin scabs is only slightly swollen and inflamed (red)
        - Later as hair is rubbed off, skin appears thick, wrinkled and ridged.
    - Transmission
        - Direct contact
        - Infested pens ,barns, blankets, brushes, etc.
    - Diagnosis
        - Clip hair & Skin scraping
        - Chorioptes bovis
            - Body-oval
            - Mouthparts - blunt
            - Pedicels (leg stalks) - short and unjointed
            - Sucker disks (Caruncles)
            - Maximum size - female about 400µm; male 35µm
    - Treatment - Scabies
        - Highly contagious, in Kansas it is recommended (Required) that all cases of scabies mites in sheep, goats and cattle be reported to state veterinarian.
        - State authorities may require immediate quarantine of infected herds and institute control measures.
        - Approved treatments for scabies mites are:
            - Ivermectin, Doramectin, Eprinomectin, Moxidectin
            - Amitraz dip; twice at 7-10 day intervals
            - Coumaphos dip; twice at 10-14 day intervals
            - Permethrin dip; twice at 14 day intervals

Analgesia and Standing Chemical Restraint in Horses

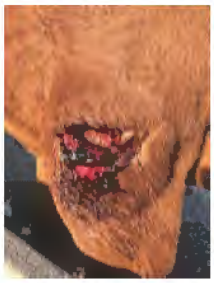
Dr. Warren Beard - Kansas State University



- Limit to sedation and analgesia for performing procedures

First consideration:

- Do I need sedation?
- Analgesia?
- Sedation and analgesia?



Other considerations

- Route of administration
  - IM, IV, CRI, epidural
- Time of onset
- Duration of effect
- Side effects of drugs
- Other drug properties
- Cost of drugs
- Record keeping requirements
- Drug residues USEF, AQHA, Racing, FEI

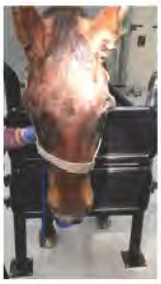
Route of administration

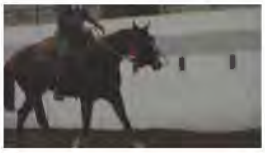
Time of onset

Duration of effect

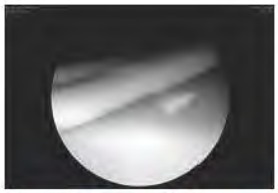



Side effects of drugs

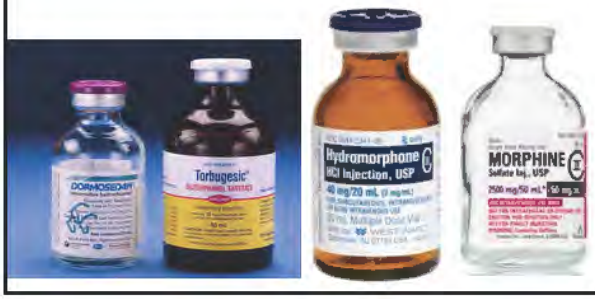


Other




Record keeping requirements



Drug residues



Cost of drugs- your cost
Always changing

- Acepromazine 50 ml — $19.43
- Xylazine 50 ml — $17.41 ($. 70)
- Detomidine 20 ml — $313.82 ($15.61)
- Detomidine gel — $15.57/dose
- Butorphanol 50 ml — $170.17 ($3.40)
- Morphine 500 mg — $$$ and not generally available
- Hydromorphone 20 ml — $26.79

- Detomidine + butorphanol ($20)

Midwest veterinary Supply
Jan, 2020

You may use the exact same criteria and reach different choices

- You place more emphasis on certain factors than I do
- Circumstances differ
- "cool factor" in using new drugs
- Desire to experiment
- Cocktails
- State regulations on compounding

Current Therapy 6 th edition

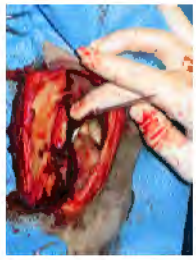
- Acepromazine
- Buprenorphine
- Butorphanol
- Chloral hydrate
- Chlorpromazine
- Cocaine
- Detomidine
- Diazepam
- Ethyl alcohol
- Fentanyl
- Guiaphenesin
- Ketamine
- Lidocaine
- Carbocaine
- bupivocaine
- Methadone
- Midazolam
- Meperidine
- Morphine
- Pentazocine
- Oxymorphone
- Promazine
- Romifidine
- Xylazine

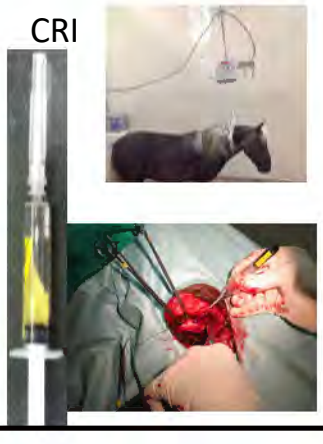
- Acepromazine
- Butorphanol
- Chloral hydrate
- Detomidine
- Diazepam
- Hydromorphone
- Guiaphenesin
- Gabapentin
- Ketamine
- Lidocaine
- Meperidine
- Morphine
- Pentazocine
- Promazine
- Xylazine

- Acepromazine
- Butorphanol
- Xylazine
- Detomidine
- Morphine
- Lidocaine
- Hydromorphone

CRI

- Lidocaine
- Detomidine
- "Pentafusion"
- "Triple infusion"





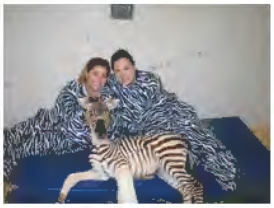
CRI

- Bolus 0.03 mg/kg IV romifidine and 0.01 mg/kg IV butorphanol
  - CRI 0.04 mg/kg/h romifidine and 0.02 mg/kg/h butorphanol
  - CRI of 0.04 mg/kg/h romifidine, 0.02 mg/kg/h butorphanol and 1.2 mg/kg/h ketamine
    - Bolus 0.03 mg/kg IV romifidine and 0.01 mg/kg IV butorphanol and 0.02 mg/kg IV midazolam
  - CRI of 0.04 mg/kg/h romifidine, 0.02 mg/kg/h butorphanol and 0.06 mg/kg/h midazolam.

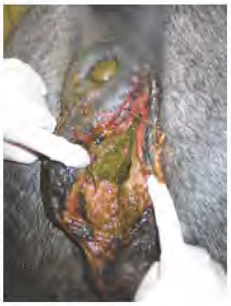
CRI

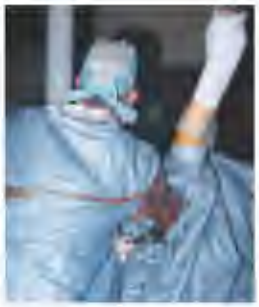
- Detomidine
- Xylazine / butorphanol
- Xylazine / morphine / ketamine
- Medetomidine / morphine
- Xylazine / lidocaine
- Xylazine / dexmedetomidine
- Dexmedetomidine / lidocaine
- Detomidine / morphine

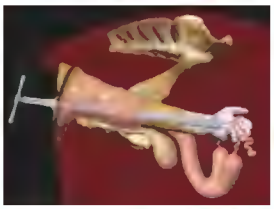
Standing joint lavage



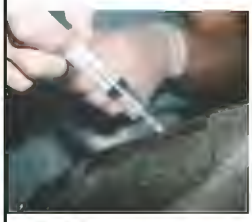
RV tear



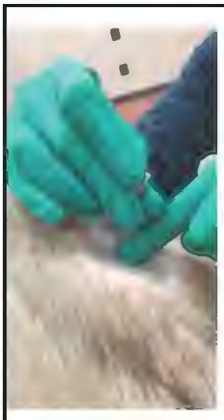


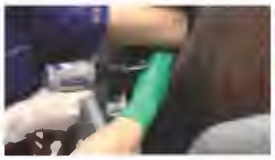


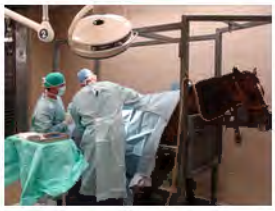
Epidural analgesia



- 18 ga, 1.5" needle
- Between coccygeal vertebrae 1 and 2 (or 2-3)
- Lidocaine, mepivocaine, or mepivocaine / xylazine
- Maximum volume of lidocaine = 7 ml
- Max total volume = 10 ml








Xylazine
Flunixin meglumine
Morphine
Pentazocine
Meperidine
Butorphanol

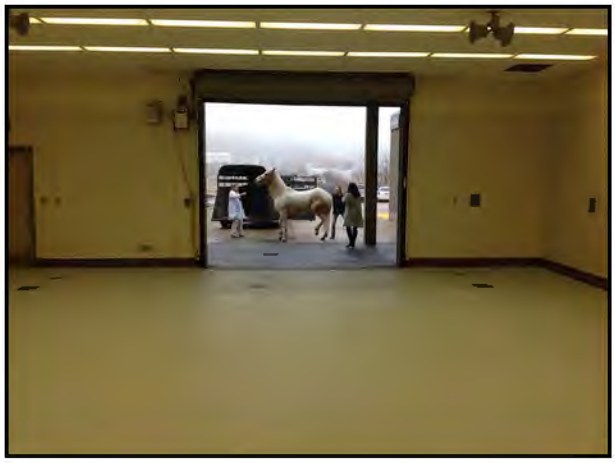
Cutaneous and visceral sensation

- Xylazine
- Flunixin meglumine
- Morphine
- Pentazocine
- Meperidine
- Butorphanol

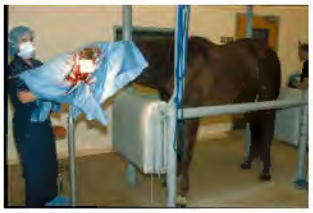
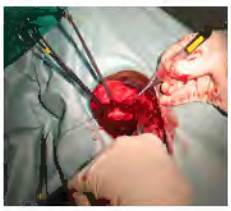
Effects of butorphanol, flunixin, levorphanol, morphine, and xylazine in ponies. Am J Vet Res. 1984 Feb;45(2):217-23.

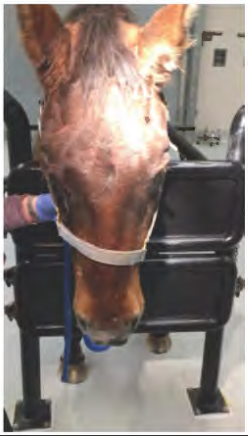
Visceral analgesia: effects of xylazine, butorphanol, meperidine, and pentazocine in horses. Am J Vet Res. 1985 Oct;46(10):2081-4.

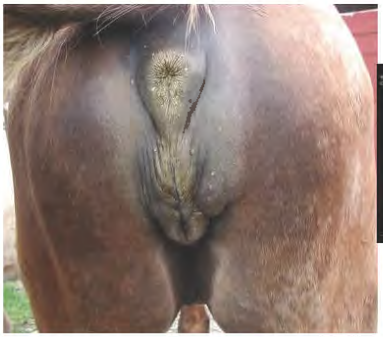
25 ga x 1.5”

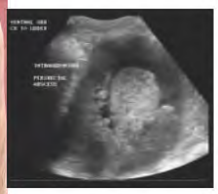



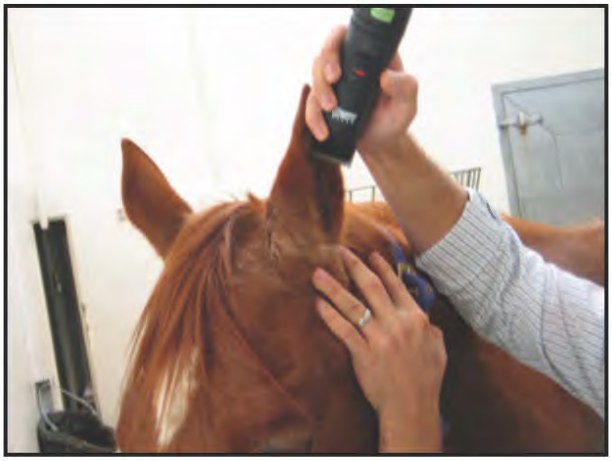
Standing enucleation

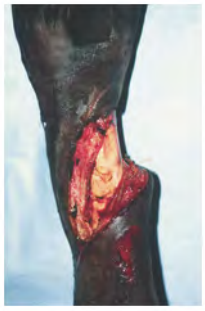



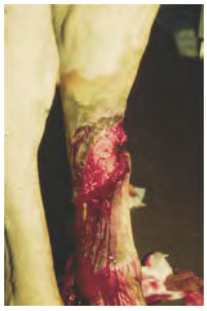












Summary

- Identify drug or drug combinations that meet your need for analgesia and sedation
- Match drug duration to a realistic time estimate
- Be aware of drug side effects
- Be aware of drug residues in performance animals
- Are you administering it or dispensing it to clients?

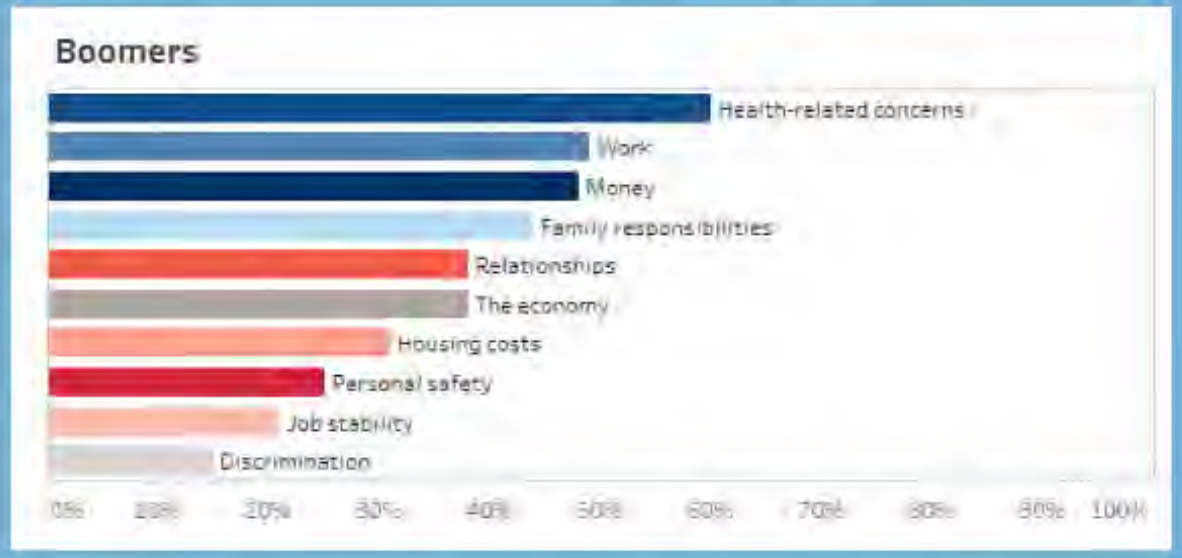
Financial & Emotional Reactions to COVID-19

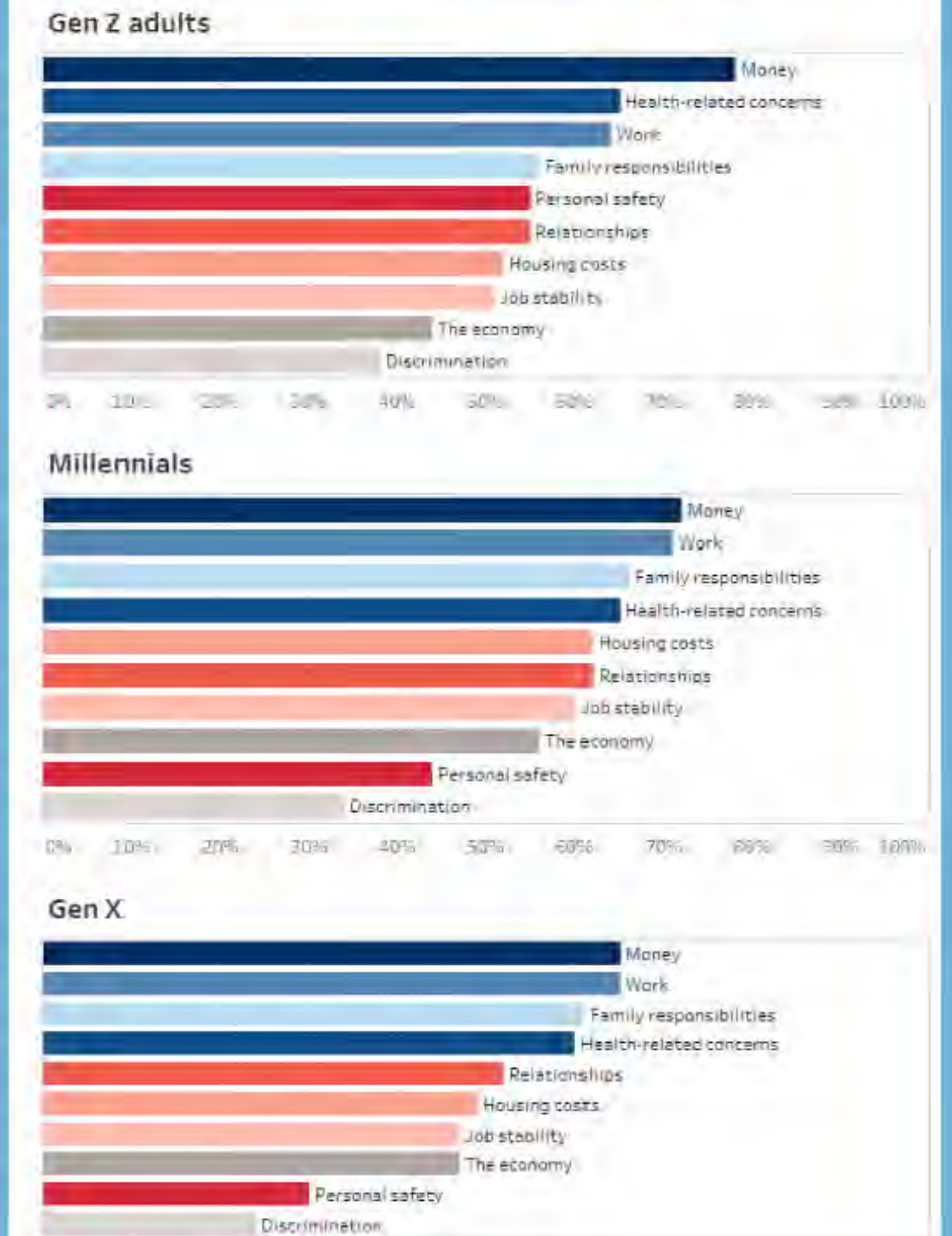
Dr. Sonya Lutter - Kansas State University

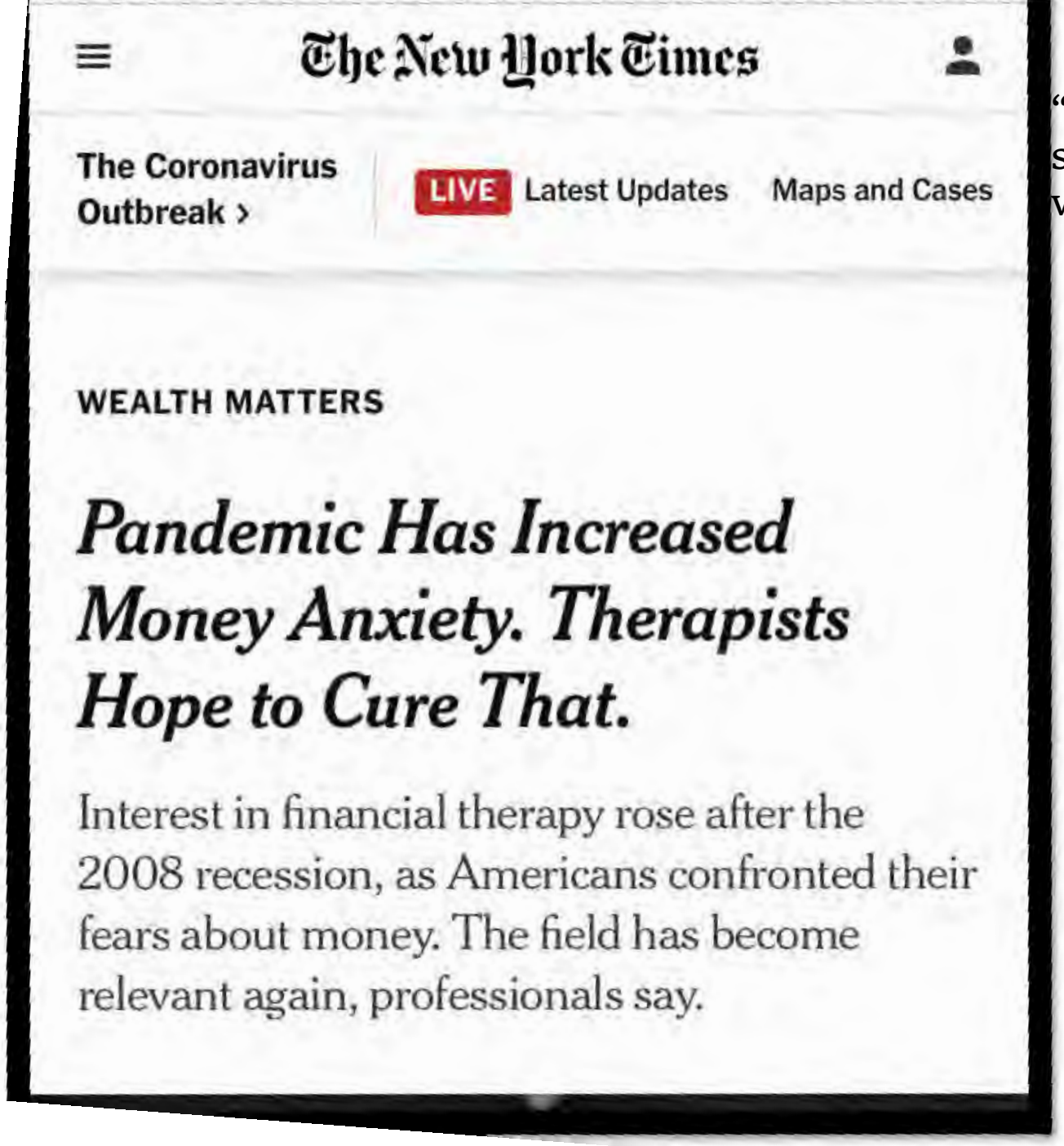
Financial & Emotional Reactions to COVID-19

Sonya Lutter, Ph.D., CFP®

Money tops the stress list







"This pandemic is like a black light," Ms. Clayman said. "It's suddenly revealing all the things that were present before but unseen."

"Americans are going to emerge from the coronavirus recession emotionally scarred in a way similar to the military veterans who suffer from post-traumatic stress disorder." – Brad Klontz

"People will get stuck in what is known as catastrophic thinking." – Amanda Clayman

"Planning for the worst-case scenario allows most people to understand that they will survive" –Brad Klontz

The lasting effects of the Great Recession

- Publications as recent as 2019 citing the negative impacts of financial loss on mental health…worldwide
  - Depression
  - Anxiety
  - Sleep disorders
  - Relationship problems
  - Substance abuse
  - Suicidal behavior

NY Times (2009)

“This feels absolutely different because it’s so widespread,” said Eric Dammann, a Manhattan psychoanalyst, in comparing this crash to 2001 and 1987. “It feels like everything is imploding at the same time as well as this sense that there is no light at the end of the tunnel.”

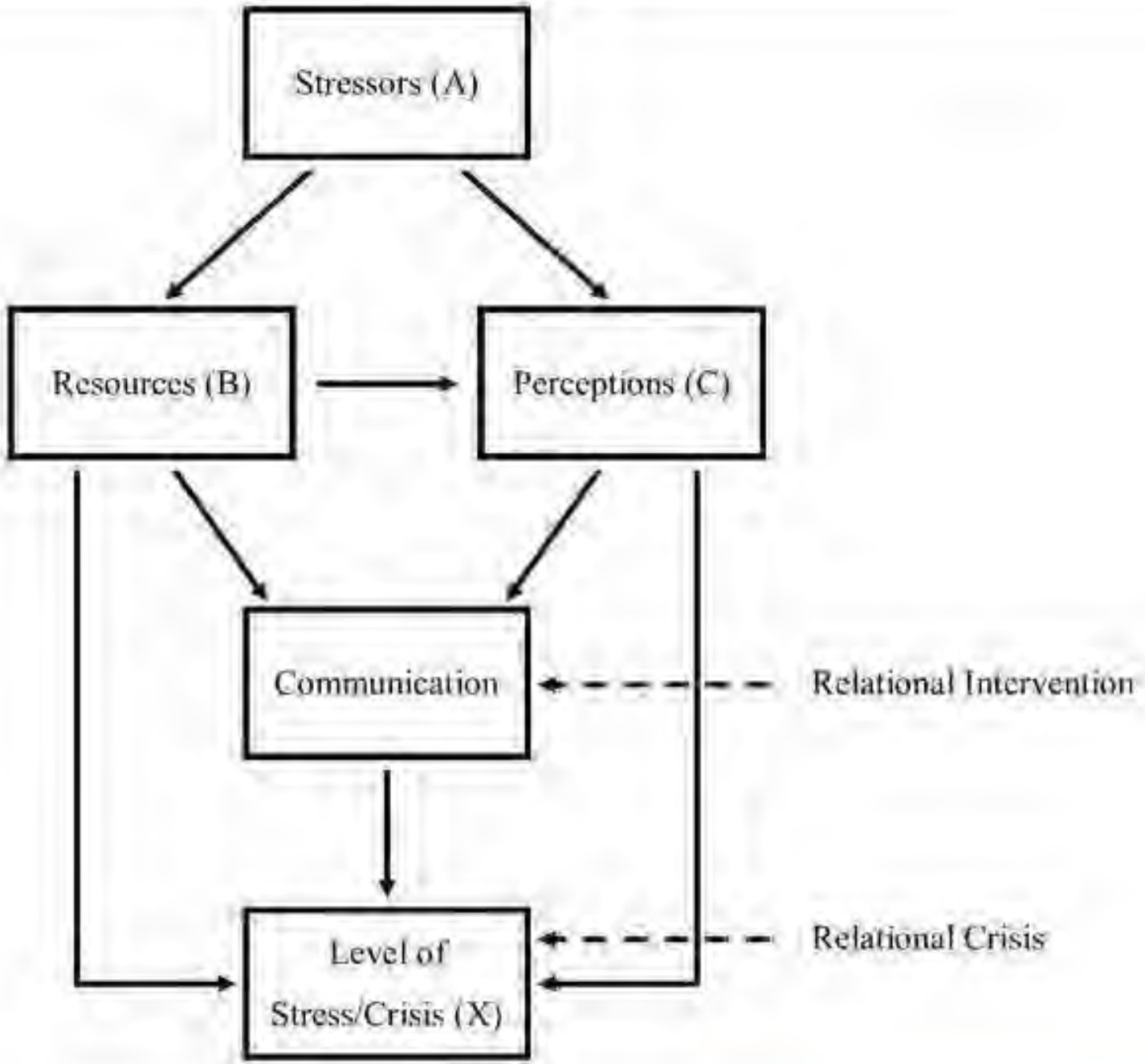
Without the chance to grieve, he said, “that’s when our thinking becomes rigid and bitter.”

There is consolation for those intent on keeping up with the Joneses. “I’ve been telling people that you’re just as rich now, because everyone has lost 30 percent,” he said.

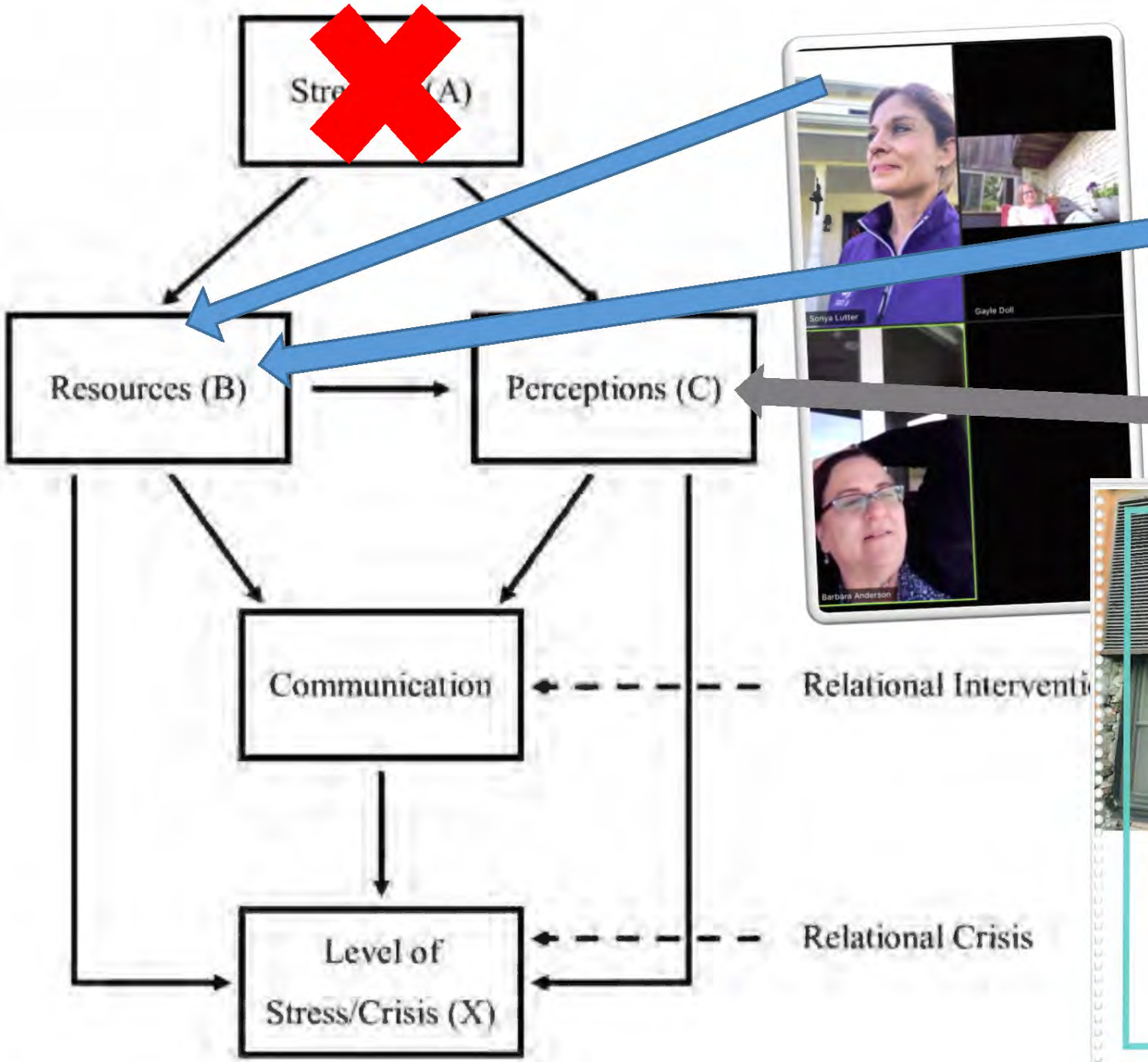
Strategies for Reducing Stress

From the American Psychological Association

1. Social media may escalate anxiety more than traditional media
2. Too much media of any kind can undermine mental health
3. Trustworthy information sinks in
4. A lack of control fuels stress
5. Managing stress ASAP can prevent long-term troubles



How do Money, Sex, and Stress Influence Marital Instability? https://newprairiepress.org/jft/vol8/iss1/3/



Nutrition

Sleep

Exercise

Reframe

AAMFT

Association for Marriage and Family

https://www.apa.org/topics/stress-tips

Talking to Clients about Money & Emotions

1. What do you do when your livelihood is dependent on livelihood of clients who are struggling?

2. How can you be a therapist without being a therapist to bring comfort to clients?

When someone shares something deeply personal, they aren’t looking for a solution.

People usually already have the solution.

What do you hope to get out of your visit today?

What do you want your situation to look at the end of today?

On a scale of 1 to 10, how much change do you anticipate as a result of your visit?

Talking to Your Partner about Money & Emotions

When someone shares something deeply personal, they aren't looking for a solution….they probably already generated dozens of ideas.

Why is it so difficult to start the conversation?

How to start the conversation

1. Schedule time
2. Define the problem
3. Identify how *each* contribute
4. Define what's worked
5. Brainstorm new solutions
6. Evaluate all ideas
7. Agree on one solution to try
8. Describe how each will contribute
9. Set another meeting to discuss progress
10. Reward progress

- **1. Set aside a good time to chat with your partner.**

2. Be kind to yourself and your partner and recognize that talking about money awkwardly is better than not talking about it at all.

3. Explore how your partner is thinking and feeling.

4. Talk about how you would like to be supported by your partner.

5. End with game planning and talk about what you can control in your lives.

Step 1: Low-stress communication

- Look for tension
- Take a break
- Walk around

Step 2: Engage all perceptions

- Values

ACTIVITY 2.1 THE MIND-BODY CONNECTION

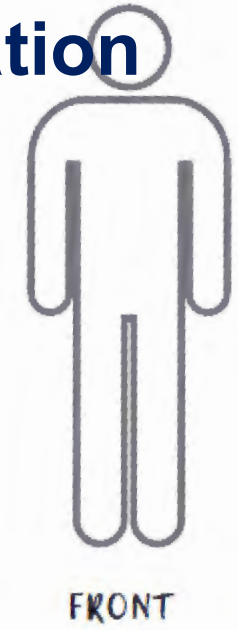
Being aware of how stress affects you physically can alert you to take action before it has a chance to build up. Where do you feel stress?

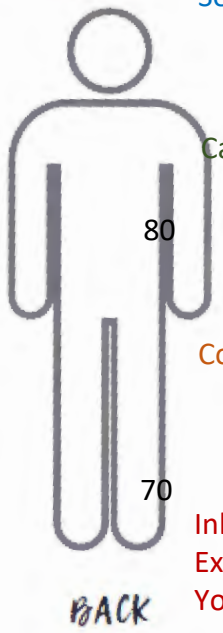
Angry

Stressed/Anxious

Frightened

Top of head, Back of head, Ears, Face, Neck, Back of neck, Left shoulder, Right shoulder, Chest, Stomach, Center of back, Lower back, Arms, Hands, Legs, Feet





90 Serene

Calm, cool, and collected

80

Concentration is wavering.

70

Inhale as you count to 5.
Exhale as you count back from 5.
Your brain needs a break.

Circle your most frequent type of stress symptom—physical, cognitive, behavioral or emotional

PHYSICAL	COGNITIVE	BEHAVIORAL	EMOTIONAL
Become fatigued	Lose focus	Drink	Feel anxious
Grind/clinch teeth	Can't sleep	Eat	Become short-tempered
Develop headache or migraine		Lose appetite	
Become physically ill		Avoid conversation	
		Lose interest in intimacy	

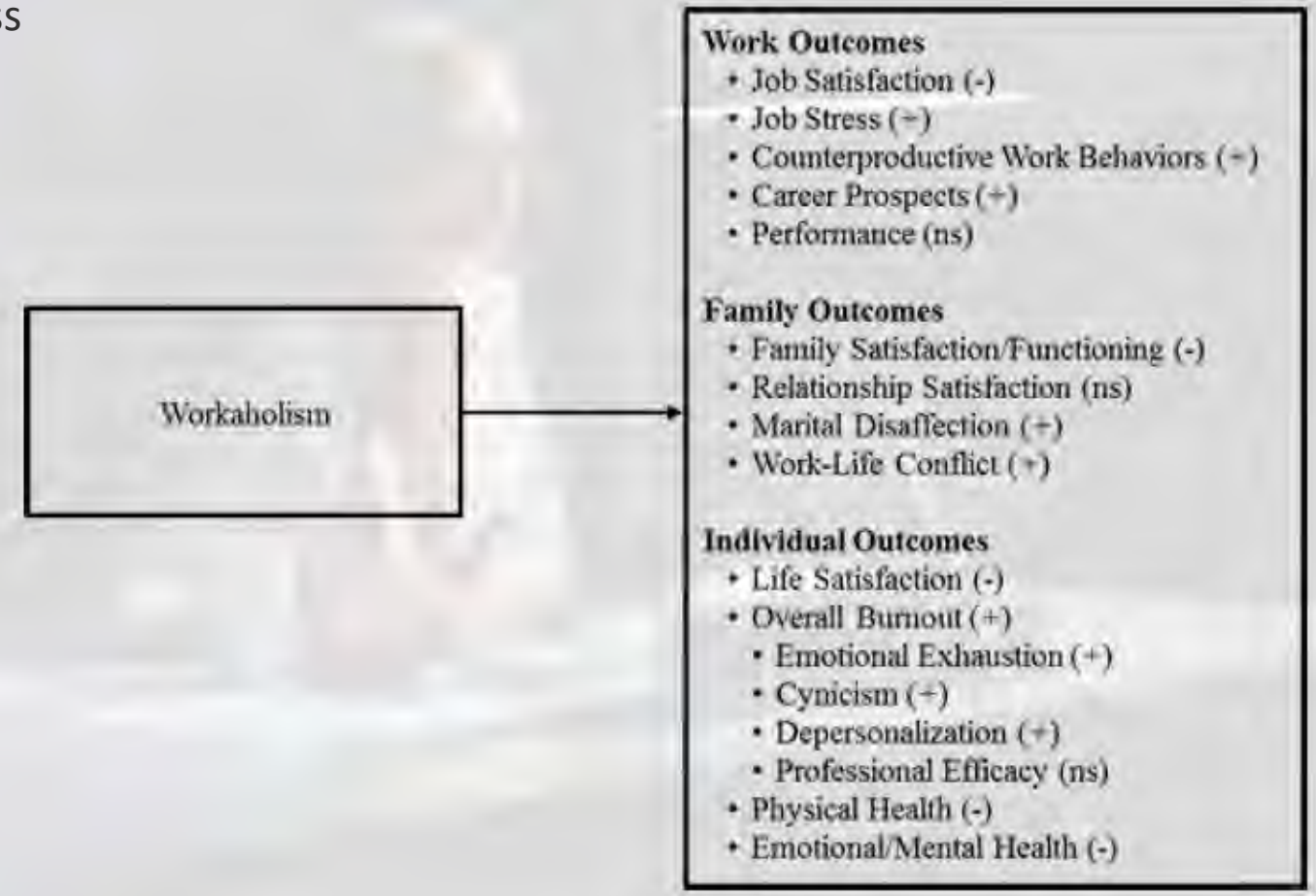
Stress = Myopism

Know what's contributing to stress and conflict

Workaholism

A. (1) Recurrent and persistent thoughts, urges, or impulses that are experienced, at some time during the disturbance, as intrusive and unwanted, and that in most individuals cause marked anxiety or distress. AND (2) The individual attempts to ignore or suppress such thoughts, urges, or images, or to neutralize them with some other thought or action (i.e., by performing a compulsion).
B. The obsessions or compulsions are time-consuming (e.g., take more than 1 hour per day) or cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.
C. The obsessive-compulsive symptoms are not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition.
D. The disturbance is not better explained by the symptoms of another mental disorder

Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition. American Psychiatric Association © 2013



Wife's perception of conflict decreases with feelings of good communication.

Husband's perception of conflict increases with more children.

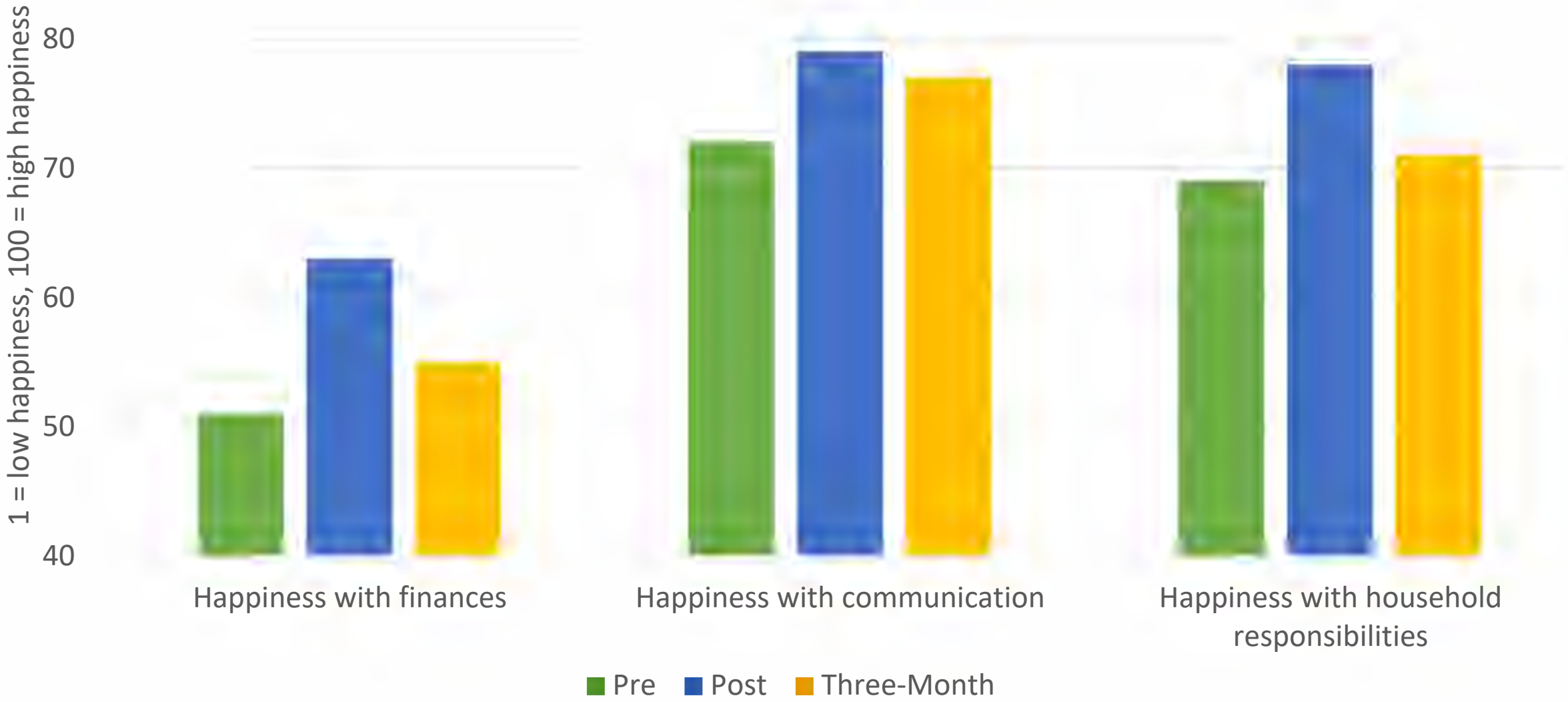
When women make more than men, conflict increases.

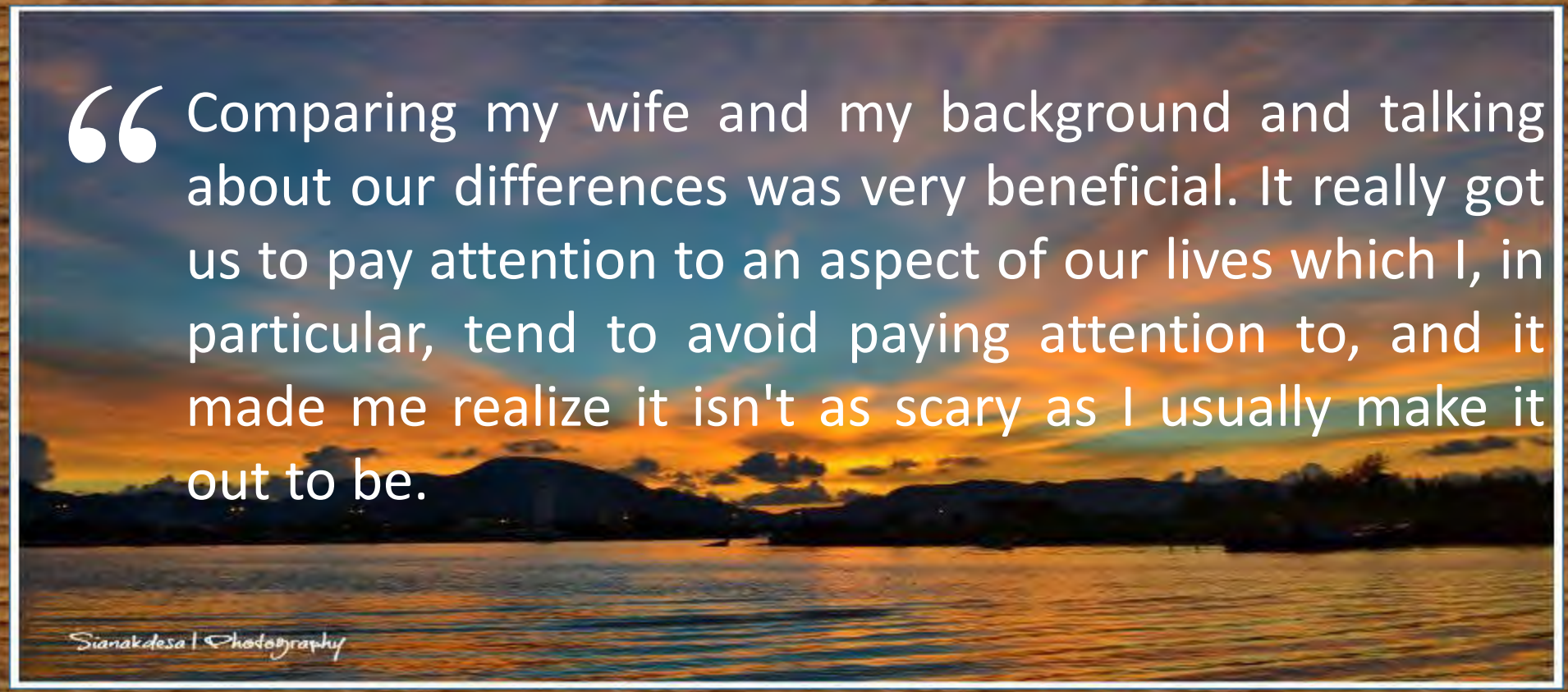
Joint or Separate Accounts?

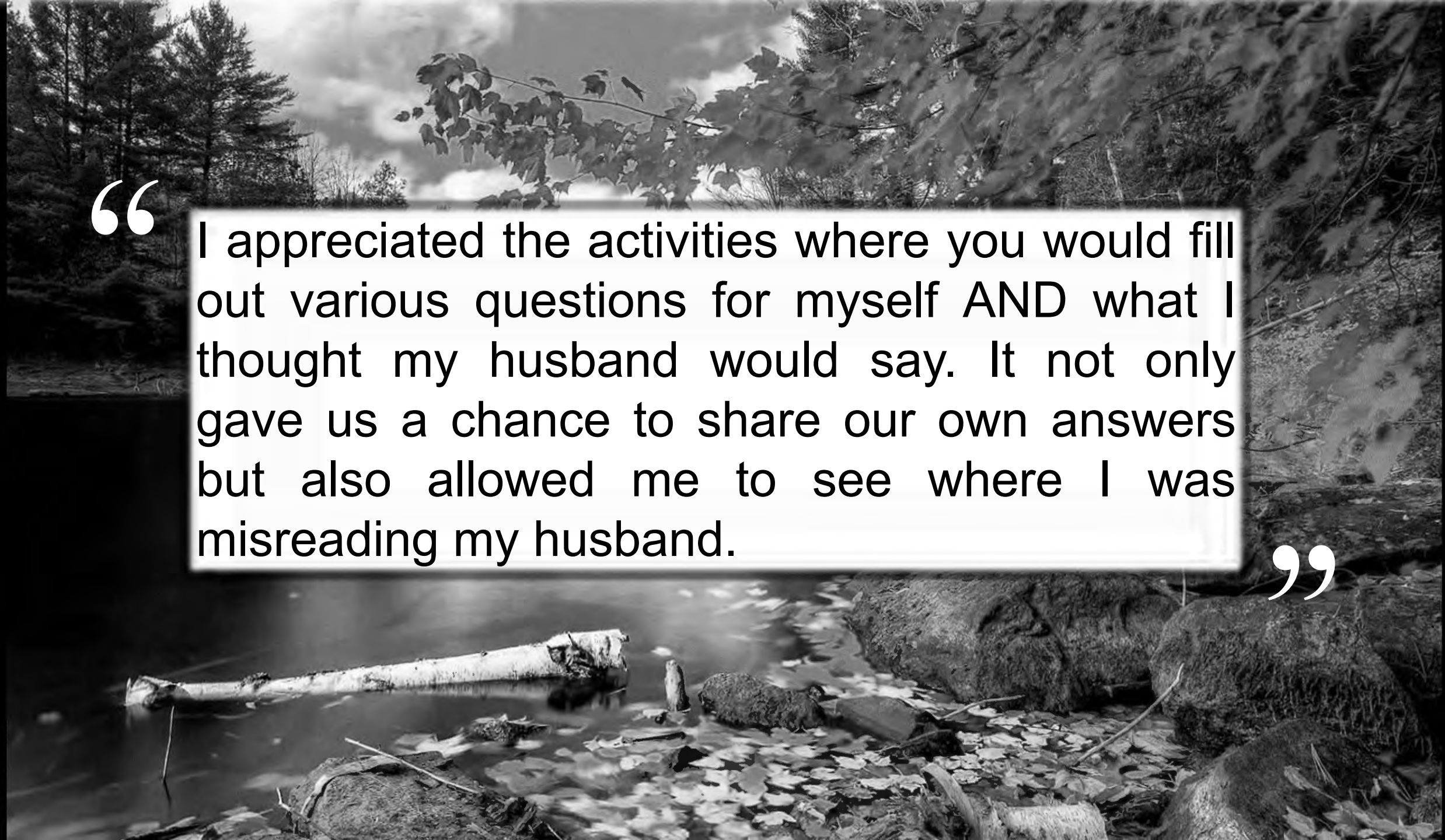
Joint accounts ⟺ Happier relationships

Those raised in homes with less arguing and more warmth usually have better financial habits.


50
40
30
20
10
1 = low stress, 100 = high stress
Financial Stress
Relationship Stress
Pre
Post
Three-Month


1 = low happiness, 100 = high happiness
80
70
60
50
40
Happiness with finances
Happiness with communication
Happiness with household responsibilities
Pre
Post
Three-Month


“Comparing my wife and my background and talking about our differences was very beneficial. It really got us to pay attention to an aspect of our lives which I, in particular, tend to avoid paying attention to, and it made me realize it isn't as scary as I usually make it out to be.”
Sianakdesa | Photography



“I appreciated the activities where you would fill out various questions for myself AND what I thought my husband would say. It not only gave us a chance to share our own answers but also allowed me to see where I was misreading my husband.”

A financial planner who participated in the curriculum stated,

> *I found a lot of value in the program and my partner and I communicate about money so much better now.*

Leading to more productive practices…

- The stress of our nation is greater than ever before in our lives.
- Be the person your clients and family need.
- Live your priorities.

Sonya Lutter, Ph.D., CFP®

lutter@ksu.edu

Notes