

AGRICULTURE NEWSLETTER

OCTOBER 2024

T.J. ADKINS, AGENT



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Lake Cumberland Area Cattle Pregnancy Determination School

**Participants will learn how to check cattle
for pregnancy using chuteside blood tests.
Class sizes are limited.**

Participants must call to reserve their spot.

**August 27th at 6:00pm EST
90 Gabby Shelton Rd
Parkers Lake, KY 42634
606-376-2524**

**October 29th at 6:00pm EST
TBD
Liberty, KY 42539
606-787-7384**

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Lungworms and Acute Respiratory Distress Syndrome in Cattle

Dr. Michelle Arnold, DVM, MPH UK Ruminant Extension Veterinarian

“Acute Respiratory Distress Syndrome” or “ARDS” is a rapid and dramatic onset of severe breathing difficulty due to lack of oxygen transfer across the air sacs (alveoli) in the lungs to the bloodstream. Affected cattle exhibit open-mouth breathing with the head and neck extended, nostrils dilated, a sway-back appearance, foam coming from the mouth, an open-shouldered stance, and sometimes become aggressive (see Figure 1). Breathing is shallow and rapid (35-75 breaths per minute) and may have a loud grunt associated with exhalation. Temperature is typically normal or mildly elevated depending on severity of the condition. In extreme cases, air pockets can be felt under the skin on the upper portions of the neck, shoulders and back (subcutaneous crepitation). Mild exercise is enough to cause the animal to collapse and die. Generally, there is no coughing nor signs of infection such as fever or depression. Severely affected animals frequently die within 2-3 days after initial onset of clinical signs.



Figure 1: Adult cow displaying signs of Acute Respiratory Distress Syndrome (ARDS) due to lungworm larvae migration. Photo courtesy of [Therapeutic Forum](#)

There are a variety of “agents” that will directly or indirectly damage the linings of the alveoli and blood vessels in the lungs of cattle, preventing gas exchange and initiating acute respiratory distress. The most common triggers include: 1) 3-methyl indole (3-MI), a toxin produced when cattle are moved from dry to lush pastures containing increased L-tryptophan levels; 2) the ketones from consuming the weed perilla mint; 3) certain respiratory viruses, especially bovine respiratory syncytial virus (BRSV); 4) lungworm infections; and 5) sepsis from bacterial infection. Although most producers recognize the symptoms of respiratory disease in cattle, lungworms are usually left off the list of possible causes. This is because respiratory disease due to lungworms is both uncommon and unpredictable, yet it can be devastating in a herd. Lungworm disease outbreaks are most often seen in grazing calves and yearlings exposed to the parasite for the first time and are therefore completely unprotected. But occasionally outbreaks are seen in adult cattle if their immunity to lungworms has waned from lack of exposure or pasture infectivity is high. The lungworm life cycle (Figure 2) begins when cattle consume infective L₃ larvae in the pasture, typically during wet summers or when grazing in swampy areas. After ingestion, larvae penetrate through the intestine, mature to the L₄ stage in the mesenteric lymph nodes, then migrate to the lungs and break into the alveoli, all within a week. These larvae continue to migrate to the airways and mature into adult worms inside the bronchi and trachea (Figure 3). From day 26-60 after infection (called the “patent phase”), the mature worms deposit eggs in the airways, where they hatch and the L₁ larvae ascend the trachea by causing the animal to cough, called “parasitic bronchitis”. The larvae are then swallowed followed by excretion in the feces. On pasture, the L₁ larvae mature to L₂ then L₃ in the fecal pat, spread to grass and are ingested to begin the cycle anew. The severity and duration of clinical signs depend on the number of larvae and how quickly they are

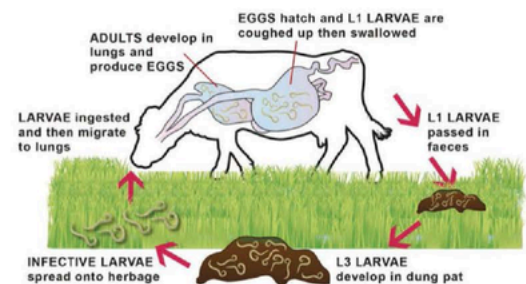


Figure 2: Lungworm life cycle. Accessed from “COWS – www.cattleparasites.org.uk”



Figure 3: Adult lungworms within the trachea, found on necropsy. Photo Courtesy of UKVDL Photo Archive

consumed. ARDS develops from a hypersensitivity (allergic reaction) to the larvae migrating through the lungs that causes an overreaction by the immune system that damages the lung tissues. Proteases and enzymes are also released by the larvae that directly damage lung tissue as they travel. Within an affected group of cattle, symptoms of lungworm infection may range from a mild, intermittent cough and weight loss up to severe difficulty breathing and death. Most animals gradually recover although it may take weeks to months. At the cellular level (visible through a microscope), this very distinct pattern of lung injury is called “diffuse alveolar damage” or “DAD” and is the most frequent microscopic finding in animals exhibiting acute respiratory distress. The old name of “Atypical Interstitial Pneumonia” or “AIP” has fallen out of favor due to confusion arising from a similar human disease with the same acronym.

Diagnosis of this type of pneumonia resulting from DAD, known as “interstitial pneumonia”, is easily performed at necropsy because it is a very different pattern from the typical shipping fever pneumonia. “Shipping Fever” or “Bovine Respiratory Disease (BRD)” causes a “bronchopneumonia” that occurs at the junction of the air sacs and the airways leading to them. On postmortem examination, damaged areas of shipping fever lungs are firm, red-tan, and mostly present in the cranioventral lung lobe. With interstitial pneumonia, on the other hand, damage is to the “interstitium” or supporting structures of the lungs including the lining of the air sacs, the lining of the blood vessels and the septa (divisions) between the lung lobes. These structures become filled with fluid and white blood cells, making the lungs feel wet, heavy, and meaty with a firm, rubbery texture and they do not collapse when the chest cavity is opened. The lungs themselves are often emphysematous or over-inflated. The entire lung may be diffusely involved or affected areas are dark red to purple and may be interspersed with normal looking lobules, creating a “patchwork” or “checkerboard” appearance. Cattle often develop secondary bacterial bronchopneumonia after the initial lung injury. Treatment decisions by the veterinarian will depend upon determining the etiology of the clinical signs but, if treatment is attempted, must be handled very cautiously. A dart gun can be used to avoid having to move the animal to a treatment facility as these animals die quickly when exercised. Treatment recommendations typically include diuretics and anti-inflammatory medications to facilitate breathing and antibiotics to prevent secondary bacterial infections.

The take-home message to remember is not all pneumonia cases in cattle are truly “shipping fever” that should be treated with antibiotics. As mentioned previously, lung damage can be from ingesting certain toxins such as when grazing cattle are moved from dry to lush pasture and develop “fog fever”, one of the first respiratory diseases known to cause ARDS. Lush pasture contains the amino acid L-tryptophan that can be metabolized by the rumen microbes to 3-methyl indole (3-MI). The 3-MI is absorbed into the bloodstream, transported to the lungs and metabolized to a new compound 3-methyleneindolenine (3MEIN) that causes widespread cellular damage to the lung. Recent studies have questioned the finding that tryptophan levels in pastures associated with fog fever are exceptionally higher than unaffected pastures. Instead, it could be the abrupt change from grazing the poor-quality dry forage to lush pasture

that increases the number of rumen microflora capable of metabolizing tryptophan to 3-MI. *Brassic*as including kale, rape, and green turnip tops are rich sources of tryptophan that can be converted in the rumen to 3-MI and potentially cause ARDS. Cattle mildly affected with fog fever show dramatic improvement within a few days with recovery spanning approximately 10 days. Severely affected animals often die but survivors can have long-term consequences of chronic emphysema or heart failure. Feeding an ionophore such as monensin or lasalocid has been shown to reduce the conversion of tryptophan to 3-MI in the rumen by as much as 90% but the ionophore must be present in the rumen at the time of exposure. Other potential causes of lung damage in animals due to ingested toxins include 4-ipomeanol from consuming *Fusarium solani*-contaminated moldy sweet potatoes, perilla ketone from grazing the weed purple mint (*Perilla frutescens*-see Figure 4), ingestion of the herbicide Paraquat, and ingestion of stinkwood (*Zieria arborescens*). Early involvement with your veterinarian is key to a proper diagnosis and appropriate treatment of any respiratory disorder in cattle. Establish a “daylight relationship” with your veterinarian now so he/she will know who you are when you call with an emergency.



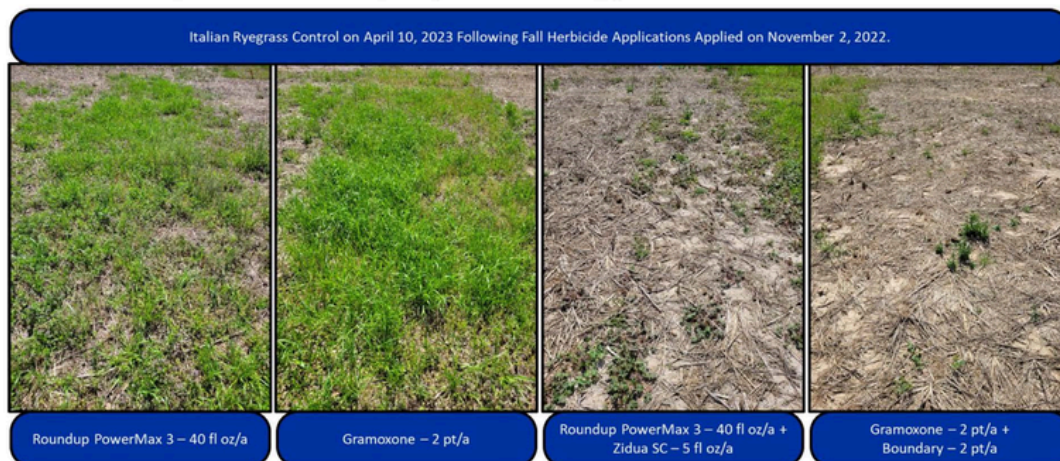
Figure 4: Ingestion of perilla mint causes acute respiratory distress syndrome as described with fog fever. Animals are frequently found dead with mature cattle most often affected but it can occur in yearlings and calves. Treatment is of limited value and severe cases seldom survive. Photo Courtesy of JD Green

Fall Residual Herbicides & Cover Crops Can Help with Ryegrass

Dr. Travis Legleiter, UK Extension Weed Specialist

Italian ryegrass (aka annual ryegrass) is becoming an increasingly problematic weed for Kentucky grain crop producers across the state. Growers are no longer just dealing with ryegrass in wheat, but rather are also dealing with ryegrass escapes and burndown failures prior to corn and soybean planting each spring.

Traditionally we have relied on spring burndown applications for control of winter annuals, including ryegrass, prior to corn and soybean planting. While this strategy is highly effective against the majority of winter annual weed species, Italian ryegrass is now challenging this strategy. We need to explore alternative strategies to reduce the pressure on spring burndown applications that are increasingly failing to control Italian ryegrass. This is where the use of fall residual herbicides is an option that can relieve the pressure on the spring burndown applications.



Several products that contain pyroxasulfone or S-metolachlor either have federal label language or 24 (c) special needs labels that allow for application in the fall for control of Italian ryegrass or fall germinating weeds. A list of products that have label language allowing for fall applications is contained in Table 1, along with application rates and replant restrictions.

Table 1. Herbicide products with federal or 24(c) labels allowing for fall applications for suppression of Italian ryegrass emergence prior to corn and/or soybean planting the following spring.

Trade Name Product	Active Ingredients (Site of Action Group #)	Labeled Application Timing	Fall application Rate (Medium Soils) ^{a,b}	Replant Restrictions
Anthem Maxx	Pyroxasulfone (15) + fluthiacet-methyl (14)	Fall applications for controlling weeds germinating in the fall or winter annuals	Corn – 4 to 5 fl oz/a Soybean – 3.5 to 4.5 fl oz/a	Corn & Soybean – 0 Months
Boundary	S-metolachlor (15) + metribuzin (5)	Control of glyphosate-resistant Italian ryegrass in the fall prior to soybean or corn planting the following spring (24c Special Needs Label)	Corn & Soybean – 1.8 to 2 pt/a	Corn – 4 Months Soybean – 0 Months
Dual II Magnum^c	S-metolachlor (15)	Fall application for residual control of glyphosate resistant Italian ryegrass in corn and soybean -	Corn & Soybean – .33 to 1.67 pt/a	Corn & Soybean – 0 Months
Helmet MTZ	Metolachlor (15) + metribuzin (5)	For control of glyphosate-resistant Italian Ryegrass in the fall prior to soybean planting the following spring (24c Special Needs Label)	Corn & Soybean – 2 pt/a	Corn – 4 Months Soybean – 0 Months
Zidua SC	Pyroxasulfone (15)	Fall/Winter application for controlling weeds germinating in the fall, or winter annual weeds	Corn & Soybean – 3.25 to 5 fl oz/a	Corn & Soybean – 0 Months

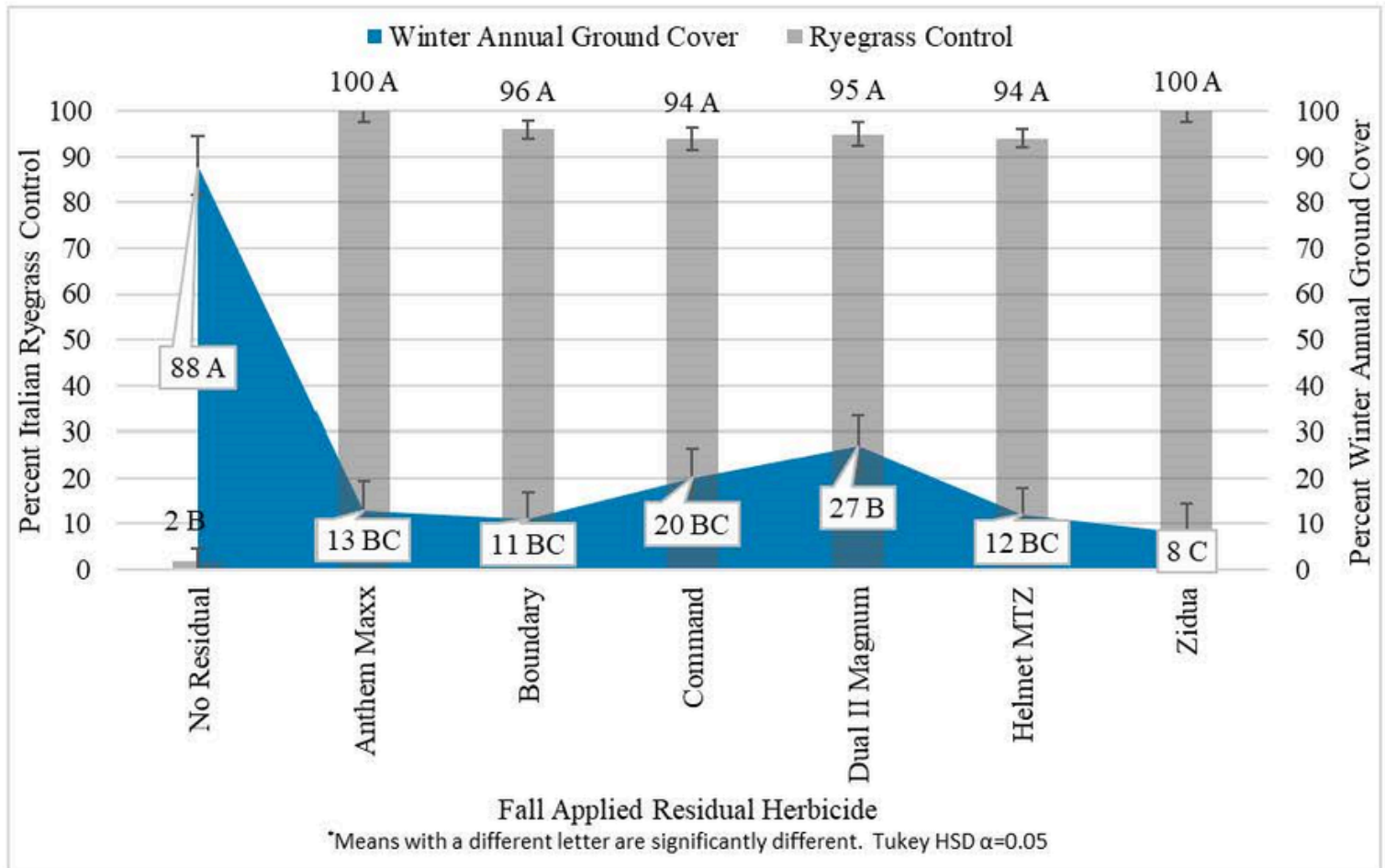
^a Check the herbicide label for product rates to use on fine and coarse soils

^b Refer to label for maximum seasonal/yearly rate allowance for each active ingredient.

^c Numerous generic formulations of S-metolachlor and metolachlor exist on the market. Check product label to assure fall applications for control of ryegrass are labeled for each specific product prior to use.

Research trials evaluating fall applied residual herbicides were conducted at the University of Kentucky Research and Education Center in Princeton, KY in 2022 and 2023. Additionally, a trial evaluating residual herbicide and cover crop combinations was implemented in 2023. Results of the experiments can be found in following figures with summary results directly below the figure:

Figure 1. Italian ryegrass control and winter annual ground cover in the spring following a fall residual herbicide application.



- All residual herbicides provided greater than 94% ryegrass control the following spring and had greater control than a burndown herbicide alone which provided 2% control of ryegrass.
- Winter annual ground cover was significantly reduced by all residual herbicide as compared to a fall burndown without a residual herbicide indicating an increased risk of potential soil erosion with the use of fall residual herbicides.

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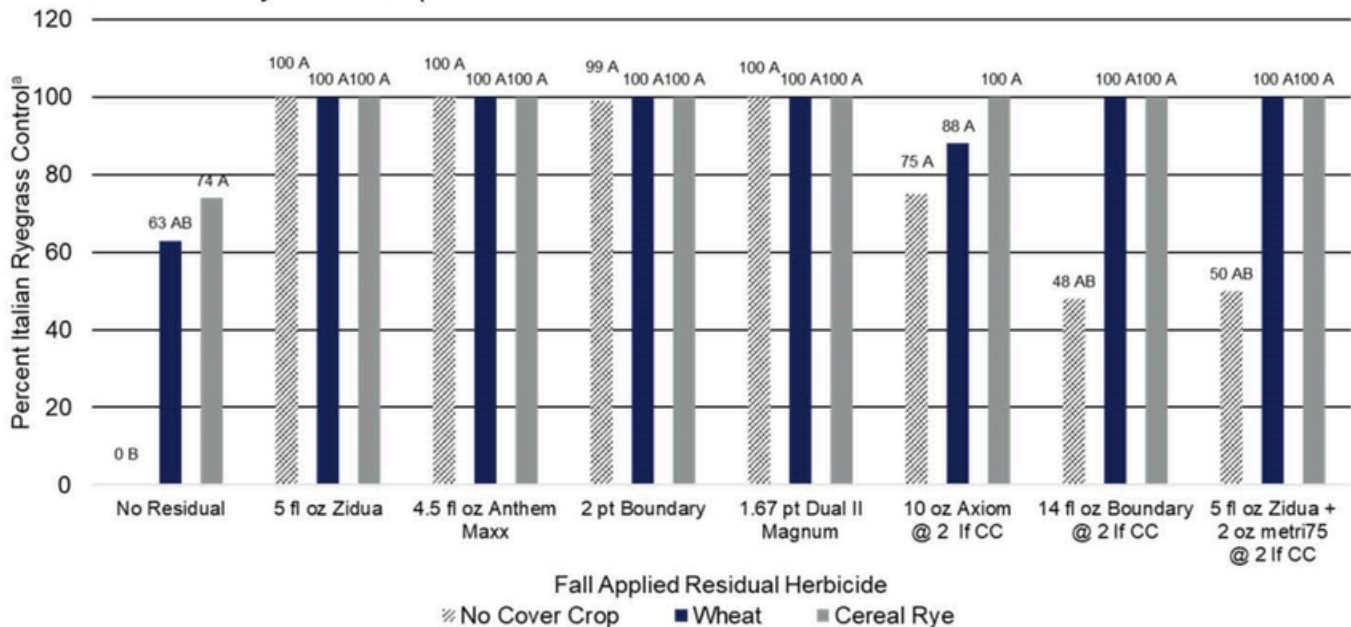
Table 2. Italian ryegrass density on April 3, 2023, following herbicide applications applied November 2, 2022.

Fall Applied Residual Herbicide	Ryegrass Plants per ft ²	
5 fl oz Zidua	0	A
4.5 fl oz Anthem Maxx	1	A
4.67 pt Dual II Magnum	2	A
2 pt Boundary	2	A
2 pt Helmet MTZ	1	A
No Residual Herbicide	14	B

^aMeans with a different letter are significantly different. Tukey HSD $\alpha=0.05$

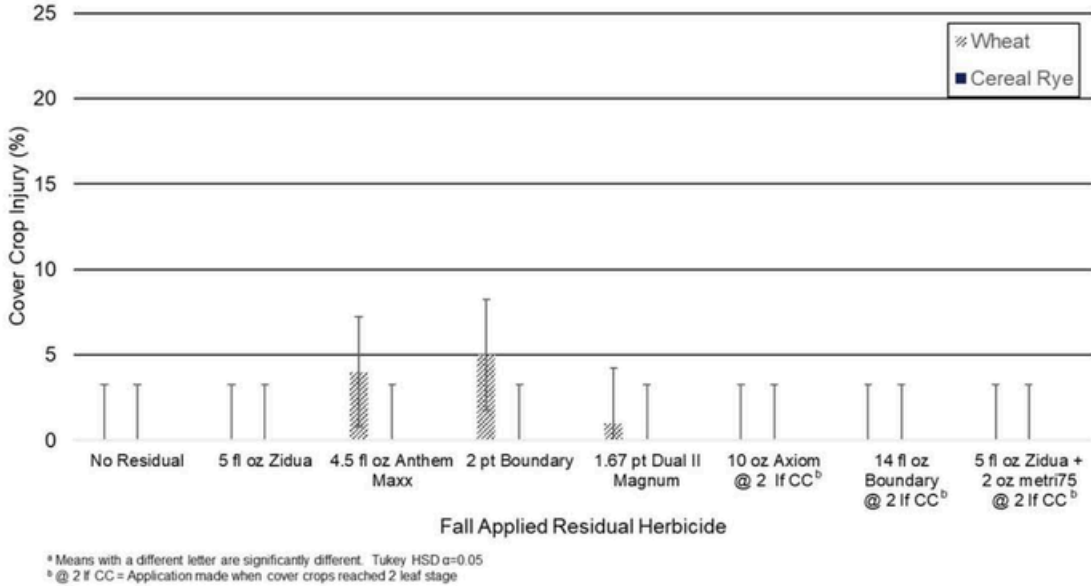
- Italian ryegrass density five months after fall residual applications was reduced to one to two plants per square foot as compared to a non-residual burndown application with 14 plants per square foot.

Figure 2. Italian ryegrass control on March 14, 2024, using fall residual herbicides with and without a wheat and cereal rye cover crop.



- The use of residual herbicides increased control of Italian ryegrass in the spring following applications both applied pre and post to cover crop emergence.
- The combination of residual herbicides and cover crops resulted in the greatest control of Italian ryegrass.

Figure 3. Cover crop wheat and cereal rye crop injury ratings following residual herbicide applications.



- Cover crop injury in 2023-24 was minimal on wheat, with less than 5% injury occurring. Cereal rye injury was insignificant in the 2023-24 trial.
- Cover crops were able to be established successfully in combination with both pre and post applications of residual herbicides allowing the use of a residual herbicide while minimizing overwinter soil erosion potential.

Recommendation for The Fall of 2024

These are my recommendations for those farmers dealing with Italian ryegrass based off these research results

- Farmers dealing with a highly suspected or confirmed glyphosate resistant Italian ryegrass population should apply a fall application of a tank mixture of paraquat (Gramoxone) plus either Boundary or Helmet MTZ. We know that paraquat and metribuzin have synergistic activity on Italian ryegrass thus the use of a residual premix with metribuzin will be beneficial.
- Farmers still able to control ryegrass with glyphosate should apply a residual herbicide with either glyphosate or paraquat. Those choosing to use paraquat see above for recommended residual tank mix partner. Those using glyphosate should include any of the residual herbicide listed in Table 1, all provided significant reductions in spring ryegrass densities.
- The incorporation of a cover crop of either wheat or cereal rye with a residual herbicide creates a scenario where Italian ryegrass suppression can be achieved while also maintaining a cover on the soil to reduce soil erosion potential.
- Plan to follow up with a spring burndown application to control any escapes. All residual herbicides provided significant reductions in ryegrass populations but did not provide 100% control of ryegrass in the spring.

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Beefy Stuffed Peppers

1 cup uncooked, whole wheat couscous	¼ teaspoon ground black pepper	4 large bell peppers
1 small tomato, diced	1 teaspoon salt	½ pound lean ground beef
½ cup garbanzo beans	½ cup low fat shredded mozzarella cheese	1 tablespoon chopped green onion
1 teaspoon dried Italian seasoning		1 tablespoon minced garlic

Cook couscous according to package directions. **Preheat** oven to 350 degrees F. **Combine** cooked couscous, tomato, beans, Italian seasoning, pepper, salt and mozzarella cheese in large bowl; set aside. **Remove** the tops, seeds and membranes from peppers. **Cook** peppers in boiling water for 5 minutes; **drain** upside down on paper towels. **Cook** beef until lightly browned in skillet. **Add** minced garlic and green onions to beef and sauté until

soft. **Drain** fat. **Toss** beef mixture into the couscous mixture. **Stuff** bell peppers evenly with mixture. **Place** in a lightly greased 9 x 9 inch baking dish. **Bake** for 15-20 minutes or until peppers are tender and cheese is melted.

Yield: 4 servings

Nutritional Analysis: 280 calories, 6 g fat, 2.5 g saturated fat, 35 mg cholesterol, 790 mg sodium, 36 g carbohydrate, 7 g fiber, 6 g sugar, 21 g protein



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