2004 BELTWIDE COTTON CONFERENCES, SAN ANTONIO, TX – JANUARY 5-9

ONSET AND PROGRESSION OF THE "HOLLOW SEED" (SEED ROT) MALADY OF SOUTH CAROLINA

J.R. Mauney
Jarman Industries
Scotsdale, AZ
J. McD. Stewart
Dept. Crop, Soil, and Environmental Sciences
University of Arkansas
Fayetteville, AR
Mike Jones
Dept. Crop, Soil, and Environmental Science
Clemson University
Clemson, SC

Abstract

Observations of bolls of the cultivar Maxxa in August and September, 2003, in Florence, SC, changed our beliefs about the timing and progression of symptoms leading to the hollow-seed malady in South Carolina. We now conclude that some outside agent is responsible for the decline in embryo growth and subsequent rotting of the seed. Symptoms were visible as early as 14 days post-anthesis (DPA) at which time the embryos were normal. The symptoms were seen to spread from the initial site to affect additional seed as the boll aged. Three sites of the initial symptoms were observed: The chalazal pore, the funiculus, and the seedcoat on the flanks to the seed. No insect puncture or disease organism could be identified as the initial cause of the symptoms.

Introduction

In 2003 we presented to this conference observations on an abnormal hiatus in seed development that we named "Hollow Seed." The abnormal seed had previously been termed "Seed Rot." Those observations grew out of a single visit to Florence, South Carolina, by JRM and JMS in September, 2002. At that time all the bolls examined had well-developed symptoms. Thus, it was difficult to determine the time-course and sequence of development of the symptoms. Our belief at that time was that the symptoms were the result of arrested embryo development. We reasoned that the failure of the embryos to complete their growth cycles in a timely fashion created an air gap between the embryo and the nutritive tissues that surround it. Under this scenario further development then ceased and the brown coloring of the nucellus and integuments was due to premature aging of these cells. This reasoning prompted us to further examine the phenomenon in 2003 to characterize the onset of the symptoms and to attempt to determine the causes of the retarded development of the embryos.

Methods

Seeds of the cultivar Maxxa were planted on May 15 in a field on the Pee Dee Research Station at Florence, SC. Maxxa was chosen because previous observations found it to be the cultivar with the highest likelihood of displaying the hollow seed malady. The plants reached full bloom about August 8, at which time flowers were tagged twice each week for 4 weeks. On August 28 bolls aged 14-17 and 21-24 days post-anthesis (DPA) were harvested, and their seeds were examined in detail with the aid of dissecting microscopes. On September 4 and again on September 20 additional bolls aged 14-17, 21-24, and 28-31 DPA were similarly examined.

Bolls were examined for embryo retardation and any cryptic symptoms that might indicate a malfunction leading to the hollow seed malady. Representative seeds on each date of sampling were recorded photographically. In addition, bolls were examined from plots of cultivars that are typically grown in the South Carolina. This was to determine if the symptoms being observed in Maxxa were present in other cultivars.

Results

Two observations of immediate import were made on the first sampling date. First, abnormal symptoms were beginning to appear by 14 DPA. Second, the earliest symptoms were not of retardation of embryo development. Robust embryos were observed in seed with distinct discoloration of the chalazal cap and the conductive pore at the center of the hypostase through which all nutrition to the embryo must pass. Thus, the premise upon which the experiments were based, namely, to discover he causes of embryo retardation, was immediately negated. Accordingly, the observations reported here are refinements of the description of symptoms that we began in 2002. We observed that:

1. The symptoms of discoloration of the seed coat seem to spread from seed to seed as the boll ages. That is, in 14 DPA bolls usually only one affected seed would be present, most often the second seed from the apex in a lock. Whereas, in 21 and 28 DPA bolls several seeds adjacent to one with more advanced symptoms would be affected.
2. Three avenues of entry of the agent were possible.
   a. Chalazal pore through the hypostase (Fig. 1).
   b. Laterally through the integuments where contact occurs with an adjacent infected seed (Fig. 2).
   c. Funiculus (Fig. 3).
3. Callus development or swelling of tissue accompanied the discoloration of the affected cells. Particularly the placental tissue below the point of funicular attachment of an infected seed was always enlarged. As a rule, infected seeds were larger in volume than normal seeds before drying.
4. Collapse of cells in the carpel wall near the hollow seed was observed frequently. Whether this was a response to the action of the agent on the seed or the point of entry for the agent could not be determined.
5. All cultivars examined had the same symptoms as Maxxa.

Conclusions

We conclude that the hollow seed malady is caused by an outside agent rather than by physiological retardation of embryo development. Three modes of entry by this agent have been observed listed in order of frequency: through the chalazal pore, Laterally through the integuments (seed coat), or through the funiculus. The agent is able to affect additional seed within a boll once it has established itself upon at least one seed. The agent induces callusing or enlargement of tissues and brown discoloration of affected areas. The embryos cease development after the symptoms are apparent, and the endosperm deteriorates, leaving air spaces within the embryo cavity. We did not see fungal hyphae in the effected areas, so our supposition is that the causal agent is an, as yet, unidentified bacterium or bacterial complex.

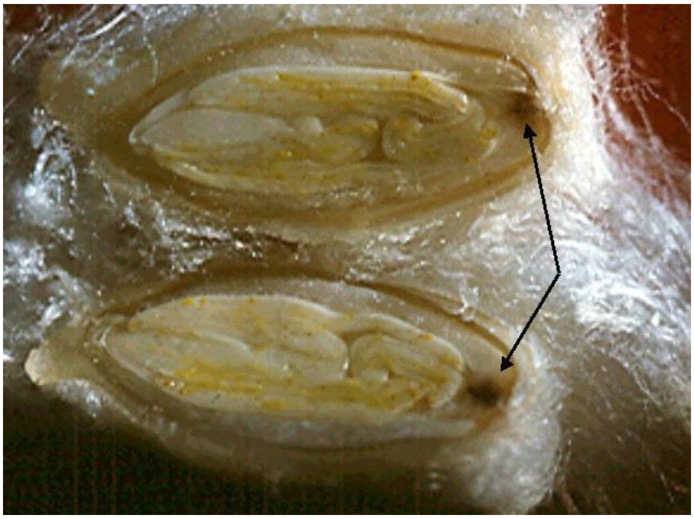

Figure 1. Seed with well developed embryo and late invasion of the chalazal pore (arrows). Following invasion and plugging of the conductive pore the embryo ceases to develop. When the invasion occurs early in seed development, the seed appears hollow at maturity.

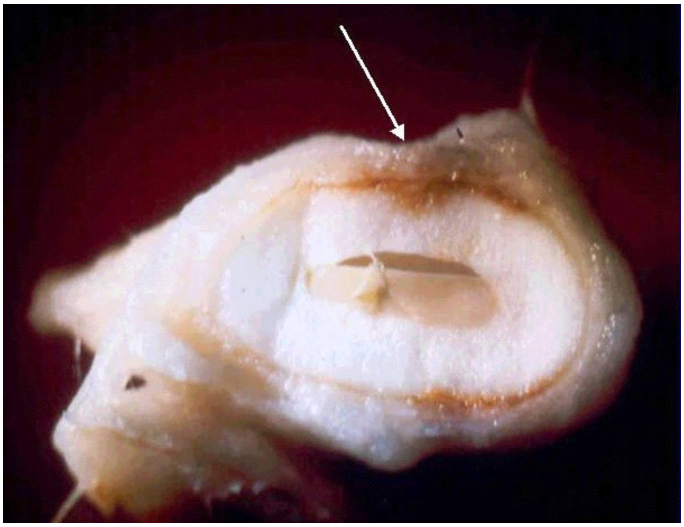

Figure 2. Invasion through the seed flank (arrow). When a developing seed is in contact with another afflicted seed in the lock, symptoms will develop at the point of contact and invasion through the integuments. The nucellar tissue will proliferate and absorb the endosperm, embryo development stops and a hollow seed results.

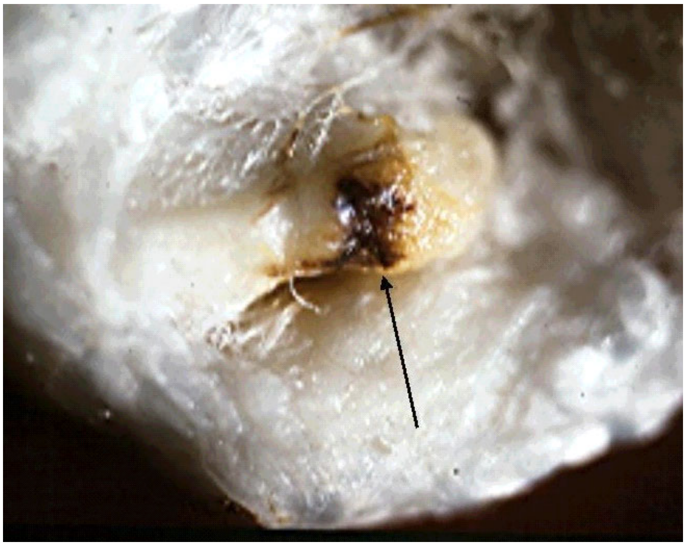

Figure 3. Invasion through the funiculus (arrow). Occasionally tissue in the area of the funiculus will become darked with cell enlargement in the adjacent area, suggesting that this may be a point of entry of the causal organisms.