

# 1928 Boll development and seed rot

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## INTRODUCTION

Boll rot is a common term used to describe a variety of diseases that influence the appearance of the mature cotton boll, even though the causal agents may differ substantially from each other. Approximately 170 organisms are described as causing "boll rot" (Anonymous, 2001). Generally, the boll will not mature completely, or if it matures under an area of high humidity, fungal organisms may attack the boll before it can open and the fibers dry. Very young bolls (0-5 DPA) tend to be sterile inside the ovary if it has not been penetrated by an insect. However, we have observed that plants under greenhouse culture produce ovaries that are much "cleaner" than plants grown under field conditions (unpublished observation). Although not examined specifically, this is probably due to the multitude of insect pests encountered in the field that feed upon the young bolls.

## EARLY STAGES OF FRUIT DEVELOPMENT

If a disease organism(s) is introduced into a boll by a piercing/sucking insect during approximately the first 10 days of development, the boll will probably abscise before disease can develop due to the hormonal influence of the wound or from the hormonal influence of the regurgitant (digestive juices) from the insect (Layton, 2000). This is especially true if the injury occurs within the first week of development. Insects that feed on the developing square (e.g., *Lygus*) are primarily targeting the developing anthers and not the developing ovules. When the flowers of squares (that have not aborted due to the feeding) open, they may be misshapen and will have only brown, shrunken vestigial structures on the side of the staminal column where the insect was feeding on the anthers (Layton, 2000). It is normal for these flowers to produce a full complement of seeds since it was the anthers that were attacked and not the ovules (which become seeds). Yield loss to piercing/sucking insects prior to flowering tends to be related to loss of the fruiting structure rather than loss of individual seeds.

## BOLL INVASION

After flowering, disease organisms may invade the developing ovary (boll) via wounds associated with insect feeding, especially stink bugs. However, the developing boll is susceptible to these piercing/sucking insects for only about the first 3 weeks. Although some evidence (Siebert et al., 2005) suggests that the brown stink bug, *Euschistus servus*, can damage older bolls. After this time, the endocarp of the boll undergoes secondary deposition and insects cannot then insert their stylus through this structure. Once the endocarp undergoes secondary deposition, the boll is also safe from chewing and burrowing insects such as the boll worm. The implication of this is that boll rotting organisms must either be endogenous (continually present) in the developing boll or must be introduced by insect feeding before the endocarp undergoes secondary deposition. It is highly likely that the latter is the case, because ovules at least 4 days of age are sterile when cultured in axenic medium (unpublished observations).

The entrance/exit of a worm into or from a boll is usually obvious as a hole in the carpel wall. The worm will feed upon the developing seeds in the carpel first entered and, if either

the secondary development of the endocarp tissue permits or the worm does not enter pupation, in adjacent carpels. In the case of external piercing/sucking insects, injury to the boll may be less evident on the external surface. However, when the insect inserts its stylus into a young developing boll, the feeding site is visible on the internal surface of the carpel wall as a small pimple (Anonymous, 2003). The insect regurgitates saliva that contains a mixture of hydrolytic enzymes that act upon the developing fibers; if feeding occurs prior to development of the fringe layer in the seedcoat, the insect may feed upon the developing seed itself.

If the developing fruit does not abscise from the insect damage (i.e., boll development is at least two weeks beyond flowering), under favorable environmental conditions of warmth and humidity the boll rotting organisms associated with the insects can grow on the damaged seed. Once established, the microbial organism(s) become invasive and move from seed to seed until the entire lock (or even the boll) is consumed with the classical rot symptom. At maturity, the rotted locks (and often the entire boll) will not open, or if it does open the fibers may not fluff and/or may exhibit the phenomenon known as "hard-lock." In these cases, the rot organism is usually a fungus.

The hard-lock phenotype potentially can occur in the absence of microorganisms vectored by insects. If a suitable environment of warmth and humidity exists in the crop canopy, as the boll matures, it may crack sufficiently that fungal spores can enter the boll. Because high humidity may prevent rapid opening of the boll, the fungus may have sufficient time to attack moist fibers and degrade the cellulose deposited in them such that the fibers do not "fluff" when the boll does open (Hollis, 2004).

### **SOUTH CAROLINA SEED ROT**

An exception to the pattern of development for boll rot described above was seen in the seed rot phenomenon that occurred in epidemic proportion in South Carolina in 1999 (Jones et al., 2000). The symptoms are also seen on *Acala Maxxa* whenever it is grown in South Carolina and has been used as a model system to study the symptomology when cultivars adapted to that environment showed no indication of the disease (Mauney and Stewart, 2003; Mauney et al., 2004). With Seed Rot, there was no visible evidence of insect feeding on the bolls and stink bug damage was easily discernable from the seed rot phenomenon by virtue of the penetration site, the blackened mat of fiber material upon which the insect fed, or in extreme cases an area of the seeds that had no fibers (caused by the hydrolytic enzymes of the insect regurgitant). However, work by Bell et al. (2005) and *Medrano and Bell (2007) suggest that stink bugs introduce the causal organism (*Pantoea*) by symptomless feeding.* In the case of the seed rot phenomenon, there were no easily discernable symptoms exterior to the seed other than the failure of the fibers to fluff upon drying in the open boll. The failure to fluff appeared to be more related to immaturity of the fibers than breakdown due to hydrolytic enzymes usually associated with boll rot.

Detailed scrutiny of the development of seed rot symptoms in *Acala Maxxa* grown in South Carolina, revealed the following progression (Mauney et al., 2004). The earliest indication of a symptom (by a dissecting microscope) cannot be discerned before about 14 days after flowering. This is a full week before symptoms are visible to the naked eye in bolls cut in cross-section. The first microscopic symptom is a slight discoloration (reddish brown) on the seed coat at the chalazal end of a developing seed. Classically, this occurs first on the seed located second from the apex of the boll. Two other symptoms were always associated with this discoloration: 1) darkening of the hypostase plug through which all nutrients supporting the embryo must pass, and 2) a slight enlargement of the placental tissue at the point of

funicular attachment of the ovule. Apparently, the degeneration of a functional hypostase is critical to the development of the cotton seed malady because embryo abortion results (unpublished observation). Once the endosperm is used up, the embryo ceases to develop and a hollow seed results. Fiber development also ceases. Either the embryo is critical to continued fiber growth (through hormone production), or the vascular system that feeds the fibers ceases to function in nutrient supply. In either case, the end result is that the fibers cease to develop normally in that they are immature at boll opening and fail to fluff. Because the disease develops between the 2-3 week-of-age period in the boll, the fibers usually reach their full length but are deficient in secondary cell wall.

After the "hard-lock" phenomenon, the symptoms first noted in this disease were observed when bolls older than 3 weeks were cut in cross-section. Premature darkening of the seed coat and the absence of an embryo were common and symptomatic of the malady. The first indication was a reddening and thickening of the seed coat (Jones et al., 2000).

Because of the unusually hot and dry environmental conditions beginning in June and lasting into August in 1999 in South Carolina, there was a tendency to associate the widespread occurrence of the disease with that weather pattern. However, it should be noted that the bolls most symptomatic of the malady in that year occurred low on the plant, and also that squares that developed during the hot dry weather did not show the symptoms. The progression of the disease suggests that, while weather may have been a factor, the predisposition for the seed to cease development was probably due to the cool raining period that preceded the hot dry period. The most symptomatic bolls were the squares that developed during that period, thus they may have been unusually conditioned and susceptible to high temperature (Mauney and Stewart, unpublished observations).

The reader will note that reference is always made to the phenomenon as a disease rather than a physiological malady. In fact, the malady appears to be an interaction of opportunistic organisms and the environment. Note (as described above) that the first symptomatic indication was on the chalazal end of a single seed. However, where this first symptomatic seed was in contact with an adjacent seed the discoloration would spread to the adjacent seed at the point of contact and could thus spread throughout the lock with the basal seeds usually being the last to show symptoms. The "spread through contact" strongly suggests that a pathogenic organism, probably bacterial in nature, is involved in the seed rot malady (Mauney et al., 2004). Also, absence of insect feeding sites, enlargement of the placental tissue, and first occurrence near the chalazal cap of the developing seed all suggest that the initial infection is probably from an endogenous opportunistic source, probably residing in the vascular system. In all cases, the tissues of the hypostase appeared to be compromised (unpublished observation).

## **CONCLUSIONS**

Most incidences of boll rot are related to introduction of pathogenic organisms to the interior of the boll by insects long before the boll matures. Obviously, only those bolls that do not abort (remain on the plant) can experience the rot phenomenon. Depending upon the environmental conditions and the microorganisms involved, only the contents of the affected lock, or in extreme cases, the entire boll including carpel walls may rot. In cases where the major symptom is "hard-lock," the causal organism may be introduced into the boll late in development and attack the fibers, or alternatively may be endogenous organisms that cause early embryo abortion that lead to immature fibers.

## REFERENCES

- Anonymous. 2001. Crop profile for cotton in Alabama. **NSF Center for Integrated Pest Management**, North Carolina State University.  
<http://www.ipmcenters.org/cropprofiles/docs/alcotton.html> (Confirmed 6-25-07)
- Anonymous. 2003. Stink bugs. Cotton Insect Control Guide. Publication 343. Mississippi State Extension Service. <http://msucares.com/insects/cotton/profile/stinkbugs.pdf> (Confirmed 6-25-07)
- Bell, A.A., J. Lopez, J. Esquivel, E. G. Medrano and J. Mauney. 2005. Isolation of cottonseed-rotting *Pantoea* spp. from stink bugs and plant bugs. In: Proc. Beltwide Cotton Conf., National Cotton Council, Memphis, TN.
- Hollis, P. 2004. Progress being made in battle against hardlock in cotton. Southeast Farm Press Mar. 1, 2004.  
[http://southeastfarmpress.com/news/farming\\_progress\\_made\\_battle/](http://southeastfarmpress.com/news/farming_progress_made_battle/) (Confirmed 6-25-07)
- Jones M.A., J.D. Mueller, D.A. Kluepfel, M.J. Sullivan, J.T. Walker, M.E. Roof, J.McD. Stewart and D.E. Linveil. 2000. Preliminary investigations on cotton seed rot in South Carolina. South Carolina Agriculture and Forestry Research Station Bulletin 675. Clemson University, Clemson, SC.
- Layton, M.B. 2000. Biology and damage of the tarnished plant bug, *Lygus lineolaris*, in cotton. Southwest. Entomol. 23(S):7-20.
- Mauney, J.R., and J.McD. Stewart. 2003. Embryo development within bolls exhibiting the "hollow seed" syndrome. Proc. Beltwide Cotton Conf., National Cotton Council, Memphis, TN (CD ROM).
- Mauney, J.R., J.McD. Stewart and M.A. Jones. 2004. Onset and progression of the "hollow seed" (seed rot) malady of South Carolina. Pp. 1967-1969. In: Proc. Beltwide Cotton Conf., National Cotton Council, Memphis, TN.
- Medrano, E.G. and A.A. Bell. 2007. Role of Pantoea agglomerans in opportunistic bacterial seed and boll rot of cotton (Gossypium hirsutum) grown in the field. J. Appl. Microbiol. 102:134-143.*
- Siebert, M.W., B.R. Leonard, R.H. Gable and L.R. LaMotte. 2005. Cotton boll age influences feeding preference by brown stink bug (Heteroptera: Pentatomidae). J. Econ. Entomol. 98:82-87.