

Canker-Infested Butternut Trees With 30-Year Survival

P.A. Rutter, January 2007. © BRC



The butternut tree (*Juglans cinerea*), a relative of the black walnut, is native to most of eastern North America. Never common, it is now severely threatened by yet another introduced fungus disease: *Sirococcus clavignenti juglandacearum*, commonly called “butternut canker”. Throughout the range of the tree, 70-90% of the trees are already dead, and most of the rest are “dying.”

We have an extremely unusual situation with our wild butternuts growing in the woods on Badgersett Farm: many of our trees are definitely infected with the canker—and yet they survive, even thrive and grow—for *decades*. So far we’ve been observing them closely for 30 years. The disease was well advanced from the outset.

The photo to the left is typical of the status of the disease here—abundant and aggressive. This tree is a youngster stemming from a major establishment year around 1978. That year we had a very heavy butternut crop, and made a concerted effort to hunt down (eat) the squirrels after the nuts were buried. That did, indeed, result in some 100 new butternut seedlings in about 45 acres of woods.

Those seedlings were “random”, from the open pollinated wild trees already present. The farm woods originally had a typical butternut population for this area, averaging 1 very large tree (DBH 2-3’) per 2 acres and about 1 young recruit (DBH 6-10”) per acre.

The fate of these seedlings has been fascinating. All of them have become infected with the

canker, but ultimately, some of them survive and even “recover”. Trees as heavily infested as the one pictured here are very unlikely to survive. This one is taking a very long time to die, however; most of its peers died 10 years ago.

The disease is still abundant here, and trees continue to die from time to time. The last of the big original trees succumbed in 2002; old age may well have been a contributing factor. One tree provides a significant exception to the general rule of decline and death: a medium-sized canopy tree (DBH 18”) 30 years ago, it continues to thrive and produce seed crops, in spite of being infected the entire time. It grows and produces nuts, even though infected. It is not a hybrid of any kind but a pure butternut. And some of its seedlings are now doing the same thing and moving into the canopy.

A technical reassurance for the plant pathologists: we’ve had the fungus identified. It is butternut canker.

What Does This Mean?

We’d love to think it means the surviving trees are genetically resistant to the fungus, but we really cannot say that yet. They may be, but there might very well be other things going on. The two most likely possibilities: we have a strain of the disease that is not as virulent as most; or something else in our woods is attacking the disease fungus, possibly another fungus, or a virus.

Finding out definitively which possibility is true would need the equivalent of a PhD thesis, at least—more likely, 3 or 4 theses.

In the long run, IT DOES NOT MATTER WHICH ONE IS TRUE. So long as the trees survive, produce seed, and produce viable seedlings, we have a functional population—one that is able to increase and evolve further in its relation to the fungus.

We want to forcefully point out here that if we’d followed standard forestry advice given 30 years ago (and even now), we would have “removed diseased trees to prevent further spread”. We would now have no disease, because there would be NO BUTTERNUT TREES LEFT ALIVE. All of them are “infected”. But for some of them, it turns out this fungus is the equivalent of the flu, not bubonic plague. They may sniffle for a long time, but they function. In fact, the ultimate severity of an infection can only be determined by allowing the disease to run its course. For many forest trees, that may mean watching the tree for a minimum of 30 years. It is impossible to look at a diseased tree once and say that it is going to die. It very well may not. In watching our trees over the years it is clear that it may appear the fungus is winning for 5 years, then the tree for 5 years, then the fungus for 4 years, etc. A drought year, or a wet year, sometimes seems to provide the trigger for a balance shift.

Training in evolutionary processes suggests a very different rule for managing trees in the middle of an epidemic: Rutter’s Rule: “If It’s Not Dead, DON’T KILL IT.”

Too often, the advice to manage disease by removing infected trees is based only on the desire to “do something”. The only thing assured by this policy is that there will be fewer trees. The scientific study of the natural processes of evolution of resistance to catastrophic selection pressures is extremely uniform—the starting point is almost without exception individual organisms that get sick, but which do not quite die. Survivors then breed with each other, and usually some progeny will turn out a little more resistant than either parent. Et cetera. Virtually never do you find the one rare individual that “just never got sick”. It doesn’t happen. In the case of the American chestnut, all “uninfected” trees ever examined turned out, after long and expensive study, to simply be statistical flukes—they’d just never been exposed. All died quickly when the fungus finally got there. Funguses are not predators; it’s purely a matter of chance whether a fungus spore arrives or not. Sometimes, rarely, it doesn’t.

Canker Development and Healing at Badgersett



Figure 1. A butternut with multiple lesions, including 3 different “basal” cankers, which frequently can open the tree to attack by other pathogens, as well. Trees thus infected are highly likely to succumb.

These cankers actually show some signs of healing—the formation of scar tissue by the tree. But for this tree, it’s usually a see-saw process: the tree heals a little, the fungus re-attacks, the tree heals a little, etc.



Figure 2. This tree shows serious infection and little sign of healing—no swelling around the black oblong lesions.

Trees this susceptible and heavily infected are not likely to live long.

The photos below illustrate the non-typical healing we've documented here.



Figure 3. Here is a lesion that has completely healed over. Potentially, this is the end of this particular infection site.



Figure 4. However, here is a canker that healed over- but then re-opened; the tree is in a see-saw with the fungus.

For many of our trees, the see-saw may take years to shift balance, and they may shift balance many times.



Figure 5. This tree is one of the 2nd generation that seems to truly heal.

Each white oval is indicating a healed canker which was sprayed with white tree paint- 6 years before this photo was taken.

At the time the paint was applied, the cankers looked like the one in Figure 3: healed, but quite obvious.



Figure 6. This is the same photograph as Figure 5 but without the white ovals, which obscure some of the detail. If the spots where the cankers were didn't have a little remaining white paint, it would be difficult to know that there had ever been a wound here.

The tree has truly, completely healed the lesions, and they do not recur.

This tree does still get occasional new cankers; but so far, after a year or two, they always close up. New cankers seem to happen on young wood only; the trunk no longer gets any.

Is Badgersett The Only Place This Happens?

It's extremely unlikely. Why hasn't anyone else documented this? We think it is because of the general practice of "destroy all diseased trees"—sometimes called "sanitation"—and the rarity of long-term trained observation.

"Sanitation" is actually a more complex mistake than it first seems. Regardless of whether our trees survive because they are genetically somewhat resistant or because some organism antagonistic to the fungus has showed up here, removing the diseased trees would have prevented EITHER pathway to survival from working.

In the case of genetics, it's very common for a single gene for resistance to become much stronger if it can be homozygous instead of heterozygous, or if it mutates into multiple copies. But if all the "half-dead" (no, they're half alive) trees are killed, obviously, they cannot breed with each other.

In the case of any organisms antagonistic to the fungus, the first thing they need to appear is...THE FUNGUS. If the disease is kept as low as possible, the ability of super-parasitic fungi (or viruses) to become established is drastically reduced.

Unfortunately, few are trained to distinguish between minor diseases, where "sanitation" can be a useful practice, and catastrophic plagues, where sanitation—if you mean killing all the sick trees—can mean extinction.

The two different situations:

1) *Minor disease*: say, 25% of the population is affected and removed.

Result: the healthy 75% of the trees are left alive to reproduce.

2) *Catastrophic disease*: say, 90% of the population is affected and removed.

Result: 10% of the trees are left alive to...actually, probably die of other causes, including later disease, or "harvest" because the wood is now rare and valuable. The gene pool is almost certainly reduced below the point of viability, and the now much less common seeds are more persistently targeted by seed predators.

Oh, look, they're all gone. Gosh, that disease was destructive.

We were probably primed to see this and think this way because of our extensive familiarity with chestnut blight, where the identical mistakes were made with the identical result: failure. Dozens of "uninfected!" survivors were found—never were they resistant. And the USDA advice to cut the trees down "in front of the blight"—because they were "all going to die"—insured that few trees ever had a chance to show any resistance they might have had. The chestnut story is even more complex than that, but we're talking about butternuts here.

Our Best Guess

Analysis of the disease dynamics and tree survival here suggest that we may indeed be looking at a phenomenon with a substantial component derived from the genetics of the trees. Because of that, we do collect seed only from those trees that show the ability to strongly heal cankers. However- the analogous situation with American chestnut has shown the rare survivors owe their lives to a complex situation of a little genetic resistance in the tree, and strains of the fungus that host a virus. We have NO evidence that such is NOT the case here.

Implications

A) We strongly recommend the widespread adoption of our rule:

“If It’s Not Dead, DON’T KILL IT.”

No matter how diseased it is. Removing diseased trees has proven useless in stopping or even slowing the spread.

In fact: a very sick tree is the best possible place to grow, find, or develop organisms antagonistic to the disease. If there’s lots of fungus growing, something will show up to eat it. Guaranteed.

B) To the extent that our trees may have some genetic resistance, it would be an excellent idea to get them established in other locations, where they can start crossing with other butternut trees.

C) Stop looking for trees with no infection. Look instead for strong trees with evidence of long-term infections, like the healed cankers pictured above.

D) At present, the population of strong survivors bearing seed at Badgersett is thriving, and expanding both by natural reproduction and our plantings. However, it is to be expected that much of the seed being produced here will be somewhat “inbred”. It’s quite likely that much of it will result from “mother–daughter” crosses, if you will forgive the peculiar genders. Since each butternut tree is both male and female, it’s a little different—pollen can likely go in both directions. Untangling is not easy or really necessary. Suffice it to say, some inbreeding is highly probable.

So far, we do not see any decrease in the basic vigor of the seedlings, but it could appear down the road. This is another reason that planting these trees elsewhere, where they may take pollen from unrelated butternuts, would be a good idea.

E) Butternut researchers and growers are invited to come, see, and discuss.

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