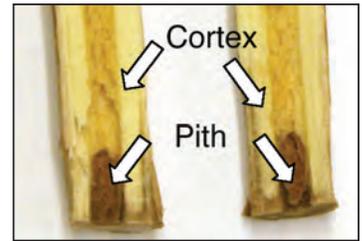


# Currant Cane Dieback in NY: Preliminary Data From the Hudson Valley Trial

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From 1891- 1913, a destructive blight of currants swept through the Hudson Valley and the Northeastern United States. During this time, Cornell scientists

**“Currant cane dieback is a devastating disease caused by *Botryosphaeria ribis* and results in young shoots, entire canes, and even whole bushes suddenly wilting and dying from spring to late summer/early fall. Although this disease was prevalent in the late 1800’s no reliable means of managing the disease were discovered then. Its re-appearance in the 21st century, means that no pesticides are specifically currently labeled for this disease in NY. In our studies sulfur sprays gave some control and were better than copper. Data from New Zealand indicates that Azoxystrobin fungicide may provide control. We are currently testing this strategy.”**

conducted extensive investigations into cause and management of this epidemic. Extension publications from the period estimated that between 25-50% of North American currants were lost to the disease, which was eventually dubbed currant cane dieback. Unfortunately, no effective means of managing the

disease was discovered at the time. As early as April 1917, *Ribes* quarantine and eradication legislation was beginning to be put into effect in an effort to manage white pine blister rust. In 1998, *Ribes* planting restrictions for New York were discussed and eventually relaxed in 2003. Prior to 2003, the disease was not likely to have been a problem due to planting restrictions on currants. In 2005-2006, a destructive blight of red currants re-emerged in the Hudson Valley. The blight was present at all of the largest currant production sites in the Hudson Valley. Soon after reports of the 2005 Hudson Valley epidemic were made public, we were informed that the disease was also present in Connecticut at one of the largest currant growers in the Northeast. One of the more disturbing observations from the ongoing epidemic was the fact that the dieback was particularly devastating to the white pine blister rust resistant cultivar ‘Titania’ to which most of the currant acreage in the Northeastern US is planted.

The currant cane dieback causes young shoots, entire canes, and even whole bushes to suddenly wilt and die

from spring to late summer/early fall (Figure 1A-C). The current season’s growth on canes often becomes infected at the shoot tips. Leaves of infected canes become chlorotic and completely wilt after final elongation and fruit set. Eventually, leaves may turn brown and necrotic as the young shoot tip begins to die (Figure 1D). The cortex and pith of young infected shoots/canes is often discolored (light tan) as the fungus kills the tissues (Figure 2A). In mature infected canes, the pith becomes completely necrotic (dark brown to black), which often completely decays (Figure 2B) leaving hollow canes that easily snap off when handled or during strong winds. The pathogen of the cane dieback epidemics of the 1890s and early 1900s was proven to be *Botryosphaeria ribis*. *Botryosphaeria* species are common pathogens of woody perennials causing limb and cane death in many bushberry, tree fruit, and woody vine crops. This fungus produces fairly diagnostic stroma (clusters of small, black, warty bumps) on mature canes from the previous season’s infections (Figure 3A-B). Prior to the extensive investigation on the disease in 1911, another fungus, *Nectria cinnabarina*, was thought to cause the disease. This fungus is weakly pathogenic on woody perennials and some sources consider this fungus to be a true cause of disease on currants. At the turn of the 20th century, *N. cinnabarina* was often found on currants already declining from dieback. The presence of coral-colored stromata on dead canes is diagnostic of *Nectria* (Figure 3C), and some sources consider its presence an indicator of *Botryosphaeria* infection.

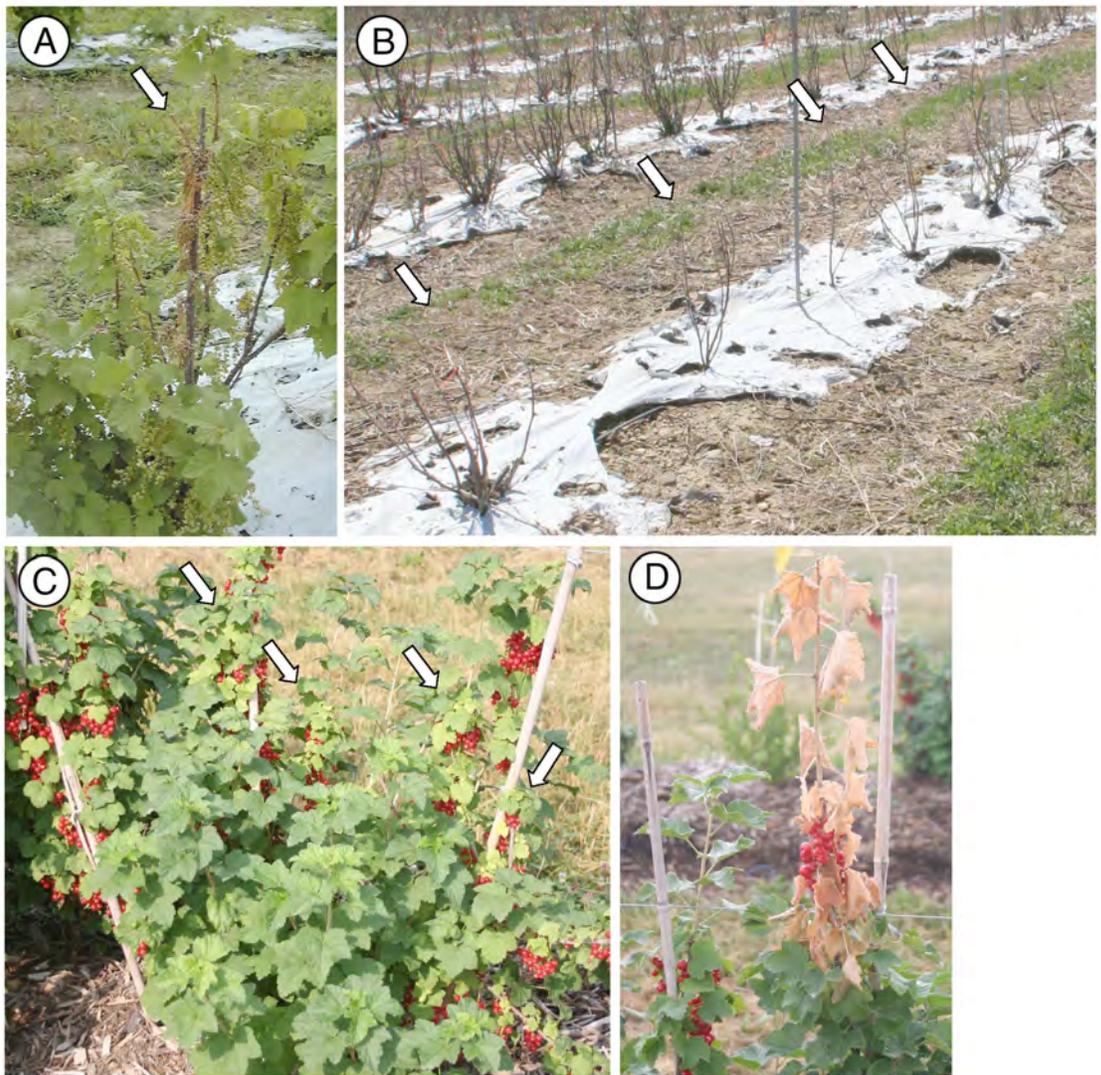
From 2005 to present, we have made extensive observations, collected numerous samples, isolated fungi from the margin of dieback cankers, and subjected them to genomic DNA sequence analysis to determine the identity of isolated fungi. Moreover, we attempted to inoculate healthy currants in the Geneva research planting with isolated fungi. In short, our observations of the ongoing epidemic and signs and symptoms of the disease were consistent with the reports from the early extension bulletins. Our fungal isolations were consistent in morphology, sporulation, and ribosomal DNA homology with the pathogen, *B. ribis*, identified in the older literature. We were also able to infect healthy plants by inoculating young buds with homogenized mycelium of *B. ribis*.

Unfortunately, no reliable means of managing the disease were discovered in all of the experimentation conducted at the turn of the 20<sup>th</sup> century. Moreover, being a new

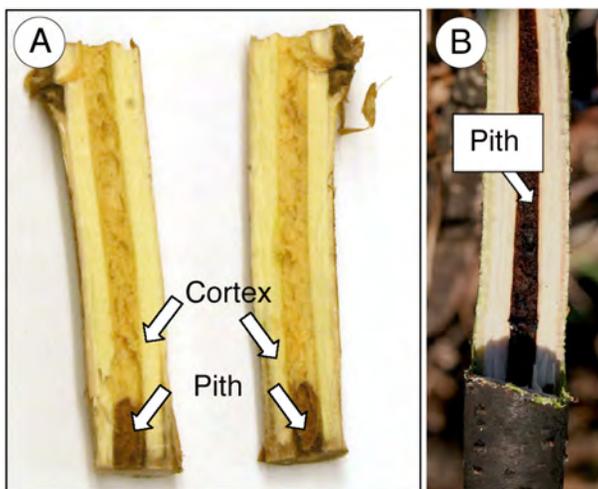
disease to the 21<sup>st</sup> century, little to no pesticides are specifically labeled for this disease in NY. Our goal was to reinvestigate the potential of cultural practices and widely labeled copper and sulfur pesticides to manage this disease at a commercial grower with natural infection. Specifically, we endeavored to: 1) determine differences in dieback resistance in a red, a pink, and a white currant cultivar; 2) evaluate effect of sanitation on the development of dead or dying shoots; and 3) ascertain whether or not extended season applications of copper and sulfur pesticides reduce the progression of dieback.

### Field Trial Design

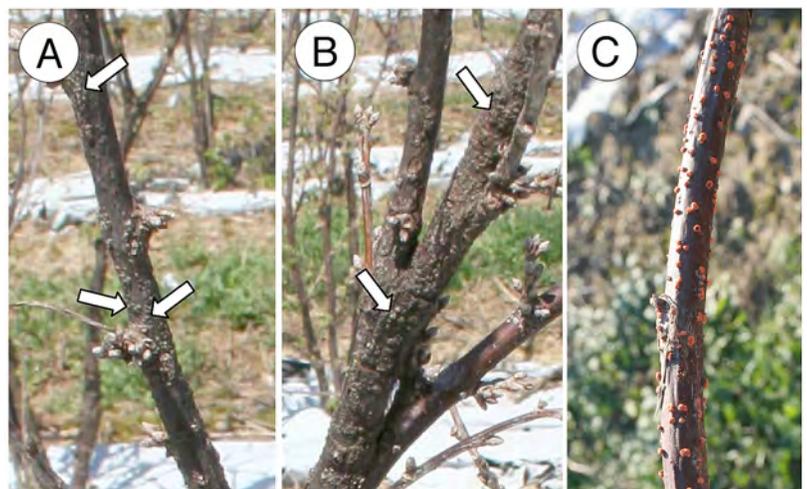
A field trial was setup to evaluate the effect of cultivar, fungicides and sanitation on the intensity of dieback and cane death. The experiment was first conducted in the summer of 2007 and is now being repeated in the summer of 2008.



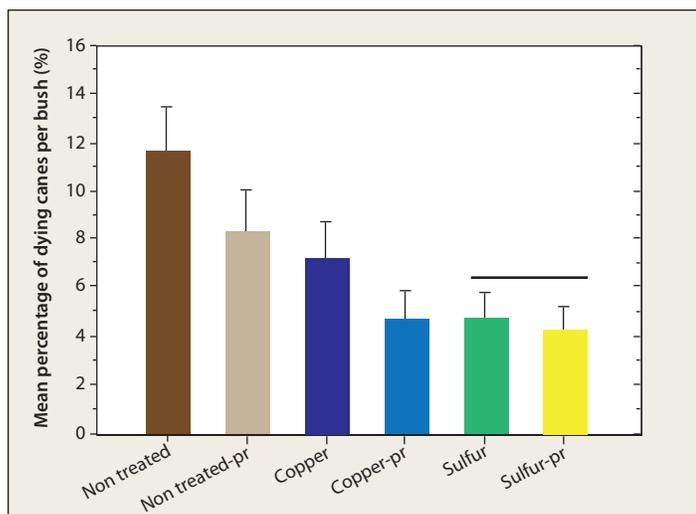
**Figure 1.** Red currants infected with currant cane dieback. A) Red currant from Germantown, NY with dead shoots (arrow) in late spring early summer. B) Several severely affected red currant bushes (arrows) at Germantown, NY before bud break in the spring. Most of the canes shown were completely dead, hollow, and brittle. C) A red currant from Geneva, NY with young shoots (arrows) becoming chlorotic. D) A young red currant shoot with distal shoot tip dieback.



**Figure 2.** Progression of currant cane dieback down an infected cane. A) Discoloration and tissue death in the advancing margin of an active dieback canker. B) Pith necrosis down the length of a currant cane resulting from primary and secondary infection of dead tissue.



**Figure 3.** Fungal stromata on dead currant canes in Germantown NY. A and B) *Botryosphaeria ribis* stromata on dead currant canes (arrows). The stromata depicted contained *B. ribis* conidia. C) Orange stromata of *Nectria cinnabarina* on a dead currant cane.



**Figure 4. Mean percentage of dying canes/shoots per bush resulting from currant cane dieback infections across all cultivars for fungicide programs in Germantown, NY. Values are means and standard errors for individual bushes across replicate 10 bush blocks.**

**The Field Site** The trial site, located in Germantown, NY, has a 7-acre hillside planting of mature 6-year-old red ('Rovada'), pink ('Pink Champaign'), and white ('Blanca') currants. The site has a naturally high level of currant cane dieback, and the Germantown area is where several of the 20<sup>th</sup> century observations about the disease were made. The planting is laid in rows of 100-150 bushes in blocks by cultivar with six rows for each cultivar. Additional blocks of 'Rovada', gooseberries, brambles, and cherries are planted nearby. Within the cultivar blocks, the six rows were divided into sub-plots of two rows randomly selected for different fungicide treatments. The two rows within the sub-plots were randomly designated as sub-sub plots for sanitation treatments.

**Fungicide programs** A copper, sulfur, and untreated fungicide program was implemented within the cultivar main plots. Copper hydroxide (Kocide DF: 40% metallic copper equivalent) and sulfur (Sulfur 6L Microflo) was applied to subplot treatments at 10 lbs. /A and 2.5 gal. /A, respectively. Fungicide applications were made at dormant to bud break, and again at full green tip to leaf burst. Applications were made using a small tractor-pulled 30-gallon vegetable sprayer with booms angled and nozzles calibrated for spraying small fruit crops. The 'untreated' treatment was sprayed with water at both timings.

**Sanitation Treatments** Within sub-plot fungicide treatments, sub-sub-plots were either sanitized or left non-sanitized. Sanitization consisted of pruning out the dead shoots and dieback infected (fruiting with fungal stromata) shoots with loppers. Such shoots were pruned to the crown and removed from the planting. In the non-sanitized sub-sub-plots, the dead shoots and shoots with dieback inoculum were left intact.

**Dieback Assessment Strategy** Ten bushes each were selected from the beginning, the middle, and the end of the

row for a total of 30 bushes rated per sub-sub-plot. Each of these 10 bush 'micro plots' were considered replicates in the experimental design. On each bush, the number of total shoots per bush was counted along with the number of dying shoots, and the number of dead shoots or canes. Currant cane dieback disease intensity was expressed as the percentage of dead or dying shoots out of the total number of shoots. The bushes were rated for dieback intensity during the 'Rovada' harvest on 16 July 2008.

## Results and Discussion

Prior to experimentation, the level of disease appeared to be evenly distributed within cultivar blocks. At 'Rovada' harvest, currant cane dieback symptoms and *Botryosphaeria* stromata were observed in all cultivars and treatment plots. Across all cultivars and treatments the percent of dying and dead canes per bush ranged from 0.0 to 79.6% and 0.7 to 17.0%, respectively.

Dying shoots or canes most likely represent within-season infections and as such would be the most reasonable indicator of treatment success. Within the design, pruning, cultivar and all interaction terms among pruning, cultivar, and fungicide treatment were not significant ( $P > 0.05$ ). Fungicide treatments of copper hydroxide and sulfur were effective, reducing the percentage of dying canes (in season infections) compared to untreated bushes (Figure 4). Of the two fungicides, sulfur applications reduced the percentage of dying shoots to a greater extent than copper hydroxide applications. Moreover, the level of control provided by sulfur across all variables was significantly greater than the control provided by copper hydroxide. However, it is uncertain whether a 3% reduction in shoot infection would translate into a noticeable improvement in planting life.

The general consensus of extension publications and fruit pathology literature is that *Botryosphaeria* canker diseases aren't really manageable using chemical control. Certainly, it stands to reason that the fungus is protected from fungicides by the woody tissues in which it resides. However, most pest management guidelines including Cornell's 2008 *Pest Management Guidelines for Berry Crops* affirm that applications of Bordeaux mixture, lime sulfur, and fixed copper to dormant bushes in the early spring to late fall may prevent initial infection and reduce overwintering inoculum of fungal shoot and cane diseases. It is known that *Botryosphaeria* fungi sporulate most heavily in the spring. Hence, the efficacy of our protective applications of copper hydroxide and sulfur is not surprising as the fungicides would be present on young currant buds where initial infections are known to take place.

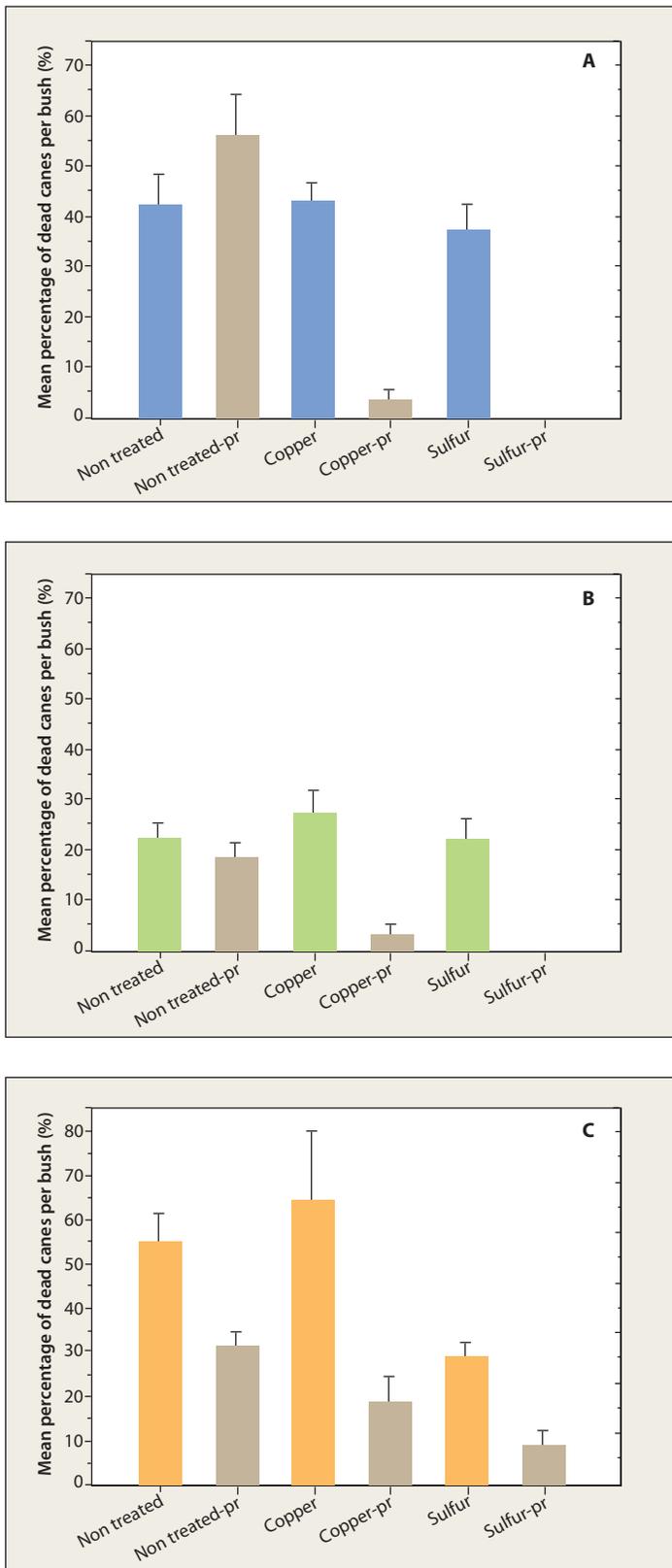
It has also come to our attention that currant growers in New Zealand apply a fungicide called Amistar<sup>®</sup> (Syngenta Crop Protection) at 400ml/ha immediately after harvest for control of this disease. Amistar<sup>®</sup> applications are reported to be effective for reducing the disease and also promote green leaf retention and, in turn, increased bud size. In the

US we don't have an Amistar® label for currants. However, the US formulation of Abound® (Syngenta Crop Protection) is analogous to the NZ Amistar® in formulation and

concentration of the active ingredient Azoxystrobin. In fact, this product is actually labeled in NY (EPA Reg. 100-1098, NYSDEC Acceptance 9/16/04) for use on Botryosphaeria canker diseases of bushberries (including currants and gooseberries) at a rate of 6.2 to 15.4floz per acre. Abound may be applied the day of harvest and has no other restrictions that would prevent immediate post harvest application. Applications of Abound would have the advantage of being less phytotoxic to the crop and could be used to protect the young shoot growth from infection for much longer periods than copper or sulfur. This season, we have a large trial in Connecticut investigating the potential of currant cane dieback management using QoI (Strobilurin) fungicides.

Dead shoots and canes most likely represent infections that occurred over the last few seasons including the season in which the trial occurred. In this case, in-season fungicide applications would have little impact on the percentage of dead canes per bush. Similarly, the pruning treatments are more likely to reduce inoculum pressure for the following season. Indeed, they will reduce the number of dead shoots within a season, but because of this, counting dead shoots isn't a good indicator of pruning treatment success. By comparison, the cultivar treatments were in place for many seasons and dead shoots would be a valid indicator of cultivar resistance to dieback. Within the design, pruning, cultivar, and fungicide were significant ( $P < 0.05$ ) factors influencing the percentage of dead shoots per bush. There were also significant ( $P < 0.05$ ) interactions between cultivar  $\times$  fungicide and pruning  $\times$  fungicide meaning that fungicides affected the level of shoot death differently for different cultivars, and that pruning was more effective for different fungicide treatments. Due to significant interactions between factors we must present treatment effects (pruning and fungicide) separately for each cultivar. Across all treatments, the cultivar 'Blanca' had a mean percentage of  $(31.8 \pm 3.5\%)$  dead shoots per bush. Of the treatments, sulfur applications with pruning and copper hydroxide applications with pruning were improved over the non-fungicide treatments and fungicide treatments alone (Figure 5A). Across all treatments, the cultivar 'Pink Champaign' had the lowest mean percentage  $(15.3 \pm 1.8\%)$  of dead shoots per bush. Similar to 'Blanca', sulfur applications with pruning and copper hydroxide applications with pruning were improved over the non-fungicide treatment and fungicide treatments alone (Figure 5B). Across all treatments, the cultivar 'Rovada' had the highest mean percentage  $(34.1 \pm 3.7\%)$  of dead shoots per bush. (Figure 5C).

Aside from the fact that dead shoots were not a good assessment of fungicide performance, there appears to be an overriding trend that sulfur applications are helpful in managing disease. This is especially supported by our shoot infection data. In regards to cultivar, there do appear to be differences in the levels of resistance to this pathogen.



**Figure 5.** Mean percentage of dead canes/shoots per bush resulting from currant cane dieback infections for fungicide and pruning programs in Germantown, NY. Shoot death data is present for cultivars 'Blanca' (A), 'Pink Champaign' (B), and 'Rovada' (C) Values are means and standard errors for individual bushes across replicate 10 bush blocks.

However, resistance to this pathogen is likely to be due to anatomical or chemical features of the variety as opposed to gene for gene interactions. Moreover, it is hard to ascertain if these differences are durable without additional years of data. In regards to pruning, we are unable to see potential effects of pruning in the first year. Because of this, we have chosen to repeat the treatments in the same blocks for a second season. Although we will sacrifice our ability to truly replicate the experiment, we may be able to shed light on the effects of pruning. The disease is a slow progressing disease, and as such, may take several seasons to completely kill the large canes. Because of this phenomenon, progress towards managing currant cane dieback in NY may not be accomplished in a single season.

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