Mortality of eastern white pine (Pinus strobus L.) in association with a novel scale insect-pathogen complex in Virginia and West Virginia

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\textbf{ABSTRACT}

Concerns about the health of eastern white pine (Pinus strobus L.) exist throughout its range in the eastern United States due to a variety of causal factors. In the central and southern Appalachian Mountains, a novel and poorly understood dieback phenomenon has been attributed primarily to a scale insect, Matsucoccus macrocicatrices Richards, and a putatively weak fungal pathogen, Caliciopsis pinea Peck. While the nature of this scale-pathogen complex and associated stem and branch cankers is still being explored, quantitative information on the extent of damage to the white pine resource has been lacking. Monitoring plots were established in Virginia and West Virginia beginning in 2012 to assess changes in live and dead volume of white pine. Most sites consisted of uneven aged, mixed oak-pine stands. No significant changes in stand volume were evident, likely because no large trees within the plots died during the study period. Mortality in sapling and pole-sized trees appeared elevated, however. Density dependent mortality associated with natural stand self-thinning was distinguished from density independent mortality associated with the scale-pathogen complex by establishing a baseline mortality for white pine age cohorts. Baseline mortality for each 2-cm diameter class was estimated to be approximately 12\% based on the aggregate study site data, and 13–14\% based on US Forest Service Forest Inventory and Analysis (FIA) data. Observed mortality within plots was then compared to baseline mortality using chi-square analysis. During the initial measurement years, observed mortality was significantly greater than baseline mortality for only the 4–6 cm diameter class. After 5 years of monitoring, observed mortality was significantly greater than baseline for five diameter classes, including 4–6, 6–8, 8–10, 14–16, and 24–26 cm. The two smallest diameter classes of these five had observed mortality over 30\%, on average, while the other three classes averaged over 20\% mortality. If these trends continue over time, white pine abundance or sustainability could eventually be compromised as mature trees gradually die and fewer cohorts of young trees are available to replace them.

1. Introduction

Eastern white pine (Pinus strobus L.) was a dominant species in many of the old growth forests that once covered the eastern U.S. (Gould, 1985). Today, most old growth white pine is gone, but the species continues to play an important role in many local economies (Chappelle, 1992) and contributes as a significant ecological resource in natural stands (Tlutsy, 1992). It is an important associate of oak-hickory and oak-pine forest types, which dominate large parts of the Appalachian landscape (Bailey, 2004). It also contributes as an important conifer component of eastern hemlock stands that form critical habitat along Appalachian riparian zones, habitat which is continually deteriorating and under threat from the non-native, invasive hemlock woolly adelgid, Adelges tsugae Annand (Man, 2016). White pine is relatively fast growing and has easily workable wood that makes it ideal for mill use. It has moderate strength and, in fact, was often used for ship masts during colonial times (Wendel and Smith, 1990). Many other uses include construction material, toys, crates, boxes, furniture and specialty items (Schumann, 1985). It is also an important species for Christmas trees, landscapes, and site reclamation, growing fairly
well on poor, sandy soils (Wendel and Smith, 1990). Unfortunately, white pine’s potential as a major resource in the forestry economy has been limited by hardwood competition, wildlife herbivory and a few important pests, most notably white pine weevil, *Pissodes strobi* (Peck), and white pine blister rust, *Cronartium ribicola* (J.C. Fisch. ex Rabenh) (Ostry et al., 2010; Jones, 1992).

Recently, a previously undescribed and damaging scale insect-pathogen complex in the central and southern Appalachians of the eastern U.S. has been noted on white pine in natural stands and plantations (Schulz et al., this issue-a; Mech et al., 2013; Asaro, 2011; Cram et al., 2009). Eastern white pine trees in mixed and pure stands throughout this region (including the mountainous parts of Virginia, West Virginia, Tennessee, North Carolina, South Carolina and Georgia) were observed exhibiting unusual symptoms of dieback and mortality as early as 2006 (Schulz et al., this issue-a; Mech et al., 2013; Asaro, 2011). Symptoms include needle browning, branch flagging, crown thinning, crown dieback, resinosis along the main stem and at branch crotches, and canker development on the main stem and branches of all size classes (Figs. 1 and 2). Cankering and other symptoms are particularly evident on smaller trees in the understory, but all size classes up to dominant and codominant trees can be impacted to varying degrees. Upon closer inspection, scale insects are often recovered from deep within cankers (Mech et al., 2013; Asaro, 2011).

Additional investigations revealed that the Canadian pine bast scale, *Matsucoccus macrocicatrices* Richards, is associated with these cankers (Schulz et al., this issue-a; Whitney et al., this issue; Mech et al., 2013). This species is believed to be native to the northeastern U.S. and eastern Canada, but has never been previously reported south of Massachusetts (Mech et al., 2013; Kosztarab, 1996). In addition, *M. macrocicatrices* has only been described as living in a symbiotic association with fungal mats of *Septobasidium pinicola* Snell, a fungus that is known to be an obligate parasite on a variety of sapsucking insects (Mech et al., 2013; Henk and Vilgalys, 2007; Watson et al., 1960; Couch, 1938). Yet in the central and southern Appalachian sites where *M. macrocicatrices* was present, *S. pinicola* was rarely found. Instead, a putatively weak fungal pathogen known as *Caliciopsis pinea* Peck was discovered in association with most cankers and *M. macrocicatrices* infestations (Schulz et al., this issue-a; Whitney et al., this issue; Mech et al., 2013; Cram et al., 2009). *Caliciopsis pinea* produces small (2–4 mm), black fruiting structures known as ascocarps, which are often visible on or near the surface of cankers. Other non-aggressive, saprophytic fungi have been less consistently isolated from cankers (Schulz et al., this issue-a; Cram et al., 2009).

To date, there is no evidence of any association between affected sites and significant bark beetle activity (*Ips or Dendroctonus* species), white pine blister rust (*Cronartium ribicola* J.C. Fisch. ex. Rabenh), major root disease pathogens such as *Armillaria* sp. or *Heterobasidion*
sp., or other usual suspects associated with white pine mortality (Mech et al., 2013; Asaro, 2011; Cram et al., 2009). The pattern of decline often starts with a single branch or twig browning up in the spring on an otherwise green tree. Over several years, this process can complete browning of multiple branches, then the entire tree. This pattern of decline is inconsistent with those familiar pests and pathogens mentioned above. Once needle browning occurs, cankers on the main branches and bole are typically evident on smaller trees in the understory (Fig. 1a and b). Cankers on the branches and boles of large, codominant trees are harder to detect, and normally patches of heavy resinosis are most visible, along with needle browning (Asaro, 2011; Lombard, 2003).

The association between M. macrocicatrices, C. pinea, other saprophytic fungi and cankers is herein referred to as a ‘complex’. The exact mechanism for canker development and subsequent dieback and decline is poorly understood (Schulz et al., this issue-a; Whitney et al., this issue). It is also unclear whether M. macrocicatrices colonizes first, creating infection courts for C. pinea, or if C. pinea infection results in cankers and bark cracks that enable easy penetration and access to the cambium by the scale. Mech et al. (2013) suggest that the scale may be driving the decline process, in concert with unknown abiotic factors (Schulz et al., this issue-b). In contrast, Munch et al. (2015) suggest C. pinea is the primary agent responsible for emerging disease among eastern white pine in the northeastern U.S. and do not make any mention of M. macrocicatrices despite its putative endemism to that region.

Many more studies are necessary to understand this biotic complex as well as abiotic factors and their respective influence on the observed decline of eastern white pine. In addition, the extent of damage and mortality in white pine stands has, to date, been poorly documented and quantified, with the long term impact to the resource largely unknown. Using data from monitoring plots established from 2012 to 2015 among a variety of stands in Virginia and West Virginia where this scale/pathogen complex was first observed, we provide herein a summary and analysis of 3-5 year trends in white pine tree mortality across a spectrum of age cohorts.

2. Methods

2.1. Site selection and plot layout

From 2012 to 2015 we established thirty-six monitoring plots (0.04 ha) in clusters of 4 plots per site for a total of 9 sites across 5 western Virginia counties and 4 eastern West Virginia counties. Most stands were located on relatively flat terrain, so aspect and slope were not considered an important variable. Elevation information is provided in Table 1. Overall stand composition as well as white pine age class distribution across sites were variable, with the majority of stands in oak-pine forest type with an uneven-age distribution, plus two nearly pure stands of plantation white pine (Table 2). At each oak-pine site chosen, M. macrocicatrices scale, C. pinea fungus, and cankers were present and impacting stands in varying degrees. Therefore, there were no true control sites, although it was clear that some oak-pine stands were much more heavily impacted than others based on the amount of cankerling, resinosis, and branch flagging on the white pine regeneration. The two plantation sites appeared to have a very low incidence of scale or disease symptoms. Plots were laid out systematically so that their centers were spaced a minimum of 60 m apart and well represented the stand variation across the site. The emphasis of the study is to monitor change in white pine condition and to measure long-term loss of white pine volume; therefore, plots were placed to insure that some measurable white pine basal area was present, although white pine was ubiquitous throughout a majority of the selected sites. Although detailed site histories are not known, generally white pine growing in hardwood mixtures originates from colonization of old-fields, gradual succession of older pine plantations into hardwood forest following a partial harvest, or some other major disturbance such as a fire or wind storm. In most cases, stands are rarely uniform but rather patchy in terms of the distribution of white pine saplings, intermediate and codominant trees, and emergent canopy dominants. The latter are uncommon in the southern Appalachians except within very rare old growth stands.

2.2. Data collection

Branch clippings with cankers were collected from each site between 2008 and 2012 and sent to the USFS Southern Research Station in Athens, Georgia to confirm that M. macrocicatrices and C. pinea were present (Schulz et al., this issue-a; Mech et al., 2013; Cram et al., 2009). In each plot the diameter at breast height (dbh, 1.36 m) of each white pine tree that was ≥2.54 cm (1 in) was measured annually. Tree heights were measured during most years with a laser clinometer. The volume of each tree was calculated using the Honer equation, which accounts for stem taper (Honer, 1967). Standing dead trees were

### Table 1

**Site locations and stand attributes.**

<table>
<thead>
<tr>
<th>Site name</th>
<th>County/State</th>
<th>Latitude</th>
<th>Longitude</th>
<th>Elevation</th>
<th>Stand type</th>
</tr>
</thead>
<tbody>
<tr>
<td>Falling Springs</td>
<td>Alleghany/VA</td>
<td>37.8779</td>
<td>–79.9877</td>
<td>451 m (1488 ft)</td>
<td>Natural/oak-pine</td>
</tr>
<tr>
<td>Headwaters</td>
<td>Highland/VA</td>
<td>38.3081</td>
<td>–79.4242</td>
<td>634 m (2092 ft)</td>
<td>Natural/oak-pine</td>
</tr>
<tr>
<td>Winding Ridge</td>
<td>Bath/VA</td>
<td>38.0333</td>
<td>–79.8558</td>
<td>582 m (1922 ft)</td>
<td>Natural/oak-pine</td>
</tr>
<tr>
<td>Deerfield</td>
<td>Augusta/VA</td>
<td>38.2154</td>
<td>–79.3285</td>
<td>630 m (2079 ft)</td>
<td>Natural/oak-pine</td>
</tr>
<tr>
<td>Matthews State Forest</td>
<td>Grayson/VA</td>
<td>36.6268</td>
<td>–80.9553</td>
<td>776 m (2561 ft)</td>
<td>Pine plantation</td>
</tr>
<tr>
<td>Sweet Springs</td>
<td>Monroe/WV</td>
<td>37.6181</td>
<td>–80.2419</td>
<td>722 m (2382 ft)</td>
<td>Natural/oak-pine</td>
</tr>
<tr>
<td>Lost River State Park</td>
<td>Hardy/WV</td>
<td>38.8956</td>
<td>–78.9223</td>
<td>641 m (2116 ft)</td>
<td>Natural/oak-pine</td>
</tr>
<tr>
<td>Watoga State Park</td>
<td>Pocahontas/WV</td>
<td>38.1111</td>
<td>–80.1301</td>
<td>848 m (2798 ft)</td>
<td>Natural/oak-pine</td>
</tr>
<tr>
<td>Brushy Fork Lake State Park</td>
<td>Pendleton/WV</td>
<td>38.4644</td>
<td>–79.3212</td>
<td>626 m (2066 ft)</td>
<td>Pine plantation</td>
</tr>
</tbody>
</table>

### Table 2

**Additional site attributes.**

<table>
<thead>
<tr>
<th>Site name</th>
<th>Total stand basal area m²/ha (ft²/acre)</th>
<th>% basal area white pine</th>
<th>% basal area all oak sp./other sp.</th>
<th>Year plots established/last year data collected</th>
</tr>
</thead>
<tbody>
<tr>
<td>Falling Springs</td>
<td>Not Available</td>
<td>Not Available</td>
<td>Not Available</td>
<td>2012/2014</td>
</tr>
<tr>
<td>Headwaters</td>
<td>24.6 (107)</td>
<td>35%</td>
<td>57%/8%</td>
<td>2012/2016</td>
</tr>
<tr>
<td>Winding Ridge</td>
<td>32.4 (141)</td>
<td>57%</td>
<td>34%/9%</td>
<td>2012/2016</td>
</tr>
<tr>
<td>Deerfield</td>
<td>34.4 (150)</td>
<td>39%</td>
<td>48%/13%</td>
<td>2015/2017</td>
</tr>
<tr>
<td>Matthews State Forest</td>
<td>53.5 (233)</td>
<td>89%</td>
<td>11%/0%</td>
<td>2015/2017</td>
</tr>
<tr>
<td>Sweet Springs</td>
<td>39.3 (171)</td>
<td>11%</td>
<td>59%/30%</td>
<td>2012/2016</td>
</tr>
<tr>
<td>Lost River State Park</td>
<td>31.5 (137)</td>
<td>31%</td>
<td>60%/9%</td>
<td>2012/2016</td>
</tr>
<tr>
<td>Watoga State Park</td>
<td>27.6 (120)</td>
<td>11%</td>
<td>77%/12%</td>
<td>2012/2016</td>
</tr>
<tr>
<td>Brushy Fork Lake State Park</td>
<td>33.9 (148)</td>
<td>97%</td>
<td>0%/3%</td>
<td>2012/2016</td>
</tr>
</tbody>
</table>
counted and measured each year until they fell over or otherwise deteriorated. To be conservative, trees were not counted as dead unless there were no visible green needles remaining. Data were collected from the Falling Springs, VA site from 2012 to 2014 only; the site was abandoned in 2015 due to access issues and vandalism on the plots. In 2016, in all the VA plots, the diameter of all non-white pine tree species ≥ 2.54 cm dbh were measured to determine the relative basal areas of all major tree species within each site (Table 2). Similarly, in 2017 basal areas of non-white pine tree species were calculated using a 10-factor prism among all the WV plots (Table 2).

In November 2017, the presence of cankers and C. pinea fruiting bodies was documented in the lower 1.8 m of each tree (live or standing dead) in all plots as follows: the number of nodes (branch whorls) along the main stem of each tree up to 1.8 m were counted, and any stem cankers or fruiting bodies associated with or proximate to each node were noted. Although mortality of white pine as it relates to the cankers has not been thoroughly studied, it is presumed that stem girdling due to the gradual buildup of cankers is what leads to tree death, particularly in small diameter saplings (Schulz et al., this issue-a,b; Whitney et al., this issue).

2.3. Initial data analysis

The number of live and dead white pine in each plot were summed and combined for each site by 2 cm diameter classes to assess the amount and pattern of mortality across the diameter distribution. Since it was apparent that most of the dead trees were in the smallest diameter classes and that no large (≥ 25 cm), dominant or codominant trees had died within the plots thus far, few significant changes were anticipated in the volume of live versus dead white pine over the observed time span. Repeated measures ANOVA of the average percent dead volume across several of the medium to large diameter classes confirmed that this was the case at all sites (p > .1). Therefore, volume data will not be presented at this time but will be utilized at a later date should larger trees begin to die in the plots. Mortality of large white pine trees attributed to this scale-pathogen complex has been widely observed across the region in natural stands as well as plantations (Asaro et al., 2011; Fig. 2). However, a limited sample size of dominant/codominant trees within the plots failed to capture this scattered mortality. Since a majority of the observed mortality occurred within the smaller diameter classes in the understory, the next challenge was to distinguish mortality due to natural stand-self thinning (i.e. baseline mortality) from density-independent mortality.

2.4. Estimating baseline mortality

Stand self-thinning is associated with aging in early and mid-successional stages, so this type of mortality is density dependent and expected. With natural self-thinning, mortality is sustainable as long as basal area is increasing among the remaining individuals (Linz et al., 2016; Oliver and Larson, 1990). However, if basal area decline occurs in concert with mortality that exceeds population growth, this could mean density-independent factors are at work (Linz et al., 2016). Density independent factors may include such things as climate change, fire, tree harvest, and damage from pests or pathogens. Efforts to accurately quantify density-independent mortality among any tree species can be challenging (Linz et al., 2016).

One method used by Manion and Griffin (2001) involves calculation of a baseline mortality value for each species across the diameter distribution within multiple stands. Observed mortality that deviates significantly from baseline mortality along any part of the diameter distribution could indicate an unstable or unsustainable stand structure. Comparing observed with baseline mortalities, therefore, provides a quantitative, ecologically-based method for determining whether an insect or disease outbreak is endangering stand sustainability or simply acting as a natural thinning agent. This analytical approach, proposed as one way to objectively measure ‘forest health’, has been reviewed extensively by Zhang et al. (2011) and Teale and Castello (2011) and is used herein.

For many uneven-aged stands, as a cohort of trees grow, the natural progression from many small stems to fewer large stems can be represented graphically by a negative exponential function, or ‘reverse J’, where diameter class on the X-axis is graphed against the number of trees per class on the Y-axis. Meyer (1952) described the ‘q ratio’ as the ratio of tree density in one diameter class to the density of trees in the next larger class. Therefore, the expected surviving fraction of trees in the next larger diameter class is 1/q, and baseline mortality can be described as 1 – 1/q or percent mortality per unit of diameter growth. If an uneven-aged stand approaches a balanced ‘reverse J’ diameter distribution, the negative exponential function that describes this relationship becomes linear when plotted on a log-linear axis. Teale and Castello (2011) point out that while other, more complex mathematical functions are more effective at describing diameter distributions at the stand level, the negative exponential function can have high predictive ability when multiple stands, in aggregate, are included in the analysis. In addition, other more complex functions, while more predictive at the stand level, can produce non-constant baseline mortality or negative mortality, which can make analysis of large data sets unnecessarily complicated and difficult to interpret.

Baseline mortality, therefore, is defined by the slope of the log-linear model which represents how many stems of a given size class must die in order for the population to maintain its current diameter distribution (Teale and Castello, 2011). Manion and Griffin (2001) calculated baseline mortality (BM) using the following formula:

\[ BM = 1 - e^{-\beta \Delta D} \]

where \( \beta \) is the model parameter (slope) of the negative exponential function (using a natural log-linear model) and \( \Delta D \) is the diameter class interval. A diameter class interval of 2 cm was used in order to detect subtle shifts in diameter class distribution and to insure the decay rate and growth rate of white pine were similar enough that tree loss and gain from each diameter class were adequately monitored. In order to use this method appropriately, Teale and Castello (2011) suggested that the following assumptions be made: (1) the method is generally only applicable at the landscape scale. It can be used at the stand level if sampling of the stand is at the appropriate level of intensity to capture its diversity; (2) the method used to quantify observed mortality assumes that dead trees remain identifiable to species for about the same time that it takes for the living trees to grow into the next diameter class. Therefore, the decay rate and the growth rate must be taken into account when determining the optimum width of the diameter classes; (3) observed mortality includes all mortality regardless of cause; and (4) the management and disease/pest history of the forest are known, and the silvics and ecology of the species comprising that forest are understood.

Seven out of nine sites contained mixed species (oak-pine) stands, and it was assumed that totaling the number of white pine trees within each diameter class across all seven sites would produce a reasonable approximation to a balanced, uneven-aged distribution associated with the negative exponential function. As stated above, this assumption was made to simplify the analysis and interpretation of the data and reduce the likelihood of drawing spurious conclusions by over-analyzing diameter distributions that are likely to be highly variable across sites. It should be noted, however, that at the stand level, a rotated-sigmoid or other more complex function would probably be more appropriate for irregular, mixed-species stands such as these (Zhang et al., 2001, 2011). We therefore included seven oak-pine sites in the baseline mortality analysis, excluding the two sites that were even-aged pine plantations (Table 1). Even-aged, managed stands have a normal or bimodal diameter distribution and do not fit the reverse-J diameter distribution at any point in time, thus would detract from producing a robust log-linear model for estimating baseline mortality of white pine in the
uneven-aged, oak-pine forest type. In addition, there is an expectation of extensive density-dependent mortality among understory, small diameter class white pine under a dense, closed canopy, as seen in high basal area white pine plantations (Table 1). Therefore, teasing out density independent mortality factors from natural self-thinning in plantations is particularly difficult, especially without more extensive site replication. Using the first and last year of measurements, respectively, for each of the 7 uneven-aged (oak-pine) sites, we estimated, for all sites collectively, a baseline mortality using the formula above, as well as the % mortality observed within each 2 cm diameter class.

2.5. Estimating baseline mortality with FIA data

To insure that the baseline mortality estimate in this study was sound, white pine data for the period 2010–2015 was gathered from the U.S. Forest Service Forest Inventory and Analysis (FIA) plot network using their online data analysis tool Evalidator (https://apps.fs.usda.gov/Evalidator/evaluator.jsp). The FIA plot network spans the entire lower 48 states of the U.S. with approximately one forested plot every 2419 ha (6000 ac). This sampling intensity is sufficient to estimate forest composition, tree species abundance, growth and mortality, among many other variables, at the state and sub-state level. An Evalidator query for white pine examined the number of live trees by diameter class white pine under a dense, closed canopy, as seen in high basal area white pine plantations (Table 1). Therefore, teasing out density independent mortality factors from natural self-thinning in plantations is particularly difficult, especially without more extensive site replication. Using the first and last year of measurements, respectively, for each of the 7 uneven-aged (oak-pine) sites, we estimated, for all sites collectively, a baseline mortality using the formula above, as well as the % mortality observed within each 2 cm diameter class.

The number of standing dead trees by diameter class was also available for query, but only for diameter classes of 5 in (12.7 cm) and above. These numbers allowed for estimation of observed % mortality, which was then compared with baseline mortality. For live and dead trees above 21 in (53.3 cm), diameter classes in Evalidator are lumped which was then compared with baseline mortality. For live and dead trees above 21 in (53.3 cm), diameter classes in Evalidator are lumped.

To calculate baseline mortality as above. Since tree count data in Evalidator was presented by 2 in. (5.1 cm) diameter class, a quotient of 2.54 was used to divide into the baseline mortality equation so that it expressed mortality rate in terms of 2 cm classes, not 5 cm:

\[
BM = \frac{1 - e^{-\beta \Delta D}}{2.54}
\]

The number of standing dead trees by diameter class was also available for query, but only for diameter classes of 5 in (12.7 cm) and above. These numbers allowed for estimation of observed % mortality, which was then compared with baseline mortality. For live and dead trees above 21 in (53.3 cm), diameter classes in Evalidator are lumped together as 21–29 in (54.3–73.7 cm) and 29+ in (73.7+ cm) categories, so these could not be included in the calculation of baseline mortality, which requires the use of equal diameter class intervals.

2.6. Statistical analysis

Chi-square analysis was used to detect any significant changes in observed mortality relative to baseline mortality across the diameter distribution for the earliest and latest measurement periods for all uneven-aged (non-plantation) stands (7 sites, as described above). Because observed versus baseline mortality is compared over multiple diameter classes, the Bonferroni correction was used to adjust for the likelihood of falsely rejecting the null hypothesis (Type 1 error) (Zhang et al., 2011). The correction involves dividing the desired alpha level by the number of comparisons to be made. Therefore, the desired \( \alpha \) level of 0.05 was divided by 10 (the number of diameter distributions which had observed mortality above 0), resulting in \( \alpha = 0.005 \). Subsequently, a critical chi-square value of 7.88 was obtained from a chi-square distribution table, based on \( \alpha = 0.005 \) and one degree of freedom (Sokal and Rohlf, 1995). A similar approach was used to examine the county-level FIA data, where a critical chi-square value of 7.47 was used for 8 diameter class comparisons of baseline versus observed mortality. All regression and chi-square analyses were carried out in Excel® 2013.

Table 3

Incidence (%) of all trees with stem cankers or C. pinea fruiting bodies in the lower 1.8 m of the main stem. Numbers represent all trees on each of 4 plots per site. An * indicates a plantation site.

<table>
<thead>
<tr>
<th>Site</th>
<th>No. trees sampled</th>
<th>% dead trees</th>
<th>% trees with cankers</th>
<th>% trees with C. pinea fruiting bodies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Falling Springs</td>
<td>82</td>
<td>35%</td>
<td>No Data</td>
<td>No data</td>
</tr>
<tr>
<td>Headwaters</td>
<td>76</td>
<td>42%</td>
<td>64%</td>
<td>64%</td>
</tr>
<tr>
<td>Winding Ridge</td>
<td>91</td>
<td>7%</td>
<td>65%</td>
<td>47%</td>
</tr>
<tr>
<td>Deerfield</td>
<td>55</td>
<td>9%</td>
<td>62%</td>
<td>40%</td>
</tr>
<tr>
<td>Matthews State Forest</td>
<td>117</td>
<td>25%</td>
<td>6%</td>
<td>0%</td>
</tr>
<tr>
<td>Sweet Springs</td>
<td>68</td>
<td>15%</td>
<td>84%</td>
<td>66%</td>
</tr>
<tr>
<td>Lost River State Park</td>
<td>101</td>
<td>9%</td>
<td>28%</td>
<td>32%</td>
</tr>
<tr>
<td>Watoga State Park</td>
<td>63</td>
<td>16%</td>
<td>44%</td>
<td>57%</td>
</tr>
<tr>
<td>Brushy Fork Lake State Park</td>
<td>71</td>
<td>20%</td>
<td>0%</td>
<td>0%</td>
</tr>
</tbody>
</table>

3. Results and discussion

3.1. Documentation of Matsucoccus macrocicatrices and calciopsis pinea

The presence of the M. macrocicatrices scale and C. pinea fungus was confirmed at all oak-pine field sites and documented in Mech et al. (2013) and Schulz et al. (this issue-a,b). The percentage of all trees afflicted with either cankers (28–84%) or Calciopsis pinea fruiting bodies (32–66%) in the lower 1.8 m of the main stem was variable among the seven oak-pine sites (Table 3). The two plantation sites (Matthews, Brushy Fork) exhibited virtually no disease symptoms, suggesting that mortality associated with those sites is primarily density dependent. While these data strongly suggest that the scale/pathogen complex is a significant disturbance at the oak-pine sites, the correlation between overall mortality at the sites and proportion of trees with cankers or C. pinea symptoms is poor (\( r < 0.1 \)). This can be explained by the fact that many living trees also have cankers and C. pinea fruiting bodies and even small saplings can live with symptoms for several or more years before dying. This analysis was also conservative by not separating out trees in good health from those that were in poor health but still alive. Most trees with cankers or fruiting bodies are < 10 cm in diameter (Fig. 3), but the number of small trees relative to medium and large trees within each plot was highly variable. Larger diameter trees typically exhibit few if any disease symptoms, particularly in the lower diameters.
Fig. 4. A–R. Diameter class distribution for white pine at each site, including standing dead trees, using data from the first and last year of measurements, respectively. The first bar in each pairing (black, striped) within each diameter class indicates total number of live + dead trees per hectare, while the second bar in each pairing (solid, red bars) indicates total number of dead trees only, per hectare. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)
1.8 m of the stem where there are likely to be very few or no branches and no smooth bark. It’s possible that the scale insect and/or *C. pinea* are less able to invade and colonize cambial tissue under thicker bark and under high resin-flow conditions, although this has not been proven (Schulz et al., this issue-a,b; Whitney et al., this issue). It’s also possible that cankers on larger diameter trees can be found further up the stem and on branches where the bark tends to be smoother; but perhaps cankers in these locations are less likely to girdle trees and lead to whole-tree mortality (Schulz et al., this issue-a,b; Whitney et al., this issue). The focus of this study, however, is not to elucidate the mechanism of mortality, but rather, to quantify trends in mortality of white pine at the landscape scale over a 5-year period and determine if there is sufficient concern for the future of the white pine resource.
3.2. Diameter distributions and mortality at individual sites

Diameter distributions and associated mortality are presented for all 9 sites using combined data from all plots and scaled up to trees per ha for the first and last year for which measurements were acquired from each site (Fig. 4A–R). Seven out of nine sites approached the negative exponential (reverse-J) diameter distribution associated with uneven-aged stands, although there was considerable variation from site to site.
The Matthews State Forest (Fig. 4I and J) and Brushy Fork Lake State Park (Fig. 4Q and R) sites showed normal to slightly bimodal distributions commonly associated with pine plantations. A visual comparison of mortality rates across the diameter distributions between the first and last year of measurement suggested significant increases in mortality among the smaller diameter cohorts at some sites, and marginal or no increases in mortality among others. The diameter distribution for the Winding Ridge, VA site (Fig. 4E and F) changed considerably from 2012 to 2016 due to the strong influence of one plot in which heavy recruitment of saplings in the 2–4 cm diameter class occurred during the 4-year time span. This plot lacked any large white pine and had little canopy overall, so heavy seedling recruitment and growth was understandable. White pine regeneration is most successful when associated with patches of disturbance that expose mineral soil or a thin litter layer (Wendel and Smith, 1990). These forest floor conditions can be created by harvesting, or naturally by wind-throw of large canopy trees (Cline and Spurr, 1942; Goodlett, 1954) or surface fires (Horton and Brown, 1960; Methven, 1973; Ahlgren, 1976).

3.3. Baseline mortality analysis for white pine at study sites

Combining the total number of live trees into 2-cm diameter classes across seven sites with uneven aged stands provided a more representative data set with which to calculate baseline mortality for white pine across the landscape. Diameter distribution was modeled using the first and last year of measurements for each site (ranging from 2012 to 2015 for the first year and 2014 to 2017 for the last year; see Table 1). The result is two diameter distributions each representing the earliest and latest tree measurements for all sites combined (Figs. 5a and 6a, respectively). Both diameter distributions are more broadly representative of the landscape as a whole compared to individual sites, and are more stable with regard to representing the negative exponential (reverse J) relationship. When tree data are transformed to the natural log (ln), a negative linear model fits each distribution with an r-square value of approximately 0.8 (Figs. 5b and 6b for earlier and later site measurements, respectively). Thus, the slope of each line and 2 cm diameter class interval can be used to calculate baseline mortality as follows:

\[
BM_{\text{earlier}} = 1 - (e^{-\beta \Delta D}) = 1 - (e^{-0.0627 \cdot 2}) = 1 - (1/e^{0.1254}) = 1 - 0.8821 = .1179 = 11.8\%
\]

\[
BM_{\text{later}} = 1 - (e^{-\beta \Delta D}) = 1 - (e^{-0.0637 \cdot 2}) = 1 - (1/e^{0.1274}) = 1 - 0.8804 = .1196\% = 12.0\%
\]

Therefore, baseline mortality estimates were nearly identical regardless of the time interval over which tree data were collected.

Using the study site estimates for baseline mortality, the next step is to graphically compare this with changes in observed mortality across the diameter distribution, both for the earlier and later data collection dates (Figs. 5c and 6c). For the early data collection dates (2012–2015), younger age classes were already showing signs of elevated (above baseline) mortality. Chi-square analysis indicated that the 6–8 cm
diameter class had significantly greater observed mortality compared to baseline mortality (Fig. 5c). Data collected 2–4 years later at each site (2014–2017) indicated several more diameter classes with a greater magnitude of observed mortality in the lower half of the diameter distribution (Fig. 6c). Chi-square analysis indicated that 5 diameter classes (4–6, 6–8, 8–10, 14–16, and 24–26 cm) had observed mortality that was significantly above baseline (Fig. 6c).

During the 5-year study there was an overall increase in observed mortality along the lower half of the diameter distribution, with observed mortality among 4 additional diameter classes exceeding expected baseline mortality. These changes were detected during only 3–5 years of monitoring. If continued monitoring reveals an expansion of this observed trend, it would suggest that white pine saplings and small trees are experiencing density-independent mortality that might be attributed to the scale-pathogen complex in question. Thus, independently of inter- and intra-specific vegetative competition, this complex may be influencing the future abundance and sustainability of white pine in mixed hardwood stands if fewer white pine understory trees are available to replace the mature cohort that will eventually die off. It remains to be seen whether mortality will increase among mid- to large diameter classes, although currently these seem stable and relatively unaffected overall by the scale/pathogen complex at the stand level.

### 3.4. Baseline mortality analysis for white pine using FIA data

The diameter distribution based on FIA plot data spanning the entire states of VA and WV, when fit to a log-linear model, produced a baseline mortality estimate of 13.4% (Fig. 7a and b). The diameter class had significantly greater observed mortality compared to baseline mortality (Fig. 5c). Data collected 2–4 years later at each site (2014–2017) indicated several more diameter classes with a greater magnitude of observed mortality in the lower half of the diameter distribution (Fig. 6c). Chi-square analysis indicated that 5 diameter classes (4–6, 6–8, 8–10, 14–16, and 24–26 cm) had observed mortality that was significantly above baseline (Fig. 6c).

### Fig. 5. Combined diameter class distribution (A), log-linear model of diameter distribution (B), and observed vs. baseline mortality (C) for all combined, non-plantation sites measured during the first year of plot establishment for each site. An * in (C) indicates where observed mortality was significantly different than baseline using chi-square analysis.

### Fig. 6. Combined diameter class distribution (A), log-linear model of diameter distribution (B), and observed vs. baseline mortality (C) for all combined, non-plantation sites measured during the final study year for each site. An * in (C) indicates where observed mortality was significantly different than baseline using chi-square analysis.

### Fig. 7. Combined diameter class distribution (A) and log-linear model of diameter distribution (B) for all FIA plots with white pine in VA and WV.
distribution based on FIA plot data spanning nine counties in Virginia and West Virginia where the study sites were located produce a baseline mortality estimate of 14.3% (Fig. 8a and b). These were calculated as follows:

$$BM_{VA, WV} = 1 - (1/e^{0.088 	imes 5.1}) = 1 - 0.6589 = 34.11$$

34.1% for 5.1 cm (2 in) diameter class

$$BM_{VA, WV} = 34.1 / 2.54 = 13.4\% \text{ for 2 cm diameter class}$$

$$BM_{Counties} = 1 - (1/e^{0.088 	imes 5.1}) = 1 - 0.6384 = 36.16 = 36.2\% \text{ for 5.1 cm}$$

$$(2 \text{ in) diameter class}$$

$$BM_{Counties} = 36.2 / 2.54 = 14.3\% \text{ for 2 cm diameter class}$$

Manion and Griffin (2001) similarly reported baseline mortality for white pine in the Adirondack State Park, New York as being 14.4% using 1 in (2.54 cm) diameter classes. Teale and Castello (2011) also present data indicating that white pine across the northern hardwood forest type in northern New York exhibits an unstable forest structure; as in the current study, observed mortality in the small sized classes was 2-3 times higher than baseline mortality, indicating that mid-size classes may decline in the future. Baseline mortality estimates from FIA plots were just over 2% greater in magnitude than the estimates derived from the study sites, providing confidence that the latter sampling was sufficiently large enough to estimate baseline mortality with a reasonable amount of accuracy. The baseline mortality rates derived from FIA data are probably more reliable because they were derived from many more plots and a much larger sample of trees, thus the very high r-squared values (0.97) for each of the log-linear models (Figs. 7b and 8b). In addition, sampling FIA plots from across only 9 counties produced an almost identical diameter distribution and r-squared as the statewide plot sampling, indicating that sampling at the multi-county level was more than sufficient. However, despite the lower sampling rate and higher amount of variability among the study sites, the baseline mortality rates derived directly from the study site data (11.8–12%) should be used to compare with observed mortality rates from those same study sites rather than the baseline mortality estimates from the FIA plots. The degree to which the scale/pathogen complex is an influence on the FIA plots, the exact locations of which are not known to the general public, is unknown, whereas the study sites were selected based on direct observation of cankers, scales, and other dieback indicators (Table 3 and Fig. 3).

No significant mortality above baseline levels was detected among the county-level FIA plots (Fig. 8c), perhaps indicating that the decline phenomenon is not yet ubiquitous. However, observed mortality data for trees < 13 cm are not available from FIA (Fig. 8c), yet these smaller diameter classes are where we would most expect observed mortality to exceed baseline based on our study sites.

4. Conclusions

While white pine is among the most shade tolerant species within the genus *Pinus*, it is considered to be intermediate in shade tolerance in general (Baker, 1949; Logan, 1966). As with many intermediately shade-tolerant species, eastern white pine ecology and silvics in mixed stands is complex. In addition, most eastern white pine literature originates from the northeast and upper mid-west of the U.S., along with southeastern Canada; very little white pine literature comes from studies in the southern Appalachians of the U.S. from Virginia and West Virginia south to northern Georgia. Making broad generalizations about what is ‘normal’ with regard to white pine ecology is, therefore, problematic. Multiple studies suggest intolerant species have higher death rates than more tolerant species (Leak, 1970; Stephens and Hill, 1971; Buchman, 1983; Buchman and Lentz, 1984), but Harcombe (1987) suggests that overall there are no obvious relationships between tree mortality rates and shade tolerance. Dovciak et al. (2003) used spatial pattern analysis to suggest that different size classes of white pine can occupy different areas of a stand due to variation in habitat conditions.

This complexity, however, does not preclude one from making reasonable generalizations. Rubin et al. (2006) suggest that baseline mortality analysis is appropriate at multiple scales, including stand, landscape and regional levels; however, sampling at levels below the landscape level often produces inadequate sampling of the larger diameter classes. This study was limited to only seven sites across a 9-county region, so results may not be robust due to site-to-site variation in soils and site index, species composition, vegetative competition, density, and other factors. Yet, baseline mortality analysis can be a sound approach when large scale landscape attributes are averaged together and there is sufficient plot replication. By using the larger sample sizes provided by the FIA plot network to estimate baseline mortality, which yielded a similar result as the study sites, it appears as though the level of sampling was sufficient to adequately detect some density-independent mortality among the smaller diameter classes. Mortality in larger size classes, which are more scattered and not well represented in the study plots, has been too small to detect within a 3-5 year time-span.

A much greater sampling of even-aged plantations over successive years would be necessary to apply the baseline mortality method to these systems. Although individual even-aged stands do not fit the reverse-J distribution at any one point in time, they generally follow it as they grow and develop (i.e. tree density progressively declines as even-aged stands grow over time). Therefore, at the landscape level, plantations exhibiting age-class diversity may be analyzed collectively at a broader spatial scale and, at a single moment, fit the reverse J distribution (Teale and Castello, 2011).
The addition of study sites and more years of monitoring will enhance our understanding of the true impact of this novel scale/parasite complex on the white pine resource. We have already seen a significant rise in mortality rate among the smaller diameter classes in just five years. If mortality rate in large diameter classes becomes apparent, it would also be appropriate to analyze potential impacts on white pine volume, but such impacts appear minimal at this time. Even so, excessive mortality in younger age classes could cause sustainability issues for white pine, especially if the mortality rate of mature trees were to accelerate for any reason.

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