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Aspen Health on National Forests in the Northern Rocky Mountain Region

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Introduction

Quaking aspen (*Populus tremuloides*) is an important and widely distributed species in the Western United States. Aspen forests support a variety of values such as diversity, wildlife, watersheds, and aesthetics. In the national forests (NF) of the northern Rocky Mountain Region this species comprises approximately 1% of the Bighorn NF cover, 3% of the Black Hills NF, and 1% of the Shoshone NF (DeBlander 2002, Menlove 2008, Witt 2008). It is a relatively rare component in these forests and there is concern regarding the health of this ecologically important species.

Long-term monitoring plots (Shaw 2004) and aerial detection surveys (Worrall et al. 2008) suggest extensive sudden decline and deterioration of aspen forests in the Rocky Mountains. Changes to the fire regimes since European settlement and heavy browsing (Bartos and Campbell 1998, Kay 1997, Romme et al. 1995, Sheppard et al. 2006), drought (Hogg et al. 2008, Worrall et al. 2008), and climate change (Rehfeldt et al. 2009, Worrall et al. 2013) have been suggested as factors contributing to decline and mortality.

Several diseases and insects are associated with aspen mortality (Dudley 2011, Fairweather et al. 2008, Frey et al. 2004, Guyon and Hoffman 2011, Hogg et al. 2008, Hogg and Michaelian 2014, Marchetti et al. 2011, Rehfeldt et al. 2009, Steed and Kearns 2010, Worrall et al. 2010, 2013). Aerial surveys in Wyoming provide general information regarding the extent and location of aspen mortality, but not the condition of regeneration, or specific damage agents associated with mortality.

To determine management recommendations, the distribution and severity of mortality and causal agents involved must be identified. In this study, damage agents and site variables were measured to determine factors contributing to aspen mortality. The objectives of the study were to: 1) evaluate tree and regeneration health, 2) quantify frequencies of damage agents, and 3) analyze tree mortality and regeneration stocking in relation to site, tree, and damage agents.

Methods

Sample design. Permanent aspen plots in the Bighorn (44 plots), Black Hills (59 plots), and Shoshone (45 plots) NFs were resampled from July 6 to August 25, 2015 (Figure 1). Plots were established in the Bighorn NF in 2009, and in the Black Hills and Shoshone NFs in 2008. All plots were previously remeasured in 2012.

The study design consisted of three circular plots in an aspen stand. Stands were greater than 2 acres, composed of 50% or more aspen, and had a minimum spacing of approximately 1 mile. For trees (≥ 3 inches DBH) 1/50 acre plots and for tree regeneration 1/500 acre plots were established. The spacing between plots within stands was approximately 2 chains. Regeneration plots were established due east and 16.7 feet from tree plot centers. Plots were installed 35 feet or more from stand edges, roads, or major trails.

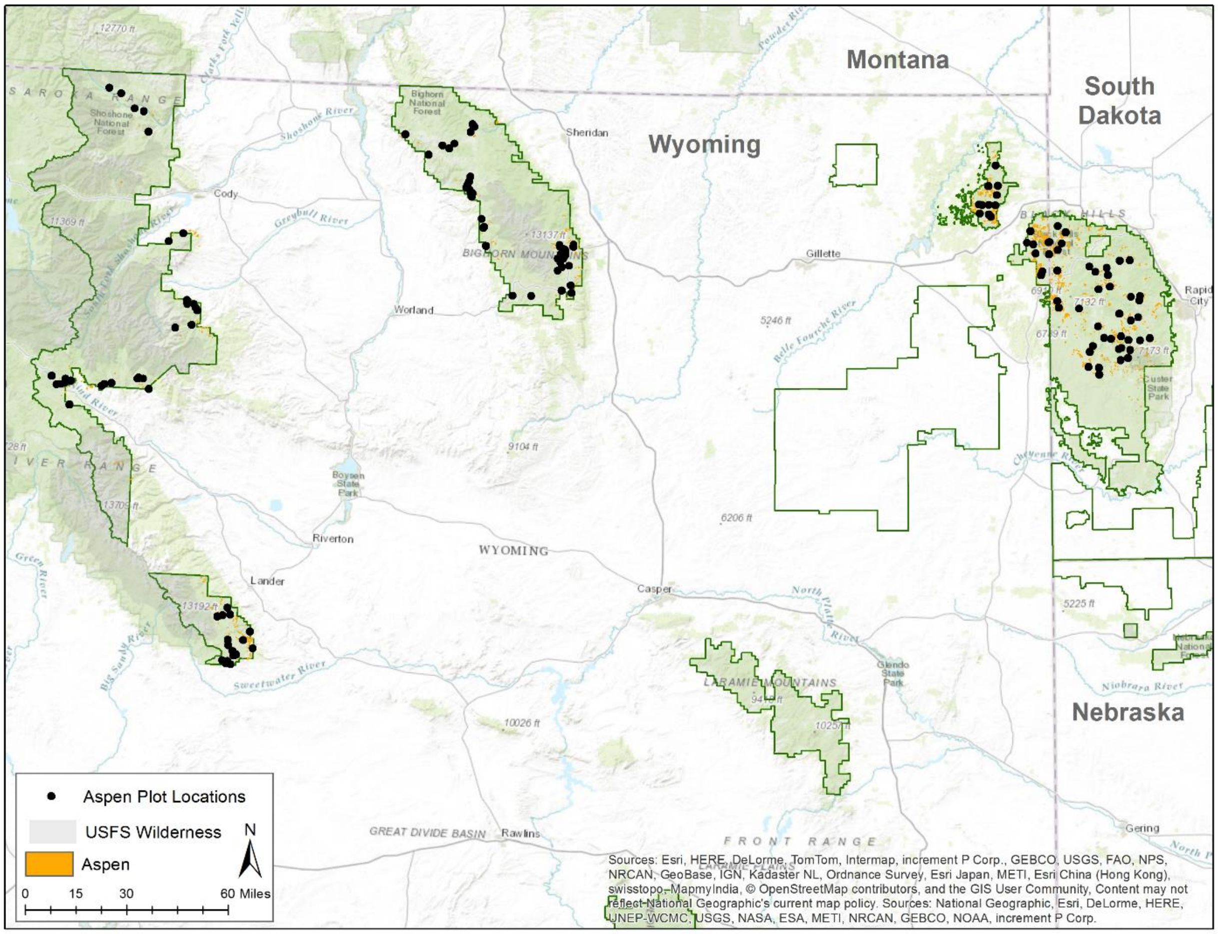

Figure 1. Location of stands and aspen cover type.

Plot and tree measurements. At each plot center coordinates, elevation, slope position, slope, and aspect were recorded. Tree species and diameter at breast height (DBH) were recorded in all plots. All trees ≥ 3 inches DBH were measured and live aspen trees were tagged. Trees were classified by condition (live or recent dead). Tree mortality rates per plot were calculated based on the number of trees that died since the last sampling divided by the number of years. For 2008/2009, mortality was estimated using recent dead trees. These trees had all their bark and at least some fine branches and were estimated to have died within 3 years.

Damage agents were recorded for each live and recent dead aspen tree, and were summarized as percentage of stems per plot. For most damage agents, any amount of damage was recorded. Foliage diseases and defoliating insects were only recorded if they damaged ≥ 25% of the crown. Animal and other physical damages were only recorded if they damaged ≥ 25% of the crown or stem.

Regeneration measurements. Regeneration was classified by species, host condition (living or dead), and size class (seedling or sapling). Seedlings were 1 foot to 4.5 feet high and saplings were > 4.5 feet and < 3 inches DBH. Regeneration variables calculated included numbers and percentages of regeneration per acre. Damage agents were tallied by host condition and size class for live and dead aspen regeneration.

Statistical analyses. Stand means were calculated from the three plots per stand. Data was analyzed using linear regression, at $\alpha = 0.05$.

Results

Bighorn National Forest. Mean tree mortality was estimated as 4% in 2009, and was 6% in 2012 and 3% in 2015 (Table 1). Aspen mortality was relatively low in most stands. Only 4 stands had more than 8% mortality (Figure 2). There was a significant correlation between mortality in 2009 and 2012 and between mortality in 2012 and 2015, indicating stands with elevated mortality continued to have elevated mortality.

The most frequently observed damage agent in trees was Cytospora canker (Table 1). While this canker was common, it was often minor (cankers <1 inch in diameter), frequently stopped expanding, and cankers healed without causing significant impact to trees. The number of Cytospora cankers has decreased on both live and dead aspen trees during the remeasurement periods. However, Cytospora canker did increase with tree mortality. Sooty bark canker was the second most common damage agent in trees, increased with tree mortality, often with large, expanding cankers, and was considered to be the most significant factor causing tree mortality. Unlike Cytospora, sooty bark has increased in both live and dead aspen over time on the Bighorn NF. Aspen trunk rot (*Phellinus tremulae*) was the third most common damage agent, increased with mortality, and resulted in stem breakage of large trees. Like Cytospora, it has declined through time, though not as dramatically as Cytospora. Both of the most common boring insects, poplar borer and bronze poplar borer, had relatively stable numbers over the evaluation period.

Mean aspen regeneration was 3,219 stems per acre in 2009, 3,433 stems per acre in 2012, and 3,045 stems per acre in 2015. Several stands had <1,000 stems per acre in 2015 (Figure 3), but only one stand had both poor regeneration and >8% mortality. Many of the stands that had <1000 stems per acre of regeneration had densest overstory, so little regeneration would be expected.

Only two damage agents were common in regeneration, but they did not increase with decreasing numbers of regeneration stems. In 2015, those damage agents included cankers (on 23% of the regeneration) and animal browsing (on 20% of the regeneration).

Table 1. Bighorn National Forest summary statistics

Variable	2009	2012	2015
DBH-Live Aspen	7.8	7.8	8.6
TPA-Live Aspen	450	409	393
Mortality (%)	4	6	3
Total Live Aspen Regeneration (number of stems)	3,219	3,433	3,045
Common Damages Recorded On Aspen Trees (% affected)			
Cytospora Canker-Live Aspen	34	39	17
Cytospora Canker-Dead Aspen	50	70	20
Sooty Bark Canker-Live Aspen	5	6	20
Sooty Bark Canker-Dead Aspen	48	61	97
Aspen Trunk Rot-Live Aspen	6	6	2
Aspen Trunk Rot-Dead Aspen	6	19	0
Poplar Borer-Live Aspen	1	7	0
Poplar Borer-Dead Aspen	0	11	0
Bronze Poplar Borer-Live Aspen	2	2	2
Bronze Poplar Borer-Dead Aspen	8	22	10

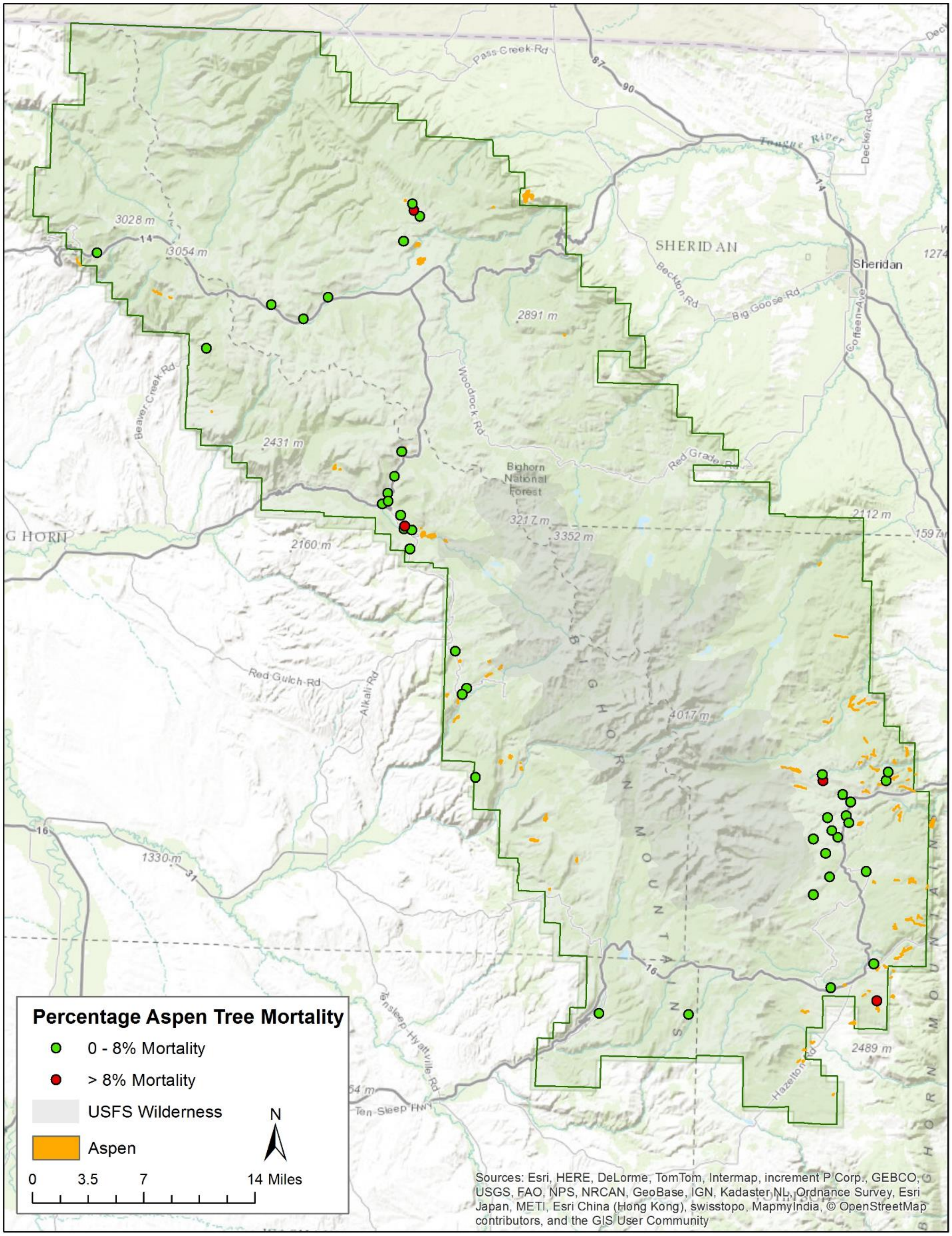

Figure 2. Aspen mortality associated with Bighorn National Forest stands in 2015.

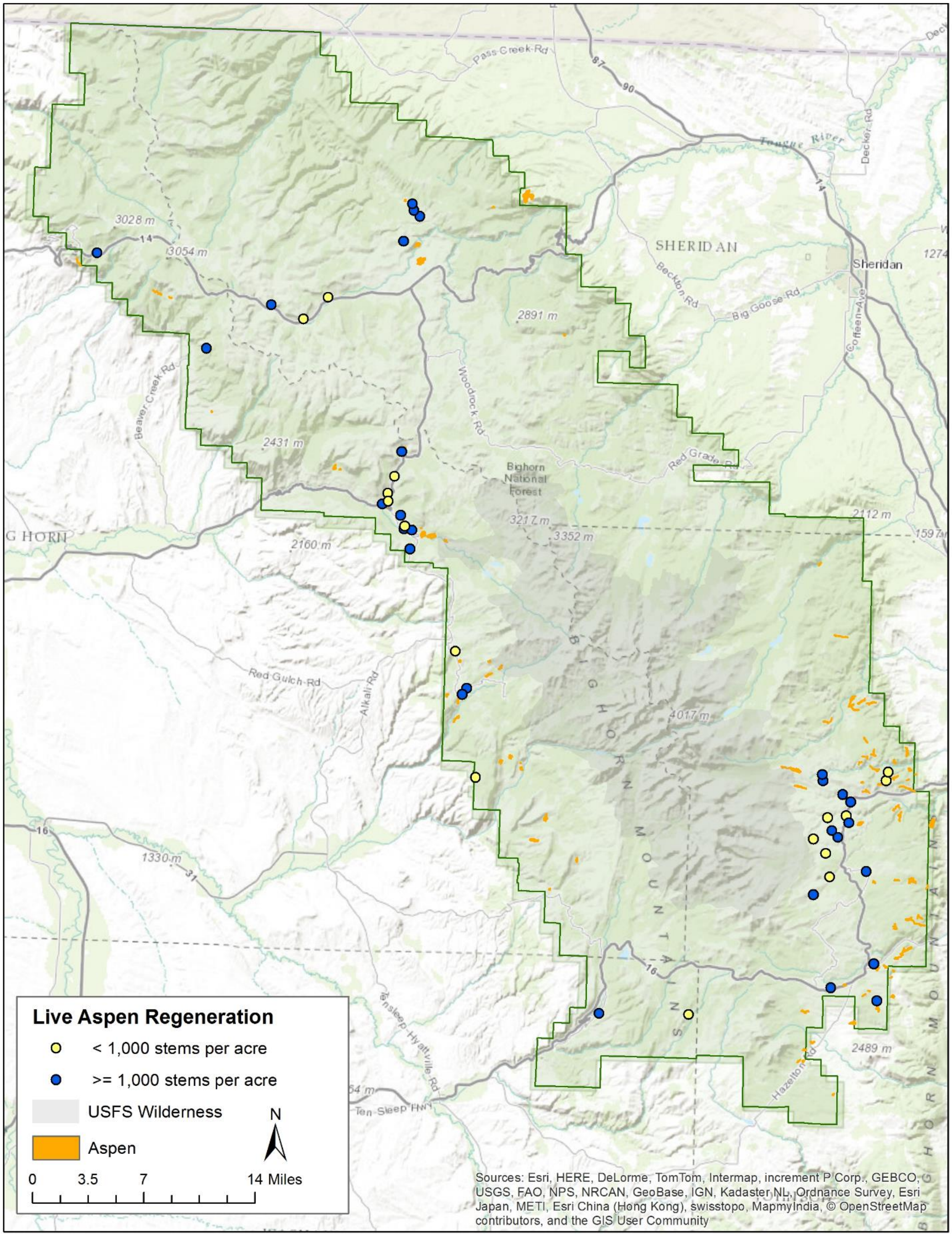

Figure 3. Amount of aspen regeneration associated with Bighorn National Forest stands in 2015.

Black Hills National Forest. Mean tree mortality was estimated as 2% in 2009, and was 6% in 2012 and 7% in 2015 (Table 2). Some of the increase in the 2015 mean percentage mortality is the result of extensive fire mortality in one stand. Aspen mortality increased over the years with a few stands exceeding 8% mortality (Figure 4). There was a significant correlation between mortality in 2009 and 2012 and between mortality in 2012 and 2015, indicating stands with elevated mortality continued to have elevated mortality.

The most frequently observed damage in trees was Cytospora canker (Table 2). While this canker was common, it was often minor (cankers <1 inch in diameter), frequently stopped expanding, and cankers healed without causing significant impact to trees. The number of live trees with Cytospora cankers remained relatively uniform from year to year. The percent of dead trees with Cytospora cankers has declined in the Black Hills. Sooty bark canker, increased with tree mortality, often with large expanding cankers and was considered to be the most significant factor causing tree mortality. The percentage of both live and dead aspen with sooty bark had a decreasing trend in the Black Hills. Aspen trunk rot (*Phellinus tremulae*) was the second most common damage agent (26% of the trees), increased with mortality, and resulted in stem breakage of larger trees. Some of the increase in mortality in 2015 is likely due to the stem breakage resulting from internal decay and high winds. Bronze poplar borer and poplar borer numbers decreased in 2015 to less than 5% of stems, and neither increased with mortality.

Mean aspen regeneration was 2,911 stems per acre in 2009, 1,906 stems per acre in 2012, and 884 stems per acre in 2015. Several stands had <1,000 stems per acre in 2015 (Figure 5). Many of the stands that had <1,000 stems per acre of regeneration had: 1) dense overstory, so a large number of aspen seedlings would not be expected; and/or 2) high hardwood regeneration (other than aspen), and in a few stands, conifer regeneration. Competing woody vegetation probably explains much of the low aspen regeneration. Although we did not look at competing grasses and forbs, this non-woody vegetation might explain some of the low aspen regeneration, and may have hidden some of the smaller aspen regeneration.

Overall, the number of seedlings and saplings is declining on the Black Hills and in 2015 averaged below 1,000 stems per acre. Only two damage agents were common in regeneration and they showed no correlation with decreasing numbers of regeneration. In 2015, those damage agents included cankers (in 32% of the regeneration) and animal browsing (in 16% of the regeneration).

Table 2. Black Hills National Forest aspen summary statistics

Variable	2008	2012	2015
DBH-Live Aspen	7.0	6.9	7.4
TPA-Live Aspen	350	321	284
Mortality (%)	2	6	7
Total Live Aspen Regeneration (number of stems)	2,911	1,906	884
Common Damages Recorded On Aspen Trees (% affected)			
Cytospora Canker-Live Aspen	34	47	28
Cytospora Canker-Dead Aspen	50	71	48
Sooty Bark Canker-Live Aspen	30	33	14
Sooty Bark Canker-Dead Aspen	71	86	45
Aspen Trunk Rot-Live Aspen	21	28	23
Aspen Trunk Rot-Dead Aspen	16	43	34
Poplar Borer-Live Aspen	4	17	3
Poplar Borer-Dead Aspen	3	23	5
Bronze Poplar Borer-Live Aspen	14	14	2
Bronze Poplar Borer-Dead Aspen	17	40	10

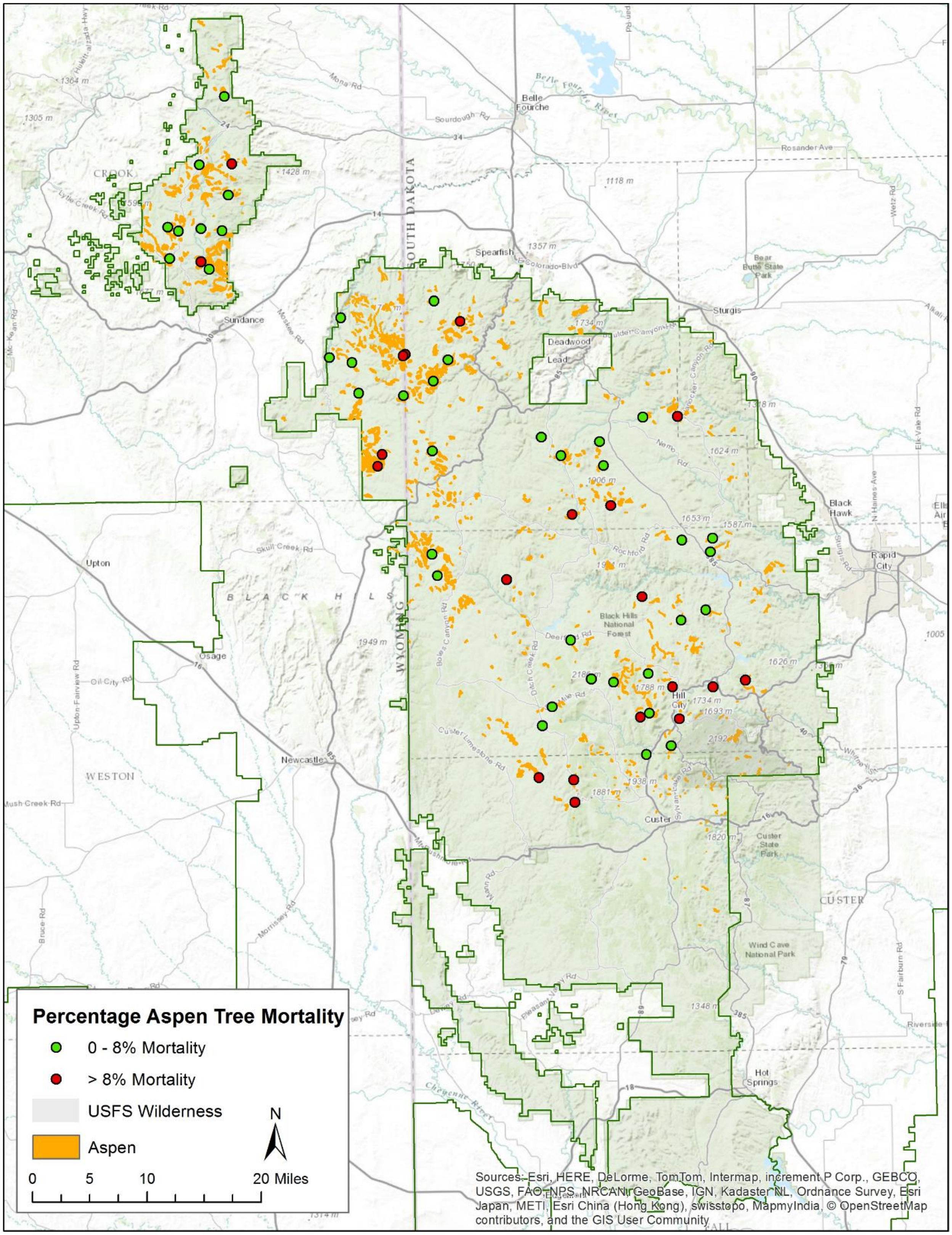

Figure 4. Aspen mortality associated with Black Hills National Forest stands in 2015.

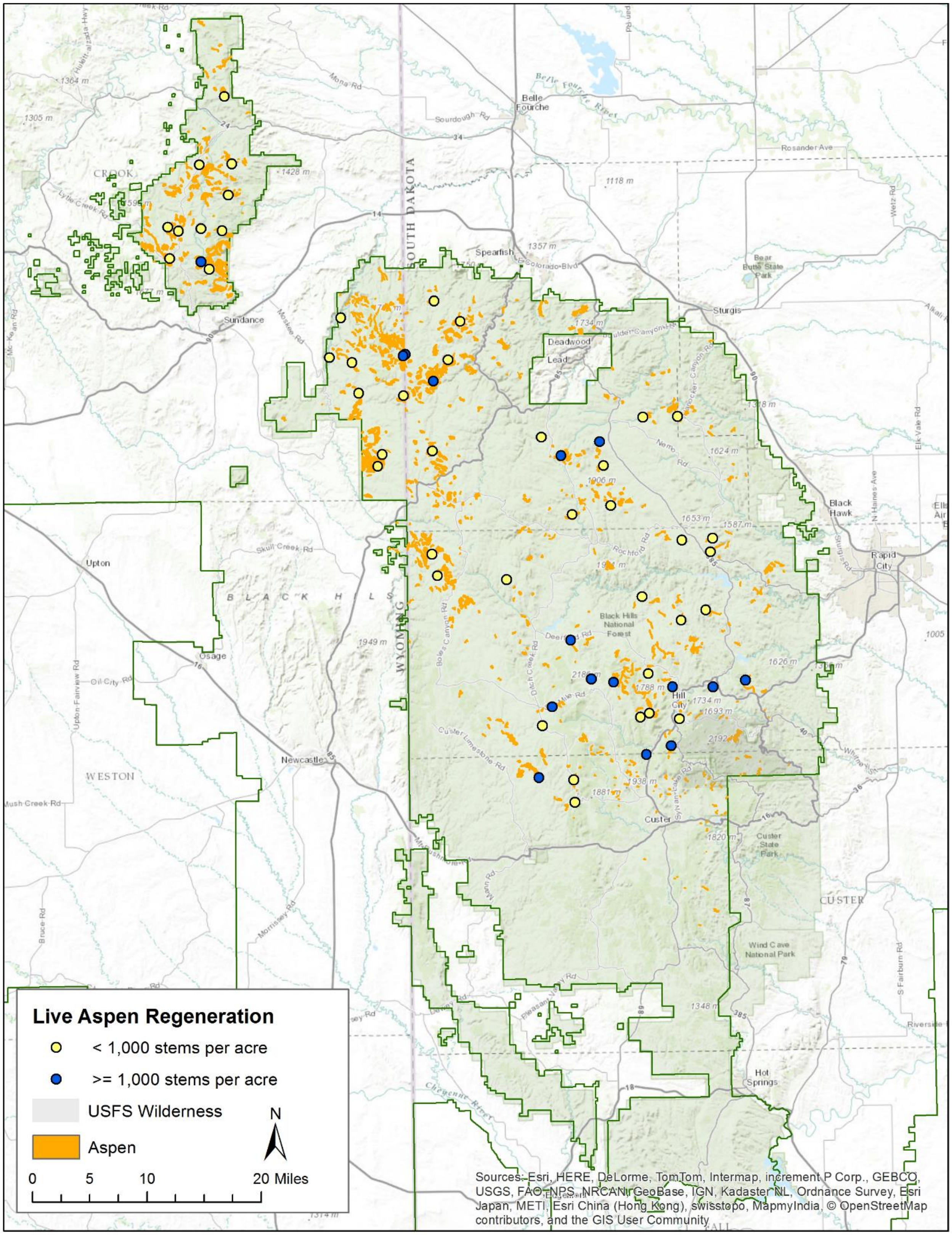

Figure 5. Amount of aspen regeneration associated with Black Hills National Forest stands in 2015.

Shoshone National Forest. Mean tree mortality was estimated as 4% in 2008, and was 6% in 2012 and 5% in 2015 (Table 3). Aspen mortality was low in most stands. Only a few stands had more than 8% mortality (Figure 6), and 4 of those stands had total (or near total) tree mortality from fire. Fire increased the mean percentage mortality in both 2012 and 2015. There was a significant correlation between mortality in 2009 and 2012 and between mortality in 2012 and 2015, indicating stands with elevated mortality continued to have elevated mortality.

The most frequently observed damage in trees was Cytospora canker (Table 3). While this canker was common, it was often minor (cankers <1 inch in diameter), frequently stopped expanding, and cankers healed without causing significant impact to trees. Cytospora cankers decreased in both live and dead aspen trees on the Shoshone NF. Cytospora cankers had a minor correlation with tree mortality. Sooty bark canker increased with tree mortality, frequently had large expanding cankers, and was considered to be the most significant factor causing tree mortality. Like Cytospora, the number of cankers in both live and recent dead trees decreased in 2015. Aspen trunk rot (*Phellinus tremulae*) appeared to remain relatively uniform over the evaluation period. It can be the main cause of stem breakage in mature trees. The percentage of trees with bronze poplar borer and poplar borer decreased in 2015, and neither increased with tree mortality in 2015.

Mean aspen regeneration was 4,070 stems per acre in 2008, 4,180 stems per acre in 2012, and 4,181 stems per acre in 2015. Only three stands had <1,000 stems per acre in 2015 (Figure 7).

Two damage agents were common in regeneration and did not increase with decreasing numbers of stems. In 2015, those damage agents included cankers (in 17% of the regeneration) and animal browsing (in 3% of the regeneration).

Table 3. Shoshone National Forest aspen summary statistics

Variable	2008	2012	2015
DBH-Live Aspen	7.0	7.1	7.5
TPA-Live Aspen	349	316	309
Mortality (%)	4	6	5
Total Live Aspen Regeneration (number of stems)	4,070	4,180	4,181
Common Damages Recorded On Aspen Trees (% affected)			
Cytospora Canker-Live Aspen	38	46	29
Cytospora Canker-Dead Aspen	71	82	50
Sooty Bark Canker-Live Aspen	12	18	9
Sooty Bark Canker-Dead Aspen	43	61	39
Aspen Trunk Rot-Live Aspen	6	11	10
Aspen Trunk Rot-Dead Aspen	13	13	11
Poplar Borer-Live Aspen	5	15	3
Poplar Borer-Dead Aspen	7	38	7
Bronze Poplar Borer-Live Aspen	7	11	1
Bronze Poplar Borer-Dead Aspen	28	48	6

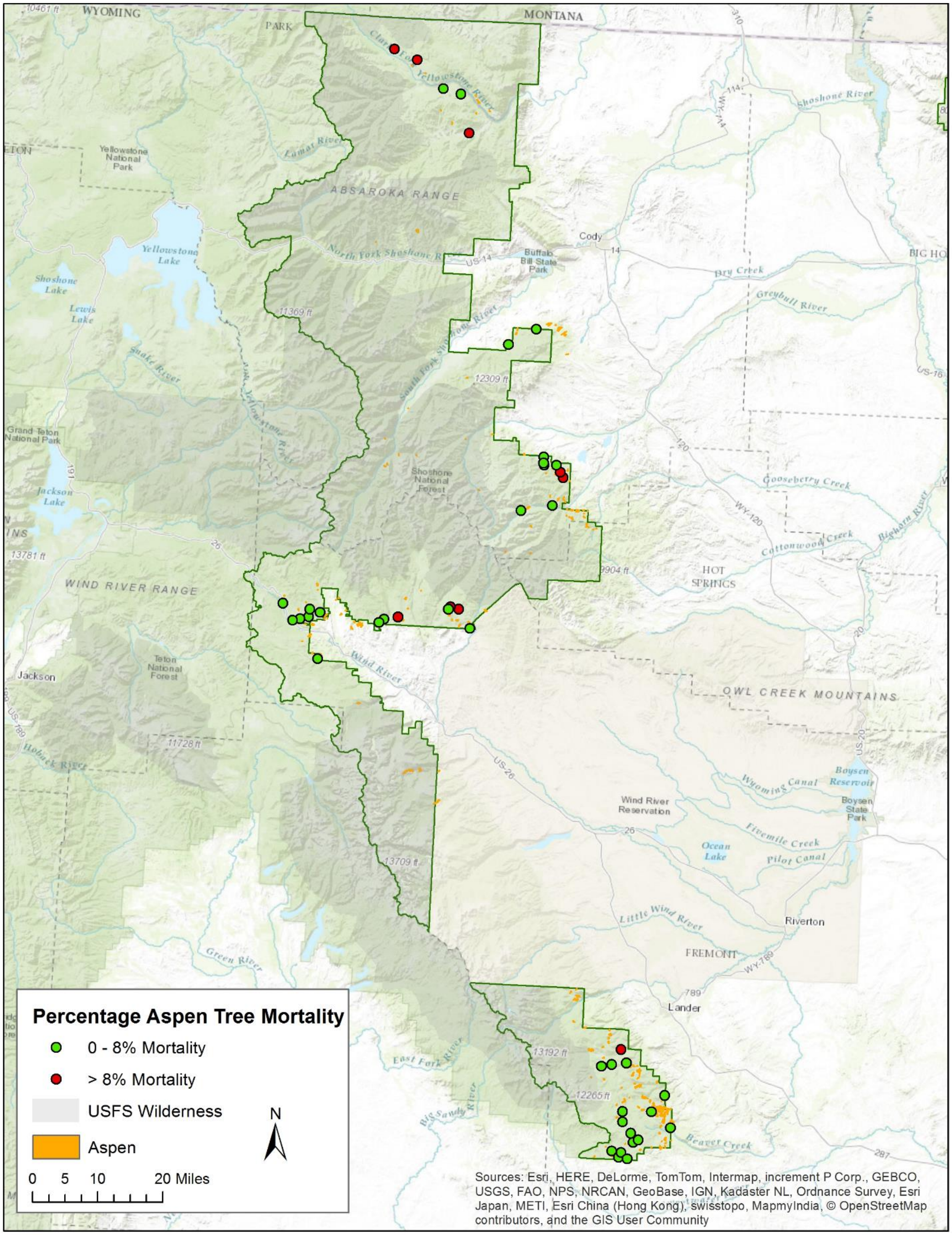

Figure 6. Aspen mortality associated with Shoshone National Forest stands in 2015.

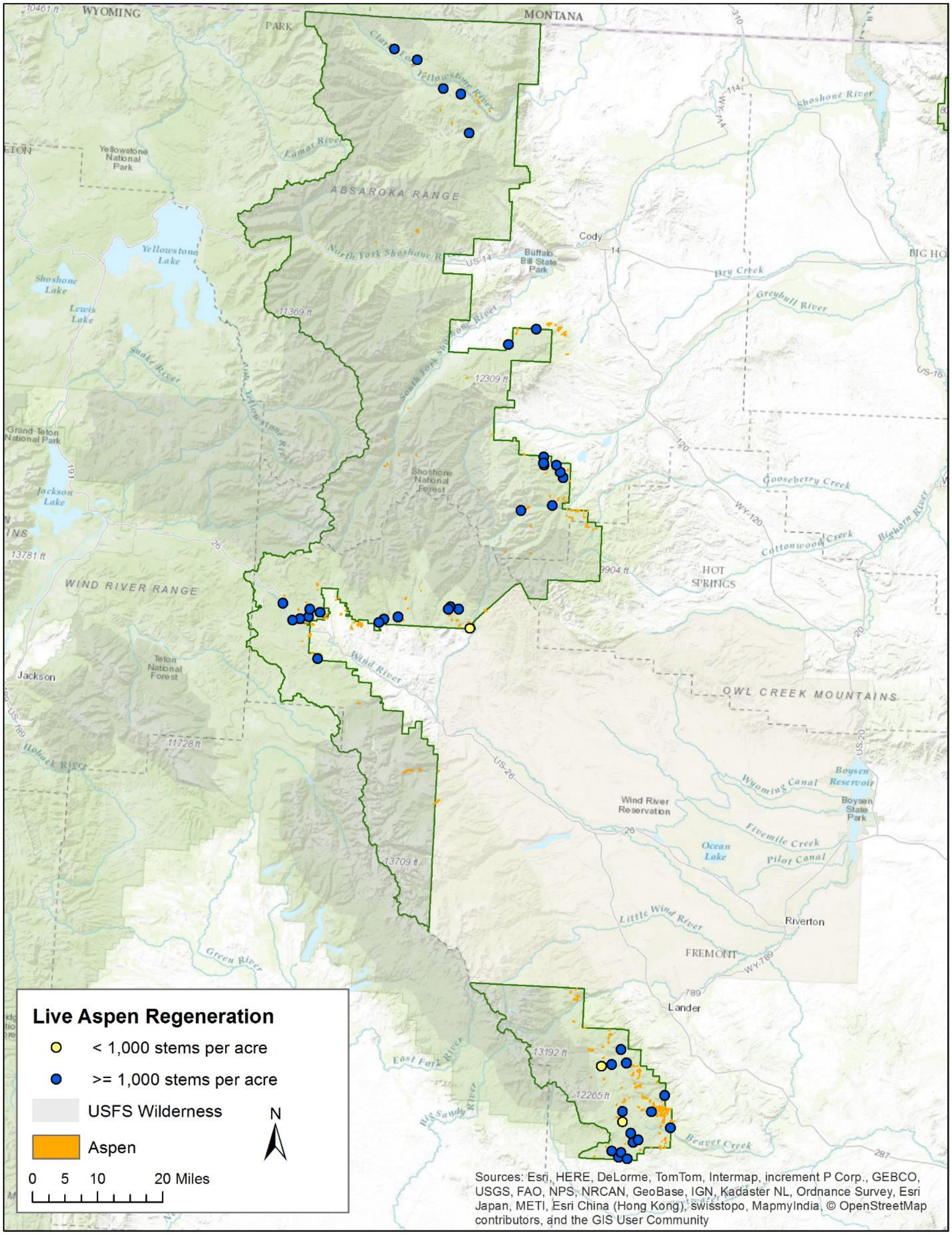

Figure 7. Amount of aspen regeneration associated with Shoshone National Forest stands in 2015.

Discussion

Most of the aspen stands in these three forests are healthy. Average stand mortality has remained low, indicating no significant tree mortality events are occurring. As is common in forests, the average tree diameter of live aspen has increased and the average trees per acre of live aspen has decreased. Slightly elevated tree mortality is associated with a few individual stands, so stands that are seeing above average mortality are expected to continue to have slightly elevated mortality. There are numerous insect and disease agents in these forests, but only three are common and show any trend with tree mortality. Various factors including drought, tree diseases, and insects have been associated with aspen mortality (Frey et al. 2004, Hogg et al. 2008, Marchetti et al. 2011, Rehfeldt et al. 2009, Worrall et al. 2010, 2013). We found no clear relationships between tree mortality and factors such as elevation, slope, aspect, tree density or age, species composition, or climate (moisture, temperature, etc.).

Thirty three damage agents were found in trees with up to eight in an individual tree. However, most of these agents were found in <1% of the trees, thus were not contributing to significant mortality at the forest level.

Although many damage agents were observed, only three agents, sooty-bark canker, Cytospora canker and aspen trunk rot were weakly correlated with tree mortality in 2015. The principal damage agents causing mortality in our study area are often associated with aspen mortality throughout the region (Dudley 2011, Guyon and Hoffman 2011, Fairweather et al. 2008, Marchetti et al. 2011, Steed and Kearns 2010). The relationships between mortality and damage agents were weak and are likely due to: multiple damage agents contributing to mortality, different frequencies of damage agents in different stands, low overall mortality in the forests, and varying site conditions among stands. Damage to live trees was included when tallying damage agents, which did not contribute to mortality in the sampled year. Therefore strong correlations were not expected.

Sooty bark canker is common in all three forests. It has been suggested as a major aspen mortality agent (Hinds 1985). This damage agent was frequently observed in trees with full, healthy crowns, but those trees were often dead the following re-measurement year (Figure 8).

Sooty-bark canker is the most damaging agent, but Cytospora canker and aspen trunk rot are contributing to mortality. Although Cytospora canker was more common and is causing some mortality, the majority of the cankers were small, already healed, or likely to heal. Aspen trunk rot was also common in all three Forests causing stem breakage of larger trees due to extensive internal decay and high winds. Other damage agents can cause tree mortality, but their correlations with mortality were not significant or they were infrequent. Consequently, damage agent aggressiveness, tendency with mortality, and frequency should all be considered when determining potential impacts on forest health at the landscape level.

Sudden increases or reductions in the percentage of trees colonized by cankers can result from environmental conditions. "Wave-years" of infection might explain the variability in the percentage of trees colonized by canker diseases among years. When conditions are ideal for infection, a larger percentage of trees are infected. Wave-years occur when ideal moisture and temperatures conditions co-occur at the time of fungal sporulation. For canker diseases, sporulation co-occurring with wounding can also be a factor in increased infection such as sporulation during hail, wind, animal, etc. damage. Likewise, unfavorable conditions for infection can result in reduced or no new infections.

Overall, aspen regeneration was common and relatively dense across all forests, but there were stands with low regeneration. As with trees, during the first years of this study there were no clear relationship between regeneration numbers and several factors such as elevation, slope, aspect, tree density or age, species composition, or climate (moisture, temperature, etc.). However, when looking at stands with poor regeneration, the most important factors in reducing aspen regeneration seemed to be related to individual sites. Factors such as dense overstory, competing regeneration from other species, and in a few cases fire, appeared to have the greatest impact on reducing aspen seedlings. However, cankers and browsing might play a role in reducing aspen regeneration in some stands.

Browsing is often cited as a factor preventing successful aspen regeneration (Bartos and Campbell 1998; Kay 1997; Romme et al. 1995). However, there were relatively high numbers of cankers found in aspen seedlings and saplings (Figure 9) in all forests. Jacobi and Shepperd (1991) found canker diseases to be a factor associated with low regeneration in clearcuts. On small trees, we often did not identify the specific pathogen that caused the cankers, however Cytospora and sooty bark cankers were identified on dead and dying seedlings and saplings. Cankers were frequently associated with browse damage. Thus, browsing damage is the likely entry point for the damaging canker pathogens. Reducing browsing will likely reduce mortality caused by the canker diseases.

Management Recommendations

- Regenerate older aspen stands by clearcutting, prescribed fire, or wildfire to stimulate regeneration and help maintain aspen on sites.
- Sooty-bark canker and aspen trunk rot (not Cytospora canker) tends to attack older trees, so managing aspen in rotations of less than 100 years would reduce losses from those diseases.
- Avoiding wounds should reduce the likelihood of infections from both canker diseases.
- Partial cutting in aspen stands is strongly discouraged since wounding often results in canker infection. Partial cutting also might result in subsequent wounding as a result of increased boring insects.
- Exclosure fencing would reduce animal damage that provides an entry point for cankers to infect both trees and regeneration.
- Clonal variation in susceptibility to decay has been demonstrated for aspen trunk rot. Favoring clones with low levels of decay would select for resistance.
- Aspen trunk rot conks are good indicators for detecting and estimating decay. Decay typically extends 8-12 ft in each direction from conks, and cull increases with number of conks.

Figure 8. Sooty bark canker in a live (left) and dead (center) aspen trees. Cytospora canker in a live aspen tree (right).

Figure 9. Animal browsing with cankers in aspen seedlings (left and center), and canker in an aspen sapling (right).

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