Chapter 11. Climate, Canker, and Alder Mortality in the Southern Rockies

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Introduction

An increase in dieback and mortality of thinleaf alder (*Alnus incana* ssp. *tenuifolia*) raised concern in southern Wyoming and Colorado two decades ago. In 1990, there was concern over extensive mortality of thinleaf alder along Big Laramie River near Jelm, WY, as well as in multiple locations in Colorado.[2] Trees of all ages were affected and Cytospora canker was associated with the mortality. In 1996, records of the Sulphur Ranger District, Arapaho National Forest indicated that a grazing allotment had severe dieback and mortality of alder (fig. 11.1). In 2005, damage was continuing, consistently associated with Cytospora canker. In other areas, property owners and biologists have noted the damage, some indicating that it began before 1995.

By 2000, land managers and others increasingly noted the problem and inquired as to the cause. The goals of the work reported here were to quantify the extent and severity of dieback and mortality from southern Wyoming to northern New Mexico, and to assess a variety of potential direct and indirect causal factors. A portion of this work has been published (Worrall 2009, Worrall and others 2010).

Methods

Two surveys were conducted. The first was an extensive survey of randomly selected, 4th-level watersheds likely to have alder in southern Wyoming, Colorado, and northern New Mexico in 2004. Transects were placed in accessible areas on public land that had alder according to the following hierarchical criteria: (1) accurately represent the proportion of dieback and mortality of alder in the area, (2) include as much alder as possible, and (3) otherwise represent the vegetation and site conditions in the area (fig. 11.2). Along 30-m transects, canopy intercept was measured, separated by live versus dead. For each genet of alder whose canopy intersected the transect, the following data were recorded: distance to edge of stream; distance to road; number of standing stems in the categories healthy, alive with dieback, and dead; and within each of those categories, the number with Cytospora canker or fruiting and the number with evidence of wood borers. Number of live sprouts of the genet was scored none, low (1-5 sprouts), medium (6-15 sprouts), and high (>15 sprouts). Evidence of browsing and notes on solar exposure were recorded.

A similar but more intensive survey was conducted within the upper Gunnison River basin, in Colorado, in 2005. The transect

[2] Johnson, D.W. 1991. Letter to State Forester of Wyoming. On file with: U.S. Department of Agriculture Forest Service, Rocky Mountain Region, Forest Pest Management, LSC-91-4, 240 West Prospect Road, Fort Collins, CO 80526.

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Figure 11.1—Dieback and mortality of thinleaf alder in Colorado and associated Cytospora canker. (A) Mortality in 2005 on a tributary of Eight Mile Creek in the Cottonwood grazing allotment near Granby, CO. The larger stems shown here were already dead in a 1996 photo (not shown). (B) Dead and dying alder in 2005 on both sides of Taylor River near Gunnison, CO, heavily used for recreation. (C) and (D) Cytospora canker associated with dieback and mortality. Note the dense fruiting completely filling the canker surface. The top (C) and side (D) of a canker are marked with white paint. [Photos by Doreen Sumerlin, U.S. Department of Agriculture Forest Service (A) and James Worrall, U.S. Department of Agriculture Forest Service (B), (C), and (D) (from Worrall 2009)]

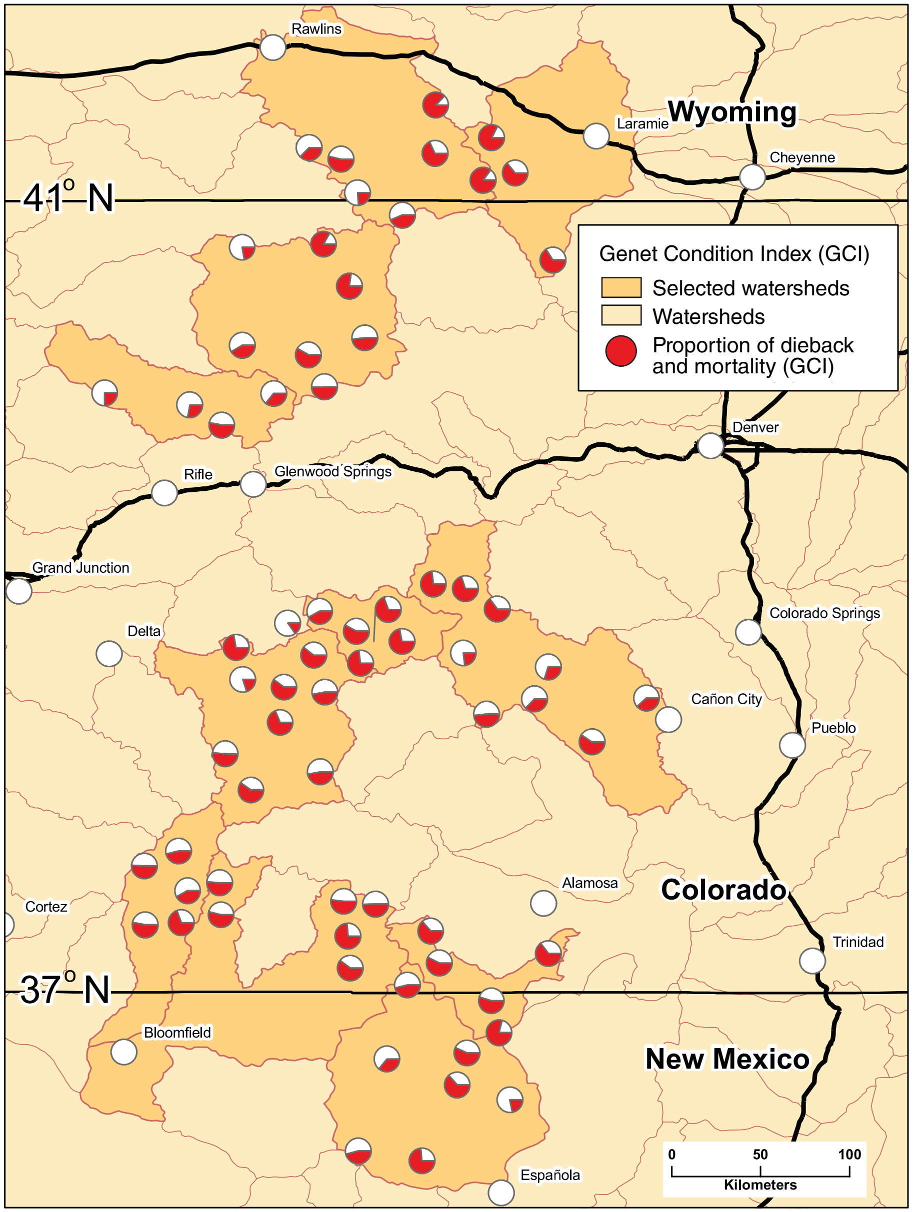

Figure 11.2—Transect and watershed locations in the extensive survey, 2004. The proportion of the circle that is filled indicates the severity of dieback and mortality as calculated by the Genet Condition Index (from Worrall 2009).

intercept of alder was recorded, and stems in each alder genet were classed as healthy, dieback, and dead. In 2006, a subsample of the 2004 transects in or near the upper Gunnison River basin was relocated and remeasured. In addition to the alder intercept, d.b.h. of each stem was recorded, and live and dead sprouts were counted on each genet.

Alder condition was expressed as Genet Condition Index (GCI). GCI was based on number of stems of intercepted genets classified as healthy (H), dieback (B) or dead (D): GCI = (0.5 • B + D) • 100 / (H + B + D). As a measure of damage, this index weights stems with dieback at one-half the value of dead stems. Growth of 50 cankers was measured monthly from March through October 2006 in locations throughout the upper Gunnison River basin, in Colorado.

Long-term climate data were subjected to spectral analysis to detect oscillations in summer temperatures. A summer heat index was calculated using the methodology of and the same reference period as Hansen and others (1998) and further described at http://data.giss.nasa.gov/csci. The three variables used were extreme maximum monthly temperature, the monthly mean maximum temperature, and the monthly mean temperature. For each year, each variable was averaged over the months of June, July, and August. The mean of the reference period (1951–80) was subtracted from the value for each year and the result divided by the standard deviation of the mean for the reference period. The resulting values for the three variables were averaged to obtain the annual index value. Various periodograms and evolutive spectra were used to assess the periodicity of the data using the SSA-MTM Toolkit for Spectral Analysis (Ghil and others 2002).

Results and Discussion

In the extensive survey, 68 transects from southern Wyoming to northern New Mexico were measured (fig. 11.2). Of 6,503 standing stems, 37 percent were dead, 29 percent had dieback, and 34 percent were healthy. Transects intercepted 1,479 m of live and 1,177 m of dead alder canopy. The second, more localized survey had 32 transects and yielded very similar results. Abundance of live sprouts decreased as dieback and mortality increased. Genets with no live sprouts had significantly poorer stem condition (GCI = 77) than those with live sprouts, and genets in the highest sprout class had significantly better stem condition (GCI = 45) than those with fewer sprouts (Fishers protected least significant difference, $P < 0.05$). Canopy dieback and mortality (GCI) were strongly correlated with percentage of sprouts that were dead at both the transect level (Pearson product moment correlation; $r = 0.72$, $P = 0.002$) and the genet level ($r = 0.40$, $P < 0.001$, $n = 157$ genets). These findings suggest that affected genets are dying and are not replacing themselves successfully through vegetative reproduction.

Damage did not vary substantially by geographic area (fig. 11.2) and was not related to elevation, animal browsing, or distance to nearest road. Symptoms were not consistent with disease of alder caused by *Phytophthora alni* in Europe, and isolations for *Phytophthora* species were negative.

Cytospora canker was consistently associated with dieback and mortality when stems and branches were examined closely. Gerard Adams, Michigan State University, identified the pathogen as *Valsa melanodiscus* (anamorph *Cytospora umbrina*), and subsequent isolations and microscopic examination of fruiting bodies indicated that it was the proximate cause of the dieback and mortality.

Canker expansion and killing of branches and stems occurred primarily in the warmest part of summer (fig. 11.3; temperature data should be regarded as relative, as most alder sites probably have somewhat cooler weather than the station in Gunnison). Most cankers grew little or not at all up to late June, except those on trees whose tops had already been largely killed by the

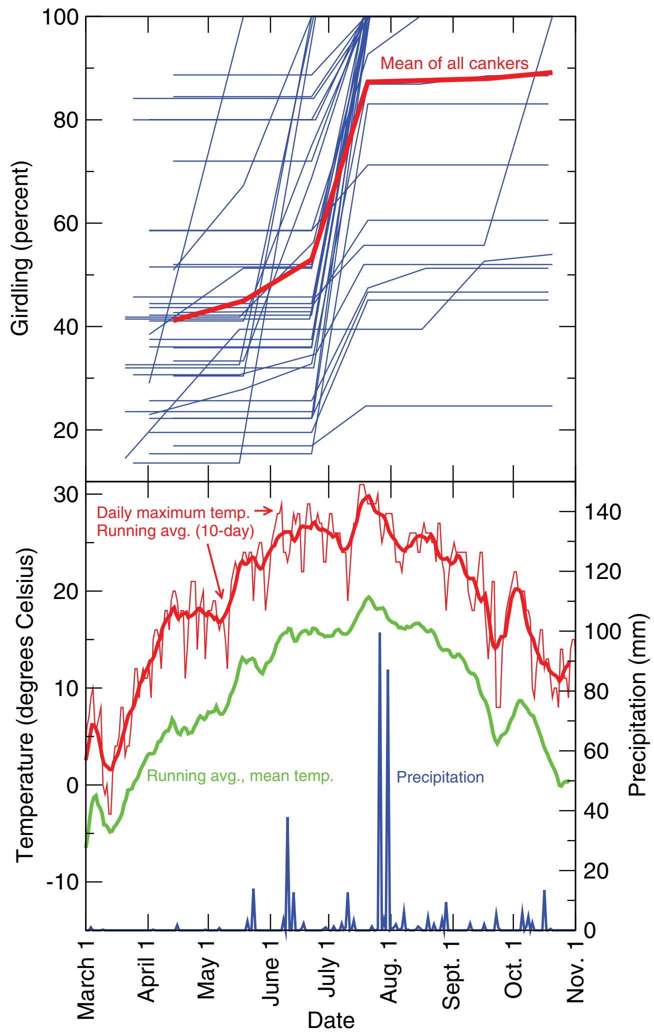

Figure 11.3—Development of 39 marked cankers (thin blue lines) that grew significantly during March-October 2006, expressed as percent girdling (growth of canker around the circumference). The bold red line is the average of all cankers. The graph below shows weather at Gunnison (weather station number 724677) for the corresponding period. Bold red and green lines are moving averages of the 10-day period centered at each point.

pathogen the previous year. However, from late June to late July almost all cankers grew rapidly and most girdled their stems. Most did not grow at all after late July. The period of rapid canker growth was the hottest part of summer (fig. 11.3). Between June 20 and July 20, maximum temperature was ≥ 25 °C on 24 days and peaked at 30 °C or above on four consecutive days. The warm period was then broken by 2 days with the highest precipitation of the summer. This marked the arrival of the summer monsoon climate pattern. Precipitation occurred on 21 days of the first 4 weeks of the monsoon, while temperature dropped to daily means generally below 15 °C and daily maxima mostly below 25 °C.

Although drought is often suspected in this damage, several facts argue against it: (a) alder condition improved slightly with distance from stream (significant but weak correlation); (b) alder damage began well before the drought of 2000–2005; (c) active dieback was common in many alders rooted immediately on the banks of streams that remained flowing, with their roots bathed in flowing water continuously through this and other droughts.

Long-term climate data were analyzed to determine if climate patterns could account for the long-term epidemic of Cytospora canker in alder. Positive values of a summer heat index indicate temperatures warmer than the mean of the reference period (1951–80) and vice versa (fig. 11.4). Spectral analysis (Blackman-Tukey and Maximum Entropy Method) indicated that the time series contained a dominant signal with a frequency of 0.047 cycles per year, or a period of 21.3 years. Multi-taper analysis led to rejection of the white-noise null hypothesis for the peak ($P < 0.01$). Singular spectrum analysis showed two empirical orthogonal functions (EOFs) forming an oscillating pair with frequencies of 0.048 and 0.049 above the 95 percent confidence interval. Reconstruction of that oscillation showed a good correspondence with variation in the summer heat index. The oscillation was particularly regular with high amplitude in the middle of the 20th century, with alternating cold and warm phases lasting about a decade each. However, the oscillation gradually became slower and dampened after the late 1970s, and a subsequent phase shift cannot be reliably identified. This coincided with the beginning of significantly increasing trend of temperatures in most regions of Colorado (Ray and others 2008).

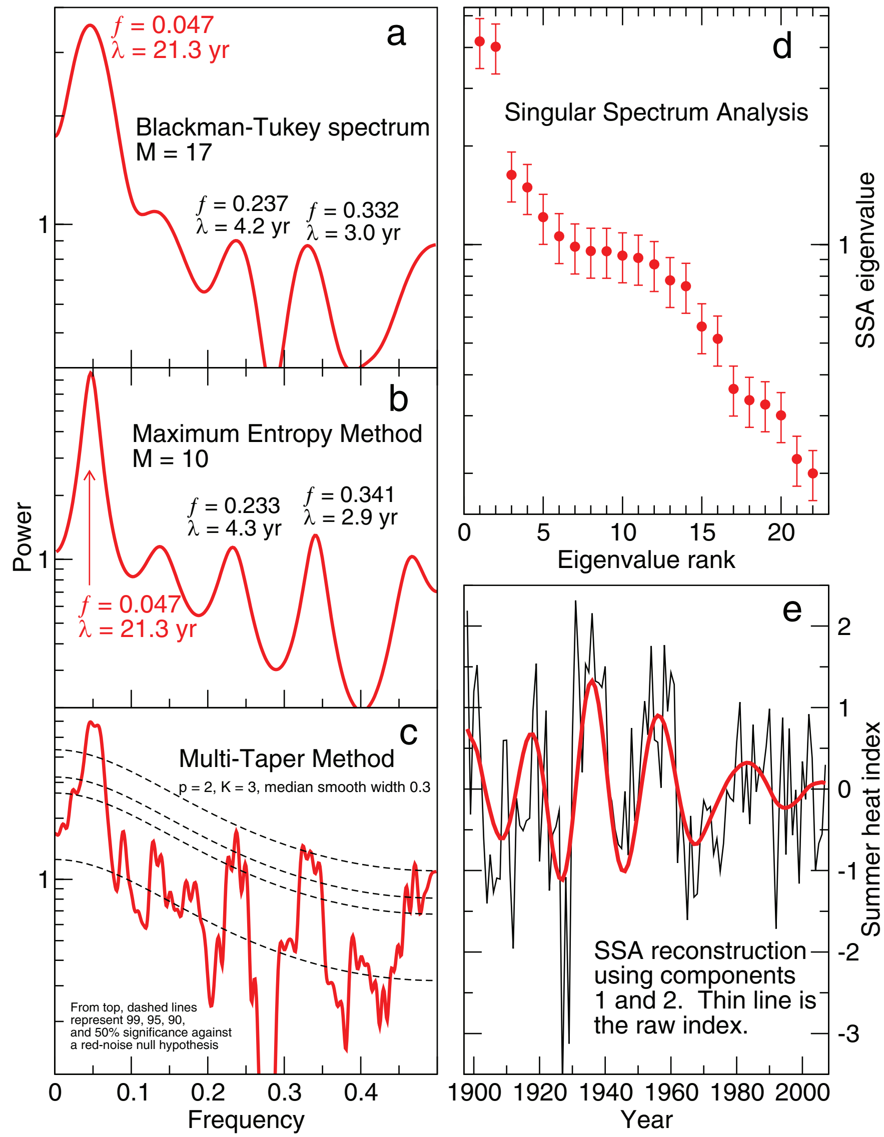

Figure 11.4—Spectral analysis of Gunnison summer heat index using Blackman-Tukey, Maximum Entropy Method, Multi-Taper Method, and Singular Spectrum Analysis. The first pair of components in the eigenvalue plot (upper right) represent a significant oscillation (f = 0.047), which is reconstructed at lower right against the raw index.

Conclusions

1. Observers noted increased dieback and mortality of thinleaf alder in the southern Rocky Mountains beginning on or before 1990.
2. As of 2004, about one-third of standing alder stems were dead, and one-third had dieback in the southern Rocky Mountains.
3. Genets with dying and dead stems have fewer live sprouts and higher sprout mortality than healthy genets. Genets appear to be dying rather than replacing themselves vegetatively.
4. Cytospora canker, caused by *Valsa melanodiscus*, is the proximate cause of the dieback and mortality.
5. Except on stems near death, cankers grow only during the hottest part of summer.
6. A spectral analysis of long-term climate data revealed a 21-year cycle of summer heat, but the frequency has slowed and amplitude has weakened since the late 1970s.
7. During periods with high summer temperatures, alder is apparently stressed and becomes susceptible to Cytospora canker. The canker grows and kills very quickly during warm periods. During cool phases, cankers may not develop, giving alder an opportunity to recover and regenerate. With the dampened cycle in recent decades, periods of consecutive cool summers may not be long enough to permit recovery.
8. With recent and projected increases in summer temperatures in the southern Rocky Mountains, more severe epidemics of Cytospora canker may be expected.

Literature Cited

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