Dieback and Mortality of *Alnus* in the Southern Rocky Mountains, USA

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ABSTRACT

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Dieback and mortality of *Alnus incana* subsp. *tenuifolia* in the Southern Rocky Mountains apparently began by the late 1980s and have become a concern to land managers. A survey of alder including 68 transects from southern Wyoming to northern New Mexico indicated that, of 6,503 standing stems, 37% were dead, 29% had dieback, and 34% were healthy. Transects intercepted 1,479 m of live and 1,177 m of dead alder canopy. A second, more localized survey with 32 transects in the upper Gunnison River watershed of Colorado yielded similar results. Abundance of live sprouts was inversely related to amount of dieback and mortality in a genet, suggesting that affected genets are dying and not replacing themselves. Damage did not vary substantially by geographic area and was not related to elevation, animal browsing, or distance to nearest road. Distance to nearest stream was weakly, inversely related to severity of dieback and mortality. Symptoms were not consistent with disease of alder caused by *Phytophthora alni* in Europe, and isolations for *Phytophthora* species were negative. Cytospora canker, caused by *Valsa melanodiscus* (anamorph *Cytospora umbrina*), is the proximate cause of the dieback and mortality.

Alnus incana subsp. *tenuifolia*, thinleaf alder, ranges from the Mackenzie River Delta on the Arctic Ocean south to the Santa Catalina Mountains of southern Arizona, and from central Alaska and the Pacific Coast east to central Saskatchewan in Canada and the Rocky Mountains in the United States (12). Although it occurs near sea level in the north, in the south it is restricted to higher elevations and riparian areas. In the arid mountains, these riparian habitats are important landscape components, and alder is often a dominant plant. It stabilizes soils, hosts bacteria that fix nitrogen, and provides various wildlife values, tree structure (often being the largest riparian species), and shade to cool streams and improve fish habitat. It has only poor to fair palatability for livestock (21), so it may persist in heavily used range better than *Salix* species.

Beginning in 2000, land managers and others increasingly raised concerns about high levels of branch dieback and mortality of alder in Colorado (Fig. 1A and B). 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.

Although many diseases and insects are known from *Alnus* species, reports of widespread mortality are rare. In northeastern Oregon, an episode of dieback and mortality in *A. incana* subsp. *tenuifolia* in the late 1980s was associated with *Cytospora* sp. (11). Because wounding was required for infection in experimental inoculations, it appeared that the epidemic was driven by wounding by large herbivores and/or river ice. In western Scotland, severe dieback and mortality of *A. glutinosa* was observed since the late 1980s. A variety of fungi were associated with cankers, including *Ophiovalsa suffusa* (13) and *Valsa oxystoma* (22), but the primary pathogen and cause of dieback were unclear. Damage tended to occur in drainages with ephemeral or highly variable water flow, suggesting that moisture stress was involved (22). Various additional episodes of dieback and mortality of alders have been documented over the years, especially in Europe (7). These include damage due to hydrological extremes, unsuitable provenances of planted alder, frost, and various biotic agents.

A recent, serious epidemic of Phytophthora disease of alder in Europe was caused by new variants grouped under the species *Phytophthora alni*, primarily *P. alni* subsp. *alni* (5,6,22). Until recently, *P. alni* was not known to be in North America. A *Phytophthora* species was reported in Minnesota with DNA sequences and

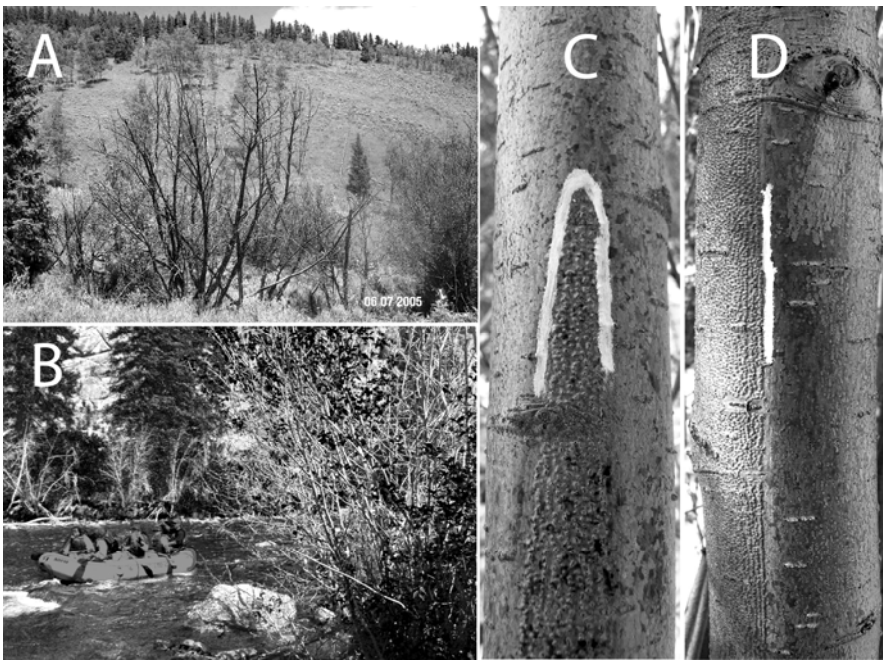

Fig. 1. Dieback and mortality of thinleaf alder in Colorado and associated Cytospora canker. **A,** Mortality in 2005 on a tributary of Eight Mile Creek in the Cottonwood grazing allotment near Granby, CO. The larger stems shown here were already dead in a 1996 photo (not shown). **B,** Dead and dying alder in 2005 on both sides of Taylor River near Gunnison, CO. **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.

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morphology suggesting similarity to *P. alni* subsp. *alni* (17), but it did not prove pathogenic to *Alnus glutinosa* (16). In late 2007, *P. alni* subsp. *uniformis* was confirmed in samples from Alaska, but there was no indication it was causing any damage to alder (1; G. C. Adams, *personal communication*). I looked for symptoms of *Phytophthora* infection and attempted to isolate *Phytophthora* species to determine if they might be causing the observed alder damage.

MATERIALS AND METHODS

The geographic scope of this study is the Southern Rocky Mountains, from southern Wyoming through Colorado to northern New Mexico. This is the Southern Rocky Mountain physiographic province (9), and is within the Southern Rocky Mountain Steppe–Open Woodland–Coniferous Forest–Alpine Meadow ecoregion province (4).

Two surveys were conducted to quantify the extent and severity of dieback and mortality and to gather preliminary data that may point to causal factors. The first was an extensive survey of randomly selected, fourth-level watersheds in southern Wyoming, Colorado, and northern New Mexico in 2004. Among watersheds that were at least partially in Colorado, overlapped the distribution of alder (12), with elevations likely to support alder (generally above 1,800 m), and with at least 20,000 ha of federally managed land (not including tribal lands), 11 were selected for sampling. Sampling effort was allocated based on amount of federal land; approximately 40 h were spent on most watersheds and 60 h on larger watersheds.

Riparian areas are long and narrow, and the occurrence of alder within those habitats is patchy. For these reasons, a random or strict, systematic sampling scheme was considered inefficient and unproductive. Transects were placed in accessible areas that had alder according to the following hierarchical criteria: (i) accurately represent the proportion of dieback and mortality of alder in the area, (ii) include as much alder as possible, and (iii) otherwise represent the vegetation and site conditions in the riparian area. All sampling within a survey was done by a single crew.

Line-intercept transects were 30 m long except in a few cases where obstacles (streams, bluffs, etc.) prevented complete transects. Along each transect, canopy intercept of each shrub species (potentially >10 cm tall), separated by live versus dead, was measured (3,8). Live and dead canopy sometimes overlapped and were measured as both live and dead in that case.

In the Southern Rocky Mountains, alder generally grows in discrete clusters of stems. In most cases, it can readily be seen that these arose as sprouts from a common root system or stems, and these are considered genets (clones). 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. Evidence of browsing along the transect, including browsing on any plants, was recorded as the type of animal (primarily separating livestock from wild herbivores) and quantified in 3 classes: 0 = no evidence; 1 = some evidence; 2 = evidence of substantial impact to soil surface and vegetation. Stems were defined as shoots ≥3 cm DBH (diameter at breast height, 1.4 m) that arose within 1 m from the ground line. Number of live sprouts (sprouts were ≥1 m long, originating <1 m above ground, and <2 cm DBH) of the genet was scored none, low (1 to 5 sprouts), medium (6 to 15), and high (>15). For most transects, notes were recorded regarding solar exposure, and transects were classified as full sun, partial shade, or full shade on this basis.

A similar but more intensive survey was conducted within the upper Gunnison River basin (largely Gunnison County), CO in 2005. The transect intercept of alder was recorded. Stems in each alder genet were classed as live, dieback, and dead without regard to incidence of borers or Cytospora canker. Browsing, road distance, stream distance, and sprout information were not recorded. Transects were marked permanently with steel rods.

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, DBH of each stem was recorded, and live and dead sprouts were counted on each genet.

Alder condition was expressed as two indices. Genet Condition Index (GCI) was based on number of stems of intercepted genets classified as healthy (H), dieback (B), or dead (D): $GCI = (0.5 \cdot B + D)/(H + B + D) \cdot 100$. As a measure of damage, this index weights stems with dieback at one-half the value of dead stems. It could be calculated for individual genets, transects, or the entire population of stems in a survey. Transect Intercept Index (TII) was based on the intercept length of live (L) and dead (D) alder: $D/(L + D) \cdot 100$. Since dead and live canopy sometimes overlapped, TII cannot be considered a percentage of alder canopy that is dead.

Isolation of *Phytophthora* species was attempted using the *Phytophthora*-selective medium CARP (14). Chips of phloem from surface-sterilized canker margins were placed in the medium. Isolation of *Valsa* species was conducted with 1.5% malt extract agar acidified after autoclaving with 3.3 ml/liter 10% (vol/vol) lactic acid. Fruiting bodies were collected in the field and used to identify species.

RESULTS

Alder dieback and mortality. Sixty-eight transects were measured in the extensive survey. The transects intercepted a total of 1,479 m of live and 1,177 m of dead alder canopy, for an overall TII of 44.3 (mean of transect TII values = 43.7).

Of 6,503 standing stems on 859 intercepted genets, approximately one-third was in each of the classes healthy, live with dieback, and dead (Table 1). The most abundant class was dead, with 37% of stems. The overall GCI was 51.3 (mean of transect GCI values = 53.8).

In the intensive survey of the upper Gunnison River basin, 32 transects intercepted a total of 572 m of live and 394 m of dead alder canopy, giving an overall TII of 40.8. Of 1,965 standing stems on 287 intercepted genets, 37% were healthy, 23% were live with dieback, and again dead was the most frequent class with 40%. The overall GCI was 51.5.

Condition of sprouts (including basal sprouts and root suckers) was associated with amount of stem dieback and mortality. In the extensive survey, genets with no live sprouts had significantly poorer stem condition (GCI = 77, $\alpha = 0.05$) than those with live sprouts, and genets in the highest sprout class had significantly better stem condition (GCI = 45) than those with fewer sprouts (Fig. 2A). In a resample of 15 of those transects in which all live and dead sprouts on each genet were counted, GCI was strongly correlated with percentage of sprouts that were dead at both the transect level ($r = 0.72$, $P = 0.002$; Fig. 2B) and the genet level ($r = 0.40$, $P < 0.001$, $n = 157$ genets).

Factors potentially associated with cause. Although transects varied greatly in severity of alder condition, there was no apparent geographic trend in the variation within the sampled region. For example, GCI often varied as greatly within watersheds as between, and there was no appar-

Table 1. Number of stems classified as healthy, dieback, and dead and percentage of each class in which Cytospora canker and borer holes were detected in the extensive survey of northern New Mexico, Colorado, and southern Wyoming, 2004

Stem condition class	No. of stems	% of total	% with Cytospora canker	% with borer holes
Healthy	2,214	34	0.4	0.05
Dieback	1,902	29	40.3	3.6
Dead	2,387	37	78.2	13.9
Total	6,503	100		

ent variation with latitude or side of the Continental Divide (Fig. 3).

In the analysis of distance to stream versus GCI, genets on four transects were excluded because distance was recorded as >100 m. These transects were located on wetlands or hillside seeps rather than along streams. Genets were also excluded from the analysis when they had fewer than three stems. A large number of the remaining 604 genets were located immediately adjacent to streams, so stream distance was not normally distributed and the nonparametric Spearman rank correlation test was employed. There was a significant negative correlation between stream distance and GCI, indicating that genet health improved somewhat with greater distance from the stream. However, the correlation coefficient was small (Spearman's rho = –0.106, $P = 0.010$).

Distance to road was tested similarly but was not significantly correlated with GCI of genets (Spearman's rho = –0.015, $P = 0.746$).

Elevation and browsing were also unrelated to alder condition. In both surveys, neither TII nor GCI was significantly correlated with elevation. For TII versus elevation in the extensive survey, the correlation coefficient (r) was 0.041; $P = 0.737$ (Fig. 4). Neither damage variable was significantly related to browsing intensity when all browsing or when only cattle browsing was considered.

Neither TII nor GCI varied significantly among solar exposure classes (full sun, partial shade, and full shade) according to an analysis of variance (Table 2). However, when transects in full sun were compared only with those in full shade, those in full sun had significantly higher TII. GCI was still not significant in that comparison.

Roots were examined at six sites with varying levels of dieback and mortality by carefully excavating, washing, examining, and measuring live and dead root mass. Roots were in good condition with few necrotic roots (data not shown). Although the quantity of nodules varied greatly among sites, that variation was not related to stem condition.

Cankers were commonly observed on live stems with dieback and on dead stems; holes left by wood borers were less commonly observed (Table 1). Incidence of cankers was probably underestimated because cankers were hidden on upper branches and because signs and symptoms were lost on dead, deteriorated bark. Although cankers were sometimes hard to find in early stages of dieback because they were restricted to upper branches, often they occurred low on the main stems and killed branches as they grew around the branch bases. Cankers were found invariably when branches could be examined closely, and death of branches and stems coincided with girdling by the canker.

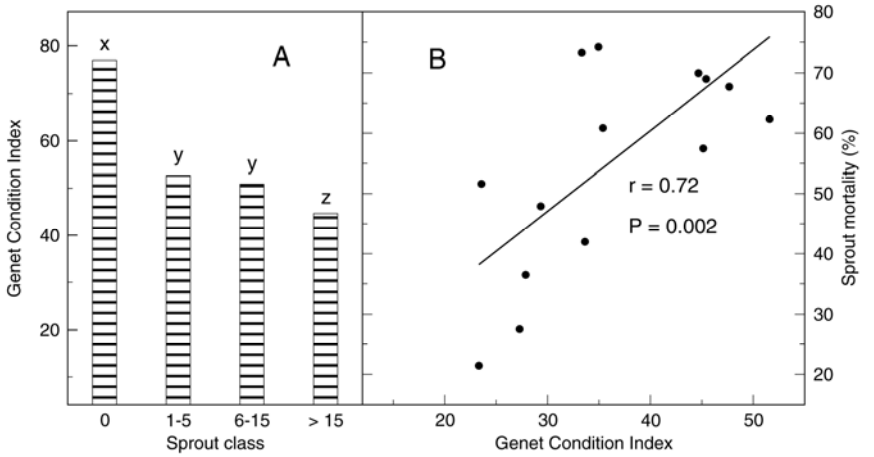

Fig. 2. Poor sprouting success in genets with dead and dying stems (high Genet Condition Index, GCI). **A,** GCI vs. sprout class in the extensive survey. Sprout classes (indicated by number of live sprouts) with different letters have significantly different GCI ($P < 0.05$, Fishers protected least significant difference). Number of genets represented in each class is 110, 324, 295, and 128, respectively. **B,** Sprout mortality vs. GCI in a remeasurement of 15 transects in which live and dead sprouts were counted. Sprout mortality was significantly correlated with GCI. Correlation coefficient (r) and its level of significance are shown.

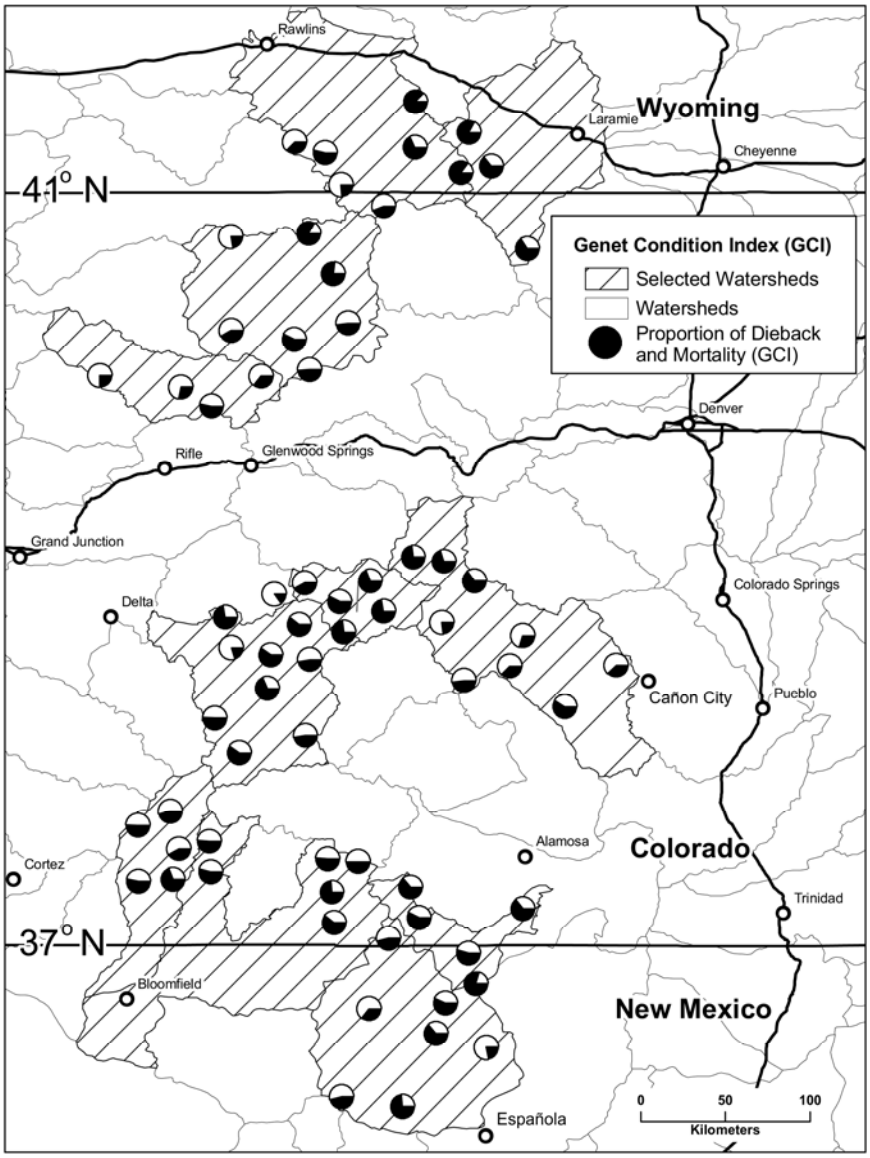

Fig. 3. Transect and watershed locations in the extensive survey, 2004. Genet Condition Index is indicated by proportion of the symbol that is shaded.

Cankers sometimes originated from shoot tips, leading to dieback and developing into branch and stem cankers as they grew proximally. However, many stem cankers did not enter the stem from branches but were initiated directly on the stem. Cankers were never observed in association with visible wounds. Cankers were generally long and narrow, sometimes reaching up to 3 m in length before girdling the stem (Fig. 1C and D). They were diffuse but sometimes stopped growing, in which case cankers became sunken and surrounded by callus.

Symptoms were compared with those of Phytophthora disease of alder species in Europe (22). Small, yellow, or sparse foliage was rarely seen before dieback and mortality. Rather than falling prematurely, foliage that was killed failed to abscise when live foliage normally did, and it often remained on through winter. Cankers were found throughout the aboveground portion of the plant rather than primarily near the stem base. Although cankers often reached the ground line, they were never found in the root crown or roots. Cankers never had liquid or tarry exudation. Isolations for *Phytophthora* species were consistently negative.

Cankers almost always were covered with fruiting bodies. Although the fruiting was sometimes sterile or immature ascomata, generally it was conidiomata of the form-genus *Cytospora* or ascomata of the genus *Valsa*. Isolations from these fruiting bodies resulted in cultures that stained the agar reddish and often produced conidiomata. This fungus was consistently isolated from active canker margins. Cultures and fruit bodies were identified by Gerard C. Adams, Michigan State University, as *Valsa melanodiscus* and its anamorph, *Cytospora umbrina*.

Beginning of epidemic. Several records show evidence of a distinct deterioration in alder condition in the early 1990s. In 1991, Wyoming Cooperative Extension Service contacted USDA Forest Service, Forest Pest Management regarding extensive mortality of thinleaf alder along Big Laramie River near Jelm, WY. Such extensive mortality also had been recorded in multiple locations in Colorado by one of the observers. A report of the observations indicated that trees of all ages were affected and that Cytospora canker was associated with the mortality (D. W. Johnson, 1991 letter to State Forester of Wyoming, USDA Forest Service, Rocky Mountain Region, Forest Pest Management, LSC-91-4).

In 1996, the Cottonwood grazing allotment, Sulphur Ranger District, Arapaho National Forest near Granby, CO, was considered to be in a deteriorated state due to overgrazing. In addition to erosion, invasive weeds, and loss of forage, severe dieback and mortality of alder was observed along Eight-Mile Creek. Biologist Doreen Sumerlin documented the damage with Polaroid photographs, and in 2005 we revisited the site (Fig. 1A). Many of the genets dead in 1996 were still standing. Although there was some ingrowth, additional alder had died and dieback was continuing. Cytospora canker was consistently associated with dieback and mortality. In other areas, property owners and biologists have noted the damage, some indicating that it began a decade or more ago.

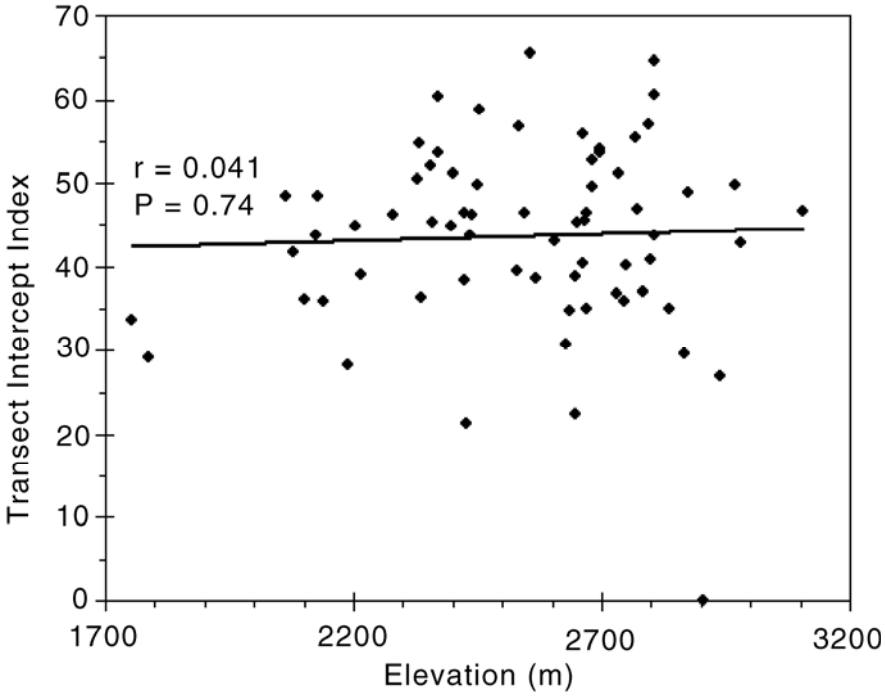

Fig. 4. Variation in Transect Intercept Index vs. elevation in the extensive survey. $n = 68$ transects.

Table 2. Alder condition variables (Genet Condition Index, GCI; and Transect Intercept Index, TII) by solar exposure of 54 transects in the extensive survey

				P (ANOVA)	
	Full sun	**Partial shade**	**Full shade**	**All 3 classes**	**Full sun vs. full shade**
n	18	23	13		
Mean GCI	52.9	59.2	46.3	0.114	0.236
Mean TII	45.9	46.1	38.7	0.079	0.039

DISCUSSION

A high proportion of alder in the Southern Rocky Mountains was damaged. About one-third of the standing stems were dead and another third were diseased. Length of transects intercepting dead canopy was 70 to 80% of that of live canopy.

It is conceivable that this condition represents a steady-state condition of alder rather than an increase in dieback and mortality. This might be possible if stems grew quickly, died early, and dead stems remained intact and standing for long periods. However, this is unlikely for several reasons. First, I conducted a stage-based matrix model analysis of alder populations based on our survey data and annual ring counts of live and dead stems (data not shown). Although insufficient data on stage transition rates are available to test the model completely, this preliminary analysis suggests that a steady state is only remotely feasible, given the status of the population. Second, alder genets with stems in poor condition have relatively few live sprouts and high sprout mortality, suggesting that the genets are dying rather than replacing themselves. Finally, reports indicate that alder condition worsened noticeably, causing concern in Wyoming and Colorado at least by 1991. Therefore, it appears that alder has experienced increased dieback and mortality beginning by the late 1980s.

Evidence of large herbivores was measured and tested because of cases like the Cottonwood grazing allotment, where it was suspected that overgrazing of riparian areas was causing the alder mortality. However, no association was found between herbivore evidence, including cattle, and alder condition. Similarly, road effects, such as dust or soil compaction, were not associated with the damage.

It was surprising that damage was related neither to elevation nor to geographic area. Many factors that may be directly or indirectly related to the cause vary with

elevation, including basic factors such as precipitation and temperature. Similarly, the Continental Divide and latitude within the Southern Rocky Mountains affect many biological phenomena. Damage certainly did vary among transects, but no potential causal factors were more than weakly correlated with that variation.

One such weak association was distance to stream. Distance to stream was measured as a test of the hypothesis that drought played a role in alder condition. Genets farther from the stream would be more subject to reduction in height of the riparian water tables associated with drought. Although stream distance was one of the few variables that was significantly related to alder condition, the relationship was the opposite of that predicted by the drought hypothesis: alder condition improved slightly with distance from the stream. The correlation coefficient was very low, and it is difficult to interpret, but it does seem to argue against the drought hypothesis.

Other facts also suggest that drought is unlikely to be associated with this dieback and mortality. First, the drought that affected the Southern Rocky Mountains recently began in 2000 and in most areas seemed to end in 2005. As noted above, alder damage probably began in the late 1980s and certainly before 2000. Second, many of the alders are rooted immediately on the stream bank. Very few of the streams that support alder dried during the drought, so the plants have had their roots bathed in flowing water continuously through this and other droughts. Active dieback is common in alders even when rooted along full streams.

The other weak relationship found was that transects exposed to full sun had poorer alder condition (only when measured as TII) than those in full shade. The fact that this was not significant in the overall analysis with partial shade included, nor for GCI, means this result should be interpreted with caution. However, it suggests that solar exposure may exacerbate the factor(s) driving this epidemic.

In Oregon, naturally occurring cankers appeared to be associated with wounding by river ice during high spring flows (10). Inoculations of alder with *Cytospora* sp. were successful only when stems were wounded (11). This is consistent with the negative correlation between infection frequency and stream distance found in this study. However, much of the disease in this study was associated with small mountain streams where wounding by ice is unlikely. Also, many cankers observed during this study were initiated in the upper crowns, and even those that began directly on the stems were not associated with visible wounds. Infection biology may differ between the two areas because of different environments or different species of *Valsa* involved (see below).

The one factor consistently associated with dieback and mortality was Cytospora canker, caused by *Valsa melanodiscus* (anamorph *Cytospora umbrina*). This fungus is most commonly collected from *Alnus* species (18). Through Koch's postulates and cross-inoculation tests, *V. melanodiscus* was shown to cause cankers on alder in Colorado and to be host-specific with respect to other woody hosts tested and *Cytospora* species from those hosts (15). An earlier epidemic of Cytospora canker leading to dieback and mortality of thinleaf alder similar to that reported here began in Oregon in the late 1980s (11). Inoculations with alder isolates showed substantial pathogenicity to alder but not to *Cornus stolonifera* or *Crataegus douglasii*. One isolate from that study was recently identified as *Valsa nivea* (G. C. Adams, *personal communication*).

A later epidemic began in Alaska in about 2002, and the putative pathogen was identified as *V. melanodiscus* (20; G. C. Adams, *personal communication*). Thousands of acres were affected, and *Alnus sinuata* and *A. crispa* (species not present in the Southern Rocky Mountains) were affected to a lesser degree. Similar damage has been seen in British Columbia and Yukon Territory of Canada (L. Trummer, *personal communication*). Koch's postulates were recently completed in Alaska by field inoculations of *A. incana* subsp. *tenuifolia* with Alaskan isolates of *V. melanodiscus* (19).

Although *V. melanodiscus* is poorly known, the genus and related genera are generally not considered primary pathogens of vigorous trees. They generally cause significant damage when host defenses are compromised by stress, such as water potential below a critical threshold; defoliation; soil compaction; waterlogging of soil; winter injury to buds, twigs, or wood; spring frost injury; sun scald; fire injury; wounding; and off-site planting (2). Thus, although *V. melanodiscus* appears to be the proximate cause of this long-term episode of dieback and mortality, additional factors that led to the epidemic must be sought.

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