

# Disease-Mediated Declines in N-Fixation Inputs by *Alnus tenuifolia* to Early-Successional Floodplains in Interior and South-Central Alaska

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## ABSTRACT

Atmospheric nitrogen (N) fixation by *Alnus tenuifolia* can account for up to 70% of the N accumulated during vegetation development along river floodplains in interior Alaska. We assessed disease incidence and related mortality of a recent outbreak of fungal stem cankers on *A. tenuifolia* across three regions in Alaska during the 2005 growing season, and determined the impacts on N-fixation rates, nodule biomass, and stand-level N-fixation inputs. The highest percentage of ramets colonized or dead with canker was found on Tanana River plots, suggesting the epidemic is most severe in the Fairbanks region. A positive relationship between % basal area loss to canker and % canopy loss provides a simple means for assessing stand-level mortality associated with disease in the field. Although specific N-fixation (SNF) rates were not influenced by canker disease incidence of individual genets, live nodule biomass beneath alder canopies was inversely correlated with the per-

centage of ramets dead or with main ramet canker. Variations in SNF and live nodule biomass translated to differences in N-fixation inputs, which ranged from 22 to 107 kg N ha<sup>-1</sup> y<sup>-1</sup> across study regions. Nodule biomass was reduced by incidence of canker disease and related mortality an average of 24% across all sites, which translates to N input reductions of 8, 16, and 33 kg N ha<sup>-1</sup> y<sup>-1</sup> for the three regions, respectively. During the 2008 growing season, we resurveyed the Tanana River plots and found that of the ramets larger than 4-cm diameter having main ramet canker in 2005, 74% are now dead; and for those without main ramet canker in 2005, 25% have developed main ramet canker, and 8% are dead. Thus, it is likely that N-fixation inputs have declined further below what we estimated for 2005.

**Key words:** Alaska; alder; canker; disease; nitrogen cycling; nitrogen fixation; succession.

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## INTRODUCTION

Plant pathogens and invertebrate herbivores influence important pathways for carbon (C) and nitrogen (N) cycling in terrestrial ecosystems. This includes direct effects on plant growth, mortality, foliage chemistry, and canopy structure (Hansen and Goheen 2000; Tainter and others 2000; Erickson

and others 2004; Stadler and others 2006), and litter composition and decomposability (Waring and others 1987; Cobb and others 2006). Plant pathogens can also have strong indirect effects on nutrient cycling through effects on soil microclimate (Jenkins and others 1999), rates of herbivory and trophic structure (Faeth and Hammon 1997; Tingley and others 2002), throughfall chemistry (Lightfoot and Whitford 1990; Reynolds and Hunter 2001), and the quality and quantity of allochthonous inputs to aquatic systems (Snyder and others 2002). Pathogen and invertebrate herbivore impacts on foundation species often have the greatest consequences for changes in nutrient cycling processes because such hosts, by definition, play a prominent role in ecosystem structure and functioning (Ellison and others 2005). Although ecosystems are expected to be resilient to outbreaks of native herbivores and pathogens, invasive herbivores and pathogens are often associated with permanent shifts in ecosystem structure and function, including significant changes in nutrient cycling dynamics (Castello and others 1995; Ellison and others 2005; Jenkins and others 2007). However, climate change may alter the interactions between plants and their native pathogens and invertebrate herbivores (Malmström and Raffa 2000; Chakraborty and Datta 2003; Scherm 2004; Mulder and others 2008), resulting in regime shifts that permanently alter nutrient cycling dynamics. This may occur through direct effects of climate warming on a whole suite of factors. These include changes to insect or pathogen life cycles (Berg and others 2006), changes in plant vulnerability to infestation by altering the interactions between pathogens and invertebrate herbivores (Mulder and others 2008), or by affecting interactions between plant parasites and other disturbance regimes affected by climate change, such as fire (Parker and others 2006; Allen 2007).

Observations by the Alaska USDA Forest Service Forest Health Protection staff of widespread dieback and mortality of thin-leaf alder (*Alnus incana* ssp. *tenuifolia*; hereafter, *Alnus tenuifolia*) throughout south-central and interior Alaska has recently focused attention on the extent, potential causes and consequences for this mortality (USDA 2008). Dieback and mortality throughout Alaska has been most frequently associated with a fungal canker pathogen (putatively *Valsa melanodiscus*, anamorph *Cytospora umbrina*) which causes diffuse, longitudinally expanding cankers that eventually girdle the stem. There are many factors that may potentially interact to increase predisposition or severity of disease, including defoliation by the native (*He-*

*michroa crocea*) and invasive (*Eriocampa ovata*) alder sawflies, regional climate warming, moisture stress, and branch damage from frost, heavy snowloads, or river ice. South-central and interior Alaska experienced the hottest summer on record during 2004 (<http://www.wrcc.dri.edu/summary/Climsmak.html>), which reduced alder growth (Nossov 2008), and may have created stressful host conditions and a favorable environment for rapid disease development. In addition to stressful abiotic agents, several other opportunistic canker fungi have been found in association with diffuse cankers on *A. tenuifolia* (USDA 2008), and it is likely that a number of biotic agents are contributing to disease incidence or accelerating the rate of dieback and mortality. It is possible that canker diseases may be natural components of the long-term population biology of the host, evidenced by the spread of disease coincident with the recruitment of early-successional alder stands along the Tanana River basin over the past 20 years (Nossov 2008). However, the widespread increased incidence of canker on *A. tenuifolia* throughout both Alaska and the Rocky Mountain states (Worrall and Adams 2006; Worrall and others 2008; Worrall 2009) suggests that the epidemiology of the disease is more complex.

Given that up to 70% of the N accumulated during forest succession along Alaskan floodplains can be derived through atmospheric fixation by *A. tenuifolia* (Van Cleve and others 1971; Uliassi and Ruess 2002), we hypothesized that high disease incidence and associated mortality of alder would have significant effects on ecosystem N inputs derived from atmospheric fixation. Differences in N-fixation rates of individual *A. tenuifolia* plants at the regional scale are expected to be driven by variation in plant growth and N demand, and influenced by site conditions such as light, soil moisture, and soil nutrient availability, notably phosphorus (P) (Uliassi and Ruess 2002). Disease-related declines in N-fixation inputs at the stand-level could be manifested through reductions in nodule biomass resulting from ramet mortality, and/or decreases in N-fixation rate per unit nodule due to physiological down-regulation of nitrogenase activity (Ruess and others 2006). Such coupled effects would ultimately influence soil N accumulation, forest stand development, and forage quality for higher trophic levels (Butler and Kielland 2008).

This study had two objectives. The first was to assess the incidence of canker disease and disease-related mortality of *A. tenuifolia* across Alaska by surveying a set of sites throughout interior and south-central Alaska. The second objective was to

determine the impact of disease incidence and related mortality on N-fixation rates and stand-level N inputs derived from N fixation by *A. tenuifolia*.

## MATERIALS AND METHODS

### Study Sites

During the summer of 2005, permanent study plots were established in three regions spanning a latitudinal transect from the Kenai Peninsula to just south of Fairbanks, Alaska. River drainages where *A. tenuifolia* was a dominant or co-dominant species were selected for study within each region. These included Quartz Creek (Kenai region), Eagle River (Anchorage region), and Tanana River (Fairbanks region) (Table 1). At each location, three 20 × 20 m<sup>2</sup> plots were established within a 2–3 km stretch of the river.

Plots along Quartz Creek had the lowest stem density and basal area of *A. tenuifolia*, and were situated on older stable terraces above the river as evidenced by the maturity of scattered white (*Picea glauca* (Moench) Voss) and Lutz (*Picea × lutzii*) spruce, and Alaska paper birch (*Betula neoalaska* Sarg.). Willows (*Salix* sp.) and rose (*Rosa acicularis* Lindl.) were scattered amongst the plots, with horsetails (*Equisetum* sp.) and bluejoint reedgrass (*Calamagrostis canadensis*) forming a dense ground cover in partially shaded and open stands, respectively. Within the Eagle River drainage, *A. tenuifolia* grows in dense stands along low-lying terraces that are flooded during spring snowmelt. Stands selected for study were nearly pure alder, with *C. canadensis* forming a dense ground cover in open stands, and white spruce recruiting in more closed stands. Along the Tanana River south-west of Fairbanks, plots were located in early-successional

stands 15–25 years of age dominated by dense stands of *A. tenuifolia*. Scattered balsam poplar (*Populus balsamifera* L.) saplings are recruiting into these stands, and a number of herbaceous species constitute a ground cover where canopy gaps permit light penetration to the ground surface.

### Soils

Ten soil cores (2.54 cm diameter × 15 cm depth) were sampled from each of the nine plots across the three regions. Soils were sampled from beneath each shrub used for N-fixation determinations (see below) after removing the surface litter layer. Soils were stored in 2 mil plastic bags on ice for up to 3 days and processed within a day of being brought to the laboratory in Fairbanks. The top 15 cm of each core was homogenized by removing large roots and organic debris, passed through a 2-mm sieve, dried to constant weight at 45°C, and ground in a steel ball-mill. Soil pH was determined on a 2:1 (water/soil) extract, and total C and N were determined on a LECO CNS 2000 (Leco Corporation, St. Joseph, Michigan, USA). Total soil P was determined colorimetrically on an autoanalyzer following perchloric acid digestion. Available P was determined by extracting 10 g soil in 100 ml deionized water containing a fine-mesh nylon bag containing 4 g Bio-Rad AG 1-X8, 0.50 mol l<sup>-1</sup> NaHCO<sub>3</sub>-charged anion exchange resin, followed by 0.50 mol l<sup>-1</sup> HCL resin bag exchange and autoanalyzer analysis (Lajtha and others 1999).

### Vegetation and Canker Survey

*A. tenuifolia* genets grow as single or multiple main stems (ramets) arising at or just above the soil surface. Within each plot, the diameters of all

**Table 1.** Location, Climate and Structural Characteristics of the *A. tenuifolia* Canopy from Three Study Regions

Location	Quartz Creek 60°31.14'N 149°39.61'W	Eagle River 61°16.95'N 149°24.80'W	Tanana River 64°44.92'N 148°9.44'W
Mean annual precipitation (mm) <sup>1</sup>	546	417	267
Mean annual temperature (°C)	2.2	2.1	-2.7
Freeze free period (days) <sup>2</sup>	139	151	145
<i>A. tenuifolia</i>			
Basal area (m <sup>2</sup> ha <sup>-1</sup> )	43.7 ± 6.1b	110.1 ± 4.5a	96.9 ± 3.0a
Total stem density (stems ha <sup>-1</sup> )	7,356 ± 258b	17,222 ± 2,500a	17,942 ± 2,211a
Live stem density (stems ha <sup>-1</sup> )	5,031 ± 428b	9,170 ± 1,768a	13,220 ± 3,124a

<sup>1</sup>Western Region Climate Center (<http://www.wrcc.dri.edu/summary/Climsmak.html>).

<sup>2</sup>Growing season length is defined as the number of consecutive days when average minimum daily air temperature is above 0°C. Numbers followed by different letters within rows are significantly different at  $P \leq 0.05$ .

*A. tenuifolia* ramets greater than 1 m in height were measured at 1.37 m (DBH). Surveys were used to develop plot-level estimates of disease incidence (percentage of live ramets colonized) and disease-related mortality (% ramets dead with canker, % basal area dead with canker, % canopy death associated with canker). Canker incidence on each ramet, based on visible presence of conidiomata or ascomata (these forms of fruiting are difficult to distinguish in the field) and discrete canker margins, was quantified as follows. For ramets smaller than 4 cm diameter, ramets were scored as either with or without canker. For ramets larger than 4 cm diameter, main ramets and side branches were scored as either with or without canker. Disease-related mortality of ramets was quantified by scoring recently dead ramets in both size classes as either with or without canker. To accurately count individual ramets, plots were roped-off into  $5 \times 10$  m<sup>2</sup> sectors. Within each sector, the percent loss of leaf area due to canker-related ramet mortality was assessed visually, and values (0–100% canopy loss) were averaged across sectors to provide one plot-level estimate. Ramets without discernible canker colonization but with dead distal branches were scored separately. All ramets larger than 4 cm diameter were labeled with a uniquely numbered aluminum tag affixed with wire for later determination of the rates of disease-related mortality.

### N-Fixation Rate

Ten genets were selected within each plot for the determination of N-fixation rate. Because we wanted to assess how the incidence of canker disease was affecting N-fixation rate at the plant (genet) level, we arbitrarily selected individual genets that spanned the range of disease incidence within the genet (proportion of live ramets colonized) and disease-related mortality within the genet (proportion of dead ramets with canker) within the plot. N-fixation rate of each genet was measured using a <sup>15</sup>N<sub>2</sub> uptake method (Anderson and others 2004). Approximately 2.5 g of fresh nodule with attached fine roots were harvested from beneath each genet, placed in a 60 ml polyethylene syringe fitted with a septum, and buried in the litter layer to maintain ambient temperatures. Ten milliliter of 99 at.% <sup>15</sup>N<sub>2</sub> (Isotec Inc., Miamisburg, Ohio, USA) was then added to the syringe to produce an incubation atmosphere of 20.3% <sup>15</sup>N<sub>2</sub>. Immediately after <sup>15</sup>N<sub>2</sub> addition, a 15-ml sample of the incubation atmosphere was removed to provide a quantitative measure of atom

percent enrichment (APE) of <sup>15</sup>N<sub>2</sub> at time zero ( $T_0$ ). These samples were stored in 10 ml exetainers (Labco, High Wycombe, Buckinghamshire) before analysis using a dual-inlet isotope ratio mass spectrometer (PDZ Europa Scientific Instruments, Crewe, Cheshire). After the removal of the incubation atmosphere sample, the syringe containing the root nodules was immediately returned to the forest floor for 10 min. Nodules were then removed from the syringe and immediately frozen in liquid N<sub>2</sub>. Soil moisture (TDR, CS620 Hydrosense with 10 cm probes, Campbell Scientific, Logan, Utah) and temperature (digital thermometer, 7 cm depth) were measured at five locations beneath the canopy of each genet.

In the laboratory, nodules were thoroughly rinsed through a fine sieve of all adhering soil and organic material, dried for 48 h at 60°C, and ground using a Wig-L-Bug ball-mill (Reflex Analytical, Ridgewood, New Jersey) in preparation for mass spectrometry analysis. A nodule sample not incubated with <sup>15</sup>N<sub>2</sub> was used as a control for the determination of APE for each nodule sample according to the following equation:

$$\text{APE}_{\text{nodule}} = {}^{15}\text{N}_{\text{enrichednodules}} - {}^{15}\text{N}_{\text{controlnodules}}, \quad (1)$$

where both <sup>15</sup>N content measures are in at.%. By combining APE with total nodule N content, dividing by incubation time, and correcting for the composition of the initial incubation atmosphere as determined by mass spectrometry, we calculated the specific N-fixation activity of the nodule samples ( $\text{SNF} = \mu\text{mol N assimilated g}_{\text{DWT}} \text{ nodule}^{-1} \text{ h}^{-1}$ ) as follows:

$$\text{SNF} = \frac{(\text{APE}_{\text{nodule}} \times \% \text{N}_{\text{nodule}})}{(\text{incubation time (h)} \times \% {}^{15}\text{N}_{\text{atmosphere}})}, \quad (2)$$

where  $\% \text{N}_{\text{nodule}}$  is the mass percent N content of the enriched nodule sample and  $\% {}^{15}\text{N}_{\text{atmosphere}}$  is the atom percent <sup>15</sup>N content of the incubation atmosphere at the beginning of the assay.

The incidence of disease or disease-related mortality on each ramet and the proportion of leaf area lost from ramet mortality were assessed (as described above) for each tree sampled for SNF. Leaf punches from the recently-matured leaves were taken in the field from each genet for which SNF was measured, and analyzed for specific leaf area ( $\text{SLA} = \text{cm}^2 \text{ g}^{-1}$ ) after drying for 48 h at 40°C in the laboratory. Total N and  $\delta^{15}\text{N}$  of leaves were determined by mass spectrometry as described above, and total P was determined colorimetrically following perchloric acid digestion.

## Nodule Biomass and N Inputs

Because the density of individual *A. tenuifolia* genets and the number of ramets per genet varied significantly among regions, it was important to select a method for quantifying nodule biomass that could be applied across all plots. We first quantified average nodule biomass beneath alder canopies (g nodule biomass per m<sup>2</sup> canopy), and then scaled values to the stand-level by developing region-specific relationships between genet basal and canopy areas. Five individual genets that spanned a range of genet size, disease incidence, and disease-related mortality were selected for sampling at each plot. The level of disease incidence was an important criterion because preliminary sampling revealed a large number of dead nodules beneath genets with high incidence of disease and associated mortality. We predicted that estimation of the percent decline in live nodule biomass would be a useful way of estimating the extent to which canker infection negatively impacted whole stand N-fixation inputs. The canopy diameter of each genet was estimated, and three evenly spaced concentric bands (rings) were marked on the soil surface beneath the canopy. Multiple soil cores (15.2 cm diameter × 15.2 cm deep) were sampled from random locations within each band. More cores were sampled from the outer bands to sample approximately 10% of the surface area from each band. Soil cores were stored in plastic bags at 4°C for up to 2 days prior to sorting. Nodules clusters were washed free of soil and sorted into live (by 1 cm size classes) and dead biomass based on color. Unlike roots, live alder nodules are easy to identify due to their characteristic bright orange color, whereas dead nodules are black. Sorted nodules were then dried and weighed. Attenuation curves (decrease in biomass away from the central ramet cluster) were established for each genet, and relationships between basal area and nodule biomass were determined across all three plots for each region. Total nodule biomass (g m<sup>-2</sup>) was then estimated for each plot from values of basal area. N inputs through N<sub>2</sub> fixation at the plot level were determined by multiplying average SNF (across ten genets) and nodule biomass for each plot.

## Statistical Analyses

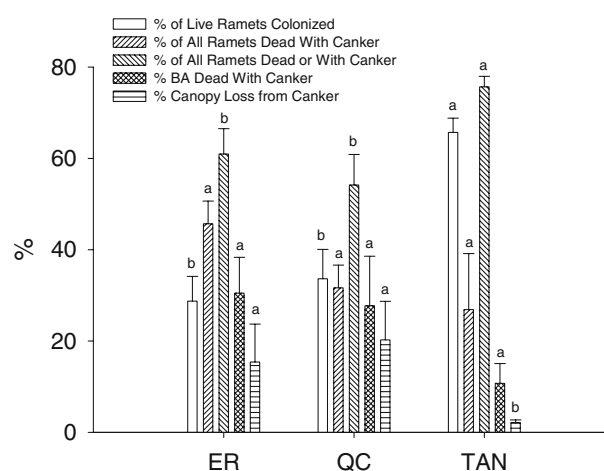
Variations among sites in site characteristics, disease incidence, canker-related mortality, and parameters related to N fixation were analyzed using ANOVA (PROC GLM) (SAS, 2002). Data were tested for normality and homogeneity of variances, and

square-root or log<sub>10</sub> (X + 1) transformed, or ranked and reanalyzed where necessary to meet ANOVA assumptions. Linear and nonlinear regressions were used to characterize relationships among plant growth traits, stand characteristics, and disease incidence and mortality. Statistical significance was determined at  $\alpha = 0.05$  for all the analyses, but marginally significant differences at  $\alpha = 0.10$  were also considered. Unless otherwise stated, data presented are means ( $\pm 1$  SE) of untransformed data.

## RESULTS

### Canker Survey

The percentage of ramets either colonized or dead with canker ranged from  $54 \pm 7\%$  at Quartz Creek to  $76 \pm 2\%$  at the Tanana River plots ( $F_{2,6} = 4.2$ ,  $P = 0.07$ ) (Figure 1). The incidence of canker disease on live ramets was also significantly higher along the Tanana River ( $66 \pm 3\%$ ) compared with plots at either Eagle River ( $29 \pm 5\%$ ) or Quartz Creek ( $34 \pm 6\%$ ). However, these differences were strongly influenced by the high disease incidence of the large proportion of smaller-diameter ramets, for which the typical diffuse canker disease symptoms were more difficult to characterize than for larger ramets. If we only consider the percentage of live ramets larger than 4 cm diameter with main ramet canker, values averaged  $17 \pm 3$ ,  $18 \pm 5$ , and  $7 \pm 2\%$  at Quartz Creek, Eagle River, and Tanana River, respectively ( $F_{2,6} = 3.1$ ,  $P = 0.12$ ). Similarly, the proportion of all ramets larger than 4 cm diameter dead with canker averaged  $27 \pm 11$ ,



**Figure 1.** Incidence of canker disease and disease-related mortality within each study region. Bars represent mean  $\pm 1$  SE for each category across three replicate stands within each region. Bars within categories sharing similar letters are not significantly different at  $P \leq 0.05$ .

29 ± 9, and 6 ± 2% at the three sites, respectively ( $F_{2,6} = 2.8$ , ns). Across sites, the percentage of these large ramets (>4 cm diameter) that were dead with canker accounted for 55% of the variation in total dead basal area ( $P < 0.05$ ) and 94% of variation in the percentage of total basal area dead with canker ( $P < 0.0001$ ).

We established a relationship between canopy death and dead basal area which also shows higher disease-related mortality for Eagle River and Quartz Creek. At Quartz Creek, Eagle River, and the Tanana River, the percentages of basal area dead with canker averaged 28 ± 11, 30 ± 8, and 11 ± 4% ( $F_{2,6} = 1.7$ , ns), and the percentage of canopy loss associated with canker averaged 20 ± 8, 15 ± 8, and 2 ± 1% ( $F_{2,6} = 1.9$ , ns) at the three sites, respectively. The interrelationship between these two metrics provides a simple means for predicting disease-related basal area loss (%DEADBA) from %canopy loss (%CANLOSS), a parameter that can be rapidly assessed in the field (%DEADBA =  $1.07 \times \%CANLOSS + 3.03$ ;  $r^2 = 0.84$ ,  $P < 0.0001$ ). A complete characterization of disease incidence and disease-related mortality by diameter class for the three sites shows that the proportions of size classes larger than 4 cm diameter that were dead with canker tended to be higher for Eagle River and Quartz Creek relative to Tanana River plots (Figure 2).

## N Fixation

N-fixation rates at Quartz Creek ( $5.52 \pm 1.23 \mu\text{mol N g}^{-1} \text{h}^{-1}$ ) were significantly less than those along the Eagle River ( $8.57 \pm 0.78 \mu\text{mol N g}^{-1} \text{h}^{-1}$ ) and Tanana River ( $9.75 \pm 1.05 \mu\text{mol N g}^{-1} \text{h}^{-1}$ ), which did not differ ( $F_{2,7,8} = 5.05$ ,  $P < 0.05$ ). SNF was positively correlated with soil temperature across sites ( $r^2 = 0.12$ ,  $P < 0.001$ ,  $n = 90$ ), but unrelated to other soil chemical characteristics or plant traits (data not shown). Data on soil chemical characteristics and *A. tenuifolia* leaf traits from the three study regions are shown in Supplementary Material: *Soil and Leaf Chemistry*, and can be found on the Bonanza Creek LTER website (<http://www.lter.uaf.edu/>).

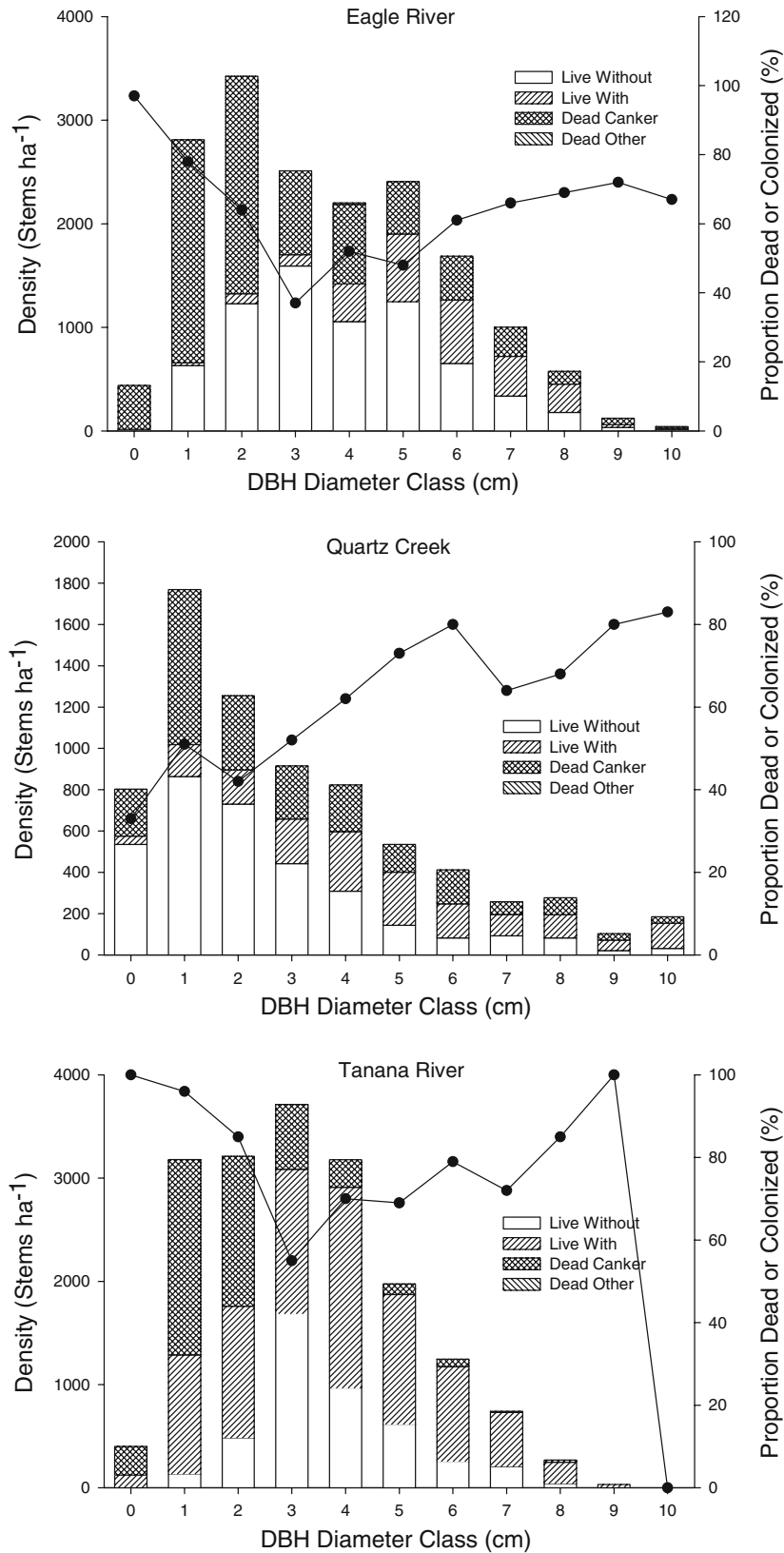
Incidence of canker disease and related mortality varied substantially among the 90 genets measured for N-fixation rate across sites, and the relationship between % basal area dead and % canopy loss generated for these genets (Figure 3) was similar to that from plot-level measurements mentioned above. Despite this variation in disease incidence and related mortality, we found no evidence that N-fixation rate per gram nodule of individual genets was influenced by the incidence of disease or

related mortality of those genets. In other words, variation in SNF among sites or replicate stands within sites could not be attributed to variation in disease-related mortality (% basal area dead with canker, or % canopy dead with canker) or disease incidence (% live ramets colonized) (ANOVA data not shown). Neither did we detect any effect on SNF by woolly alder sawfly damage, which was only present at Eagle River, but where leaf area loss from sawfly among genets sampled for N fixation averaged  $18 \pm 5\%$  (range 1–75%).

## Nodule Biomass

Live nodule biomass beneath *A. tenuifolia* canopies averaged  $28.0 \pm 5.9$ ,  $22.0 \pm 4.4$ , and  $36.4 \pm 2.0 \text{ g m}^{-2}$  at Quartz Creek, Eagle River, and Tanana River sites, respectively (Figure 4A). The proportion of nodule biomass dead at Tanana River ( $37.7 \pm 3.5\%$ ) and Eagle River ( $42.9 \pm 6.4\%$ ) was significantly greater than that at Quartz Creek ( $25.6 \pm 3.4\%$ ) ( $F_{2,33} = 3.66$ ,  $P < 0.05$ ). Tanana River sites had significantly higher ratios of canopy nodule biomass to basal area relative to either of the other sites, for ratios expressed as either total ( $F_{2,33} = 3.67$ ,  $P < 0.05$ ) or live components ( $F_{2,33} = 3.14$ ,  $P < 0.05$ ). There were also notable differences in the size distributions of live nodules among regions, with Tanana River sites having significantly higher proportion of nodules in smaller size classes relative to the other two regions (Figure 4B).

By design, individual genets chosen for nodule biomass sampling had a wide range of incidence of canker disease and related mortality both within and among sites. For example, the coefficient of variation in % basal area dead, % canopy loss, and % incidence of main ramet canker all averaged over 100% among the 12 genets sampled within each region. Across all sites, live and dead nodule biomass were correlated with a number of measures of canker disease incidence and related mortality, suggesting that higher incidences of the disease led to declines in live nodule biomass at all sites. For example, genets showing increasing percentages of basal area dead with canker also tended to have more dead nodule biomass as a fraction of total nodule biomass ( $r^2 = 0.10$ ,  $P = 0.09$ ). Similarly, increased canopy loss associated with canker (%) tended to be inversely correlated with live nodule biomass ( $\text{g m}^{-2}$ ) across regions ( $r = -0.30$ ,  $P = 0.08$ ). The most useful relationship for estimating the negative effects of canker disease on nodule biomass was the inverse correlation between live nodule biomass (LIVENOD,  $\text{g m}^{-2}$ )



**Figure 2.** Density and levels of stem canker incidence and related mortality (*bars*), and the % of all ramets that are dead or colonized (*dots*) by ramet diameter classes measured during 2005 for each of the three study regions. DBH diameter classes are incremented by centimeter, beginning with "0" (= 0–1 cm) and ending with "10" (= > 10 cm).

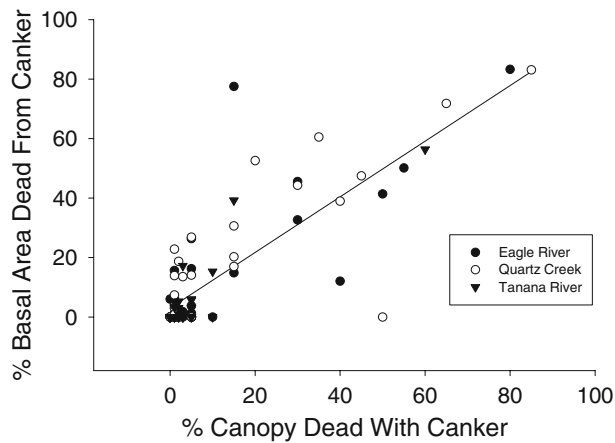


Figure 3. Relationship between the % of basal area dead with canker and the % of canopy leaf area dead associated with canker disease for individual shrubs measured for N<sub>2</sub>-fixation rate at replicated stands within each of the three study regions (*n* = 30 shrubs/region).

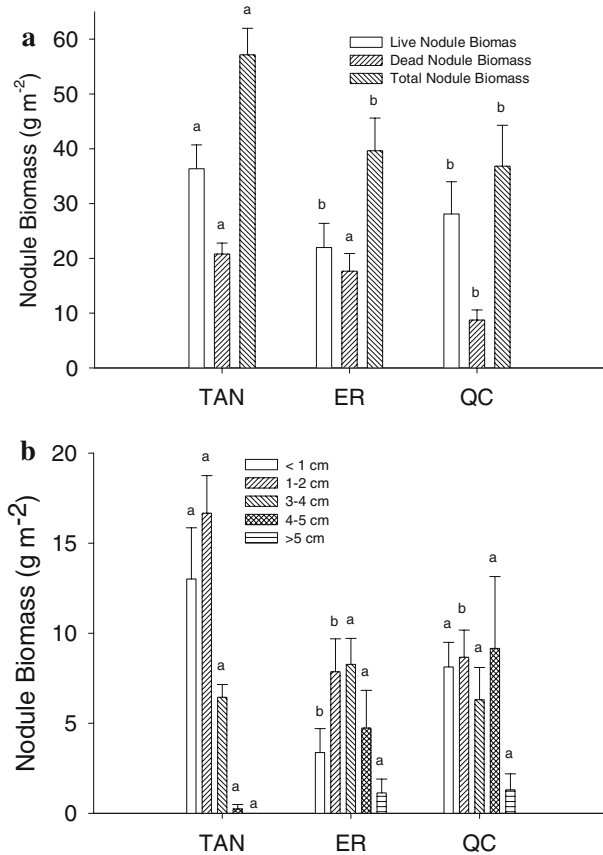


Figure 4. Live and dead biomass (A) and biomass by size class (B) of nodules beneath *A. tenuifolia* canopies at replicated stands within each of the three study regions (*n* = 15 shrubs/region). Statistical notations follow Figure 1.

and the percentage of ramets dead or with main ramet canker (CANK) ( $r^2 = 0.21, P < 0.01$ ):

$$\text{LIVENOD} = 36.35 \times e^{(-0.0147 \times \text{CANK})}$$

### N-Fixation Inputs

We developed negative exponential relationships between canopy area (m<sup>2</sup>) and basal area (cm<sup>2</sup>) of individual shrubs for each region, which when multiplied by total stand basal area produced averages of 4854 ± 677, 7407 ± 300, and 9923 ± 309 m<sup>2</sup> alder canopy cover per hectare for Quartz Creek, Eagle River, and Tanana River stands, respectively. This allowed stand-level estimates of live nodule biomass, which averaged 146 ± 74, 164 ± 43, and 359 ± 31 kg live nodule biomass per hectare for the three stands, respectively. We then estimated daily rates of N input for each of the nine stands as the product of N-fixation rates and stand-level nodule biomass, assuming fluxes were constant over a 24-h period, as we have observed (R.W. Ruess, unpublished data) and has been shown elsewhere at high latitudes (Weisz and Sinclair 1988; Huss-Danell and others 1992). Daily rates of N input, which averaged 0.24 ± 0.10, 0.46 ± 0.11, and 1.16 ± 0.19 kg N ha<sup>-1</sup> day<sup>-1</sup> for Quartz Creek, Eagle River, and Tanana river, respectively, were extrapolated to the growing season using a simple step function of plant growth and N fixation based on our previous studies (Uliassi and Ruess 2002; Anderson and others 2004). This function assumes that N-fixation rates are half-maximal from 20–31 May, maximal from June 1 to August 15, half-maximal for the last 2 weeks of August, and quarter-maximal for the first 2 weeks of September. This generated N-fixation inputs values of 22 ± 9, 42 ± 10, and 107 ± 17 kg N ha<sup>-1</sup> y<sup>-1</sup> for Quartz Creek, Eagle River, and Tanana River sites, respectively.

To determine the extent to which canker disease incidence and related mortality has impacted N-fixation inputs, we predicted what live nodule biomass would have been in the absence of canker disease from region-specific negative exponential relationships between LIVENOD and %CANK (see above). Estimates suggest that live nodule biomass per m<sup>2</sup> canopy has been reduced 24.4, 25.3, and 22.5% by the canker disease and associated mortality at Quartz Creek, Eagle River, and Tanana River stands, respectively. Such declines in nodule biomass would translate to N input reductions of 8, 16, and 33 kg N ha<sup>-1</sup> y<sup>-1</sup> for the three regions, respectively. Disease-free stands at Quartz Creek, Eagle River, and Tanana River would be predicted

therefore to be accumulating 30, 58, and 140 kg N ha<sup>-1</sup> y<sup>-1</sup> through N fixation, respectively. We view the translation of these values to declines in N-fixation inputs to these stands as conservative, because the percentages of dead nodule biomass that we found far exceed values we remember seeing a decade ago when we conducted similar N input estimations along the Tanana River.

## DISCUSSION

### Spread of Canker on Thin-Leaf Alder in Interior Alaska

Our data show that the spread of fungal stem canker disease in *A. tenuifolia* is both recent and widespread throughout interior and south-central Alaska, and has led to significant ramet mortality and associated declines in ecosystem N inputs derived from N fixation. Although there are no previous records of canker epidemics on *A. tenuifolia* in Alaska, *V. melanodiscus* is most likely native to interior Alaska, because *V. alni* (a synonym for *V. melanodiscus*) is listed from old Alaskan fungal collections (Cash 1953; Connors 1967). One of the most intriguing questions is whether the high disease incidence and widespread canker-related ramet mortality that we documented is new to interior Alaska, or is simply a return of a natural epidemic that has occurred either regularly or episodically for centuries. If similar epidemics have occurred previously, then legacies of periodic interruptions to N-fixation inputs during early-successional development may potentially be found in growth or chemical signatures of annual rings from cohorts of long-lived species (such as white spruce) recruited at that time. However, if the widespread disease and mortality of *A. tenuifolia* is a recent phenomenon, then associated declines in N-fixation inputs may have novel implications for successional and landscape development that will ultimately depend on the rate of alder reestablishment.

The unusually hot and dry summer climate throughout Alaska during the current decade may have contributed to the recent increase in incidence and severity of canker disease on *A. tenuifolia*. Interactions between drought and fungal diseases are a growing concern, particularly throughout Europe and North America where climate warming has led to unusually warm and dry conditions over the past decade (Desprez-Loustau and others 2006; Bigler and others 2007; Worrall and others 2008). Predisposition by water stress may involve both improved growth conditions for the fungi in water-stressed plants or a decrease in active disease

resistance when plants are water stressed (Guyon and others 1996; Desprez-Loustau and others 2006). *Cytospora* canker fungi are characteristically opportunistic pathogens that attack stressed or damaged hosts, and water stress is thought to play an integral role in the initiation and development of *Cytospora* canker diseases of hardwoods. Drought stress and wounding are often combined with experimentally initiate infection (Bloomberg and Farris 1963; Filip and others 1992; Kepley and Jacobi 2000), and critical levels of bark moisture content have been correlated with increased susceptibility to *Cytospora* canker fungi (Bloomberg 1962). Worrall and others (2008) reported that drought was likely an important driver of *Cytospora* canker contributions to mortality of trembling aspen throughout the Rocky Mountain States. Drought has similarly been implicated in facilitating *Cytospora* canker-induced dieback of *A. tenuifolia* throughout Oregon, Washington, and Colorado (Filip and others 1992; Worrall and Adams 2006).

Branch damage, severe defoliation, and interactions with other pathogens may also contribute to disease initiation or disease severity. Throughout Alaska, *A. tenuifolia* often suffers extensive branch damage during winter when heavy snow can flatten even the largest shrubs, particularly after ramets have accumulated heavy layers of frost (personal observation). Wounds are classically known to create infection courts for canker pathogens, and natural modes of stem wounding or branch damage have been implicated in canker-related ramet mortality in *Alnus incana* and *Alnus glutinosa* (Moricca 2002). It also appears that stress induced by defoliator outbreaks from native and non-native sawflies may have contributed to the canker spread along the Eagle River, the only region showing any sawfly damage in this study. For example, among the shrubs sampled for nodule biomass along the Eagle River, the proportion of canopy defoliated by sawfly was positively correlated with the proportion of the canopy dead from canker ( $r^2 = 0.74$ ,  $P < 0.001$ ). Such dramatic changes in host condition may allow disease to develop rapidly. One possibility is that a shift in host physiology can result in disease development by releasing a quiescent pathogen from latency, a relatively long period of host colonization without damage to the host (Stanosz and others 2000). Several *Cytospora* species have previously been isolated from asymptomatic hosts (Chapela 1989; Adams and others 2005), suggesting the potential for vigorous hosts to impose latency. Disease progression can also be facilitated by weakly parasitic fungi, which have characteristics that allow quick invasion of

weakened host tissue and can facilitate disease progression (Kehr 1992). Although the ability of *V. melanodiscus* to incite cankers on *A. tenuifolia* is confirmed (Stanosz and others 2008), other closely-related fungi have been found in association with diffuse cankers and dieback on alders in Alaska (USDA 2008). The role of biotic and abiotic factors in the initiation and development of the diffuse canker disease and dieback process is currently being investigated.

Although we found lower canker-related mortality along the Tanana River, the higher incidence of disease in younger-aged ramets suggests that demographics of the local population may play a role in disease development. Tanana River stands also had a much higher proportion of root nodules in smaller size classes, suggesting there is a more rapid turnover of nodules along the Tanana River. Higher nodule turnover rates could also simply be a function of the higher relative growth rate of *A. tenuifolia* along the Tanana River relative to the other two regions, as suggested by higher leaf N, thinner leaves, higher N-fixation rates, and higher apparent allocation to nodules in the Tanana River plots. However, the average contribution of young nodule clusters ( $\leq 1$  cm diameter) to total live nodule biomass (32.8%) in this study was notably greater than for similarly sized alders sampled along the Tanana River in 1994 (24.0%) prior to the canker outbreak (Uliassi and Ruess 2002). That previous study found that 20.3% of nodule biomass was in clusters 3 cm diameter or larger, whereas we found only 1% of nodule biomass in these older size classes, suggesting that nodule lifespan has declined appreciably since the increased incidence of disease and associated widespread mortality.

One explanation for the high disease incidence within early-successional stands along the Tanana River and Eagle River is simply the high density of alder ramets in these stands. A positive correlation between host density and disease incidence is typical of many plant fungal diseases (Gilbert 2002), and often results directly from increasing pathogen-host encounter probabilities. Additional indirect effects include density-dependent physiological plant responses that increase host vulnerability to infection, and the effects of increased host density on improving environmental conditions favorable to the pathogen (Burdon and Chilvers 1982; Burdon and others 1989; Gilbert 2002). Disease incidence and related mortality was positively correlated with stand density across all stands we surveyed in 2005 (Figure 5), suggesting that canker epidemics may be facilitated by periodic alder recruitment events.

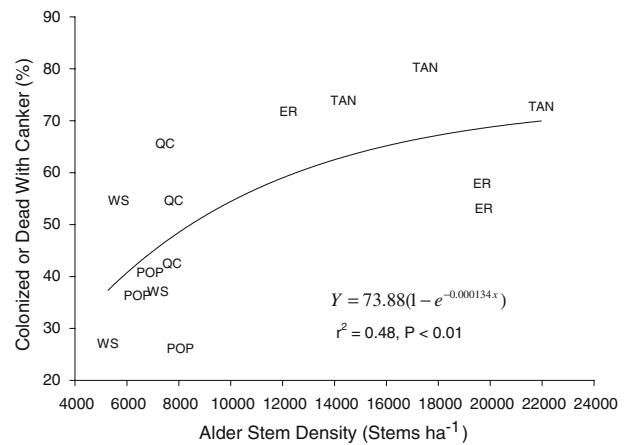


Figure 5. Relationship between the proportion of *A. tenuifolia* ramets either dead or colonized by stem canker at replicate plots along the Eagle River (ER), Quartz Creek (QC), and Tanana River (TAN). Additional mid-successional (balsam poplar, BP) and late-successional (white spruce, WS) stands ( $n = 3$  stands per successional stage) along the Tanana River where *A. tenuifolia* dominates the understory shrub canopy were surveyed for canker incidence and associated mortality during 2005 using the same methods outlined above.

### Effects of Canker on N-Fixation Inputs

We estimate that during the 2005 growing season, canker disease incidence and canker-related mortality reduced N-fixation inputs by 8, 16, and 33 kg N ha<sup>-1</sup> y<sup>-1</sup> within alder stands along Quartz Creek, Eagle River, and the Tanana River, respectively. Live nodule biomass scaled inversely with the percentage of ramets dead or live with main ramet canker, resulting in disease-related reductions in live nodule biomass per m<sup>2</sup> canopy of 24.4, 25.3, and 22.5% at the three study regions, respectively. N-fixing plants typically up- or down-regulate nodule production in response to factors affecting plant growth and plant N demand. Growth and allocation to nodules in response to ratios of available soil N to P are well described (Wall and others 2000, 2003; Uliassi and Ruess 2002; Binkley and others 2003; Valverde and Wall 2003; Jia and others 2004; Gokkaya and others 2006), but fewer studies have examined nodule mortality in response to stress in natural stands. Increased mortality and turnover of nodule biomass has been shown for neotropical leguminous trees that are managed with pruning (Nygren and Ramirez 1995), similar to the response of fine production and lifespan to aboveground browsing (Ruess and others 1998). There is also some evidence that nodule biomass decreases and nodule lifespan increases with stand age (Sharma and Ambasht

1986; Pearson and Vitousek 2001). Although we previously found no significant effects of defoliation on allocation to nodule biomass in greenhouse-grown *A. tenuifolia* seedlings (Ruess and others 2006), increased levels of sawfly defoliation among plots along the Eagle River were associated with increased levels of canker-related ramet mortality in this study. Given the modular growth of multiple ramets arising at the ground surface from a single genet of *A. tenuifolia* growing in the field, it is logical that root and nodule dieback would scale to the proportion of genet basal area death related to canker, and differ from the response of single-ramet seedlings grown in the greenhouse.

Our estimates of potential N-fixation inputs to alder stands unaffected by canker disease along the Tanana River ( $140 \text{ kg N ha}^{-1} \text{ y}^{-1}$ ) are similar to previous studies conducted within the same region. Using a mass balance approach accounting for all vegetation and soil to a depth of 1 m, Van Cleve and others (1971) reported an average total system N accretion of  $156 \text{ kg N ha}^{-1} \text{ y}^{-1}$  (range  $75\text{--}362 \text{ kg N ha}^{-1} \text{ y}^{-1}$ ) for alder thickets at 5, 15, and 20 years from initial surface establishment. Uliassi and Ruess (2002) estimated N-fixation inputs of  $59 \text{ kg N ha}^{-1} \text{ y}^{-1}$  for *A. tenuifolia* stands with stem densities (mean  $6,667 \text{ stems ha}^{-1}$ ) and basal areas (mean  $20.5 \text{ m}^2 \text{ ha}^{-1}$ ) that were less than half those of this study. Our estimate of a 24% decline in N-fixation inputs during 2005 by *A. tenuifolia* along the Tanana River is a maximal value, because it assumes that all dead nodule biomass resulted from canker-induced ramet mortality. However, our experience is that dead nodules are relatively rare in early-successional alder stands.

### Consequences for Ecosystem Function and Landscape Change

The influence of N-fixing shrub encroachment on biodiversity and ecosystem function is a topic of considerable interest and concern (Vitousek and Walker 1989; Hibbard and others 2001; Caldwell 2004; Rice and others 2004; Baer and others 2006; Hughes and others 2006). Yet the consequences for loss of native N fixers on ecosystem function are poorly documented. Mass accumulation estimates suggest that up to 70% of ecosystem N accumulated over the Tanana floodplain 200-year successional sequence is fixed by *A. tenuifolia* within the first 30 years of succession (Van Cleve and others 1971, 1991). Paleoecological records indicate that watershed biogeochemistry and aquatic trophic dynamics were substantially influenced by alder

expansion throughout southern and northwestern Alaska during the early- to mid-Holocene (Oswald and others 1999; Hu and others 2001).

During the 2008 growing season, we resurveyed the Tanana River plots used for this study and characterized the incidence and related mortality from canker disease on all ramets, including those larger than 4 cm diameter, all of which were individually tagged in 2005. Of the ramets larger than 4 cm diameter having main ramet canker in 2005, 74% are now dead; and for those without main ramet canker in 2005, 25% have developed main ramet canker, and 8% are dead. Moreover, recruitment of new individuals is nonexistent within alder thickets (data not shown). Thus, it is highly likely that N-fixation inputs have declined significantly below what we estimated for 2005. Increase in disease incidence and disease-related mortality have also continued, but at a slower pace, in mid-successional balsam poplar stands (data not shown), where *A. tenuifolia* forms an understory less-dense than in the younger alder stage (Viereck and others 1993). However, vegetative propagation from downed ramets (principally snowfall induced) is extensive in these stands, ostensibly due to higher light availability. Additional data suggest that much of the young, sparsely-distributed *A. tenuifolia* recruiting on newly-formed sandbars (<10 years of age) also has lower levels of disease incidence and related mortality than that was found for dense alder thickets described in this study (Nossov 2008). Thus, although the dense alder thickets along the Tanana River appear to be dying out, it remains to be seen whether *A. tenuifolia* in less-dense stands can persist through the current canker disease epidemic.

How the loss of dense alder stands will influence plant growth and successional development along river terraces throughout interior Alaska is of course unknown. A complete die-out of thin-leaf alder would clearly have serious consequences for long-term ecosystem productivity. However, as mentioned above, we are uncertain whether the current epidemic represents a threshold shift in N cycling dynamics (Chapin and others 2006), or merely a return of an infrequent co-occurrence of events. Because alder has strong effects on P mobilization and cycling (Giardina and others 1995; Binkley and others 2000; Mitchell and Ruess submitted), even a temporary loss of alder will likely have effects on biogeochemical cycling beyond the immediate influence on soil N stocks. Analogs for how the loss of alder might influence ecosystem structure and function can be found along interior Alaskan river corridors where low

moose population densities increase the ratio of willows to alder (Butler and others 2007). We are currently studying the influence of alder on biogeochemistry and stand development at a regional scale of varying moose densities to understand the implications for permanent disturbance-mediated shifts in alder abundance.

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