REVIEW / SYNTHÈSE

Predicting landscape patterns of aspen dieback: mechanisms and knowledge gaps

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Abstract: Sudden dieback and deterioration of mature aspen stands is commonly observed throughout North America. This dieback process has tremendous ecological and economic importance, yet remains poorly understood. This paper summarizes our understanding of aspen dieback in North America, identifies potential processes that contribute to reduced vigour and dieback of aspen stands, and examines the scales (stand, ecosite, regional) at which these processes operate. Many factors including pathogens, nutrition, or successional changes may be involved in the decline of aspen vigour and thereby contribute to the dieback process. However, insect defoliation, drought, and thaw–freeze events appear to be the most likely factors initiating dieback in mature aspen stands. Further study is clearly needed to elucidate the mechanisms and landscape patterns of dieback. Information needs related to identifying processes and modeling landscape patterns of dieback are indicated.

Résumé : Le dépérissement soudain et la dégradation des peuplements matures de peuplier faux-tremble sont couramment observés un peu partout en Amérique du Nord. Ce dépérissement a une énorme importance écologique et économique; pourtant il demeure mal compris. Cet article résume notre compréhension du dépérissement du peuplier faux-tremble en Amérique du Nord, identifie les processus qui pourraient contribuer à la perte de vigueur et au dépérissement des peuplements de peuplier faux-tremble et examine l'échelle (peuplement, aire écologique, région) à laquelle opèrent ces processus. Plusieurs facteurs incluant les agents pathogènes, la nutrition et les changements du stade de succession pourraient être responsables de la perte de vigueur du peuplier faux-tremble et contribuer par conséquent au processus de dépérissement. Cependant, les facteurs plus probablement à l'origine du dépérissement dans les peuplements matures de peuplier faux-tremble semblent être la défoliation par les insectes, la sécheresse et les épisodes de gel et dégel. D'autres études seront évidemment nécessaires pour élucider les mécanismes du dépérissement et son comportement à l'échelle du paysage. Il serait utile de combler les besoins d'information reliée aux processus d'identification et à la modélisation du comportement du dépérissement à l'échelle du paysage.

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Introduction

The sudden dieback of mature aspen (*Populus tremuloides* Michx.) stands is a widely observed phenomenon throughout North America. With the onset of dieback, aspen stands often deteriorate rapidly, leading to tree death and stand breakup. In fact, apparently healthy stands of mature aspen may be reduced to only a few dying stems in as little as 3–6 years (Shields and Bockheim 1981; Peterson and Peterson 1992). The dieback process thereby represents a major transitional phase in the life of aspen stands with important implications. Ecologically, the dieback and breakup of aspen stands add snags and coarse woody debris to the site, and the opening of the canopy results in opportunities for other species to dominate. Furthermore, because of its wide distribution and use as a fibre source, early dieback of aspen has tremendous economic importance in many regions of North America.

The dieback of entire cohorts has been observed in other species (Mueller-Dombois 1987), but dieback is particularly intriguing in aspen because it is clonal and capable of regenerating itself through root suckers. Sucker regeneration is common in the understory of stands experiencing dieback (Schier 1975), and this may ensure that aspen retains control of the site after a dieback event. Indeed, this self-replacement process may be responsible for delaying succession to conifers (Cumming et al. 2000). However, some declining stands show little regeneration, which can increase dieback of the root system and favour succession to conifers

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(Shepperd et al. 2001). Aspen dieback with limited regeneration will thus reduce the vigour of the clone and its persistence on the landscape.

Although aspen decline has long been a concern (e.g., Basham 1958; Stoeckler 1960; Schier 1975; Shields and Bockheim 1981), recent episodes of dieback in northeastern Ontario (Candau et al. 2002) and western Canada (Hogg et al. 2002*a*; Brandt et al. 2003) have heightened interest in the dieback problem. There is also a concern that the warmer and drier conditions predicted for much of western North America over the next few decades could lead to significant increases in aspen dieback and mortality (Hogg and Hurdle 1995; Hogg et al. 2002*a*). Several recent studies have correlated dieback with factors such as age, extreme weather (drought, thaw–freeze events), defoliation, and (or) the activity of pathogens (Candau et al. 2002; Hogg et al. 2002*a*; Brandt et al. 2003). Still, the critical processes and mechanisms driving aspen dieback remain poorly understood (Hogg 1999), limiting our ability to explain the highly variable mortality rates evident among different stands (Peterson and Peterson 1992). An improved understanding of these driving processes is thus necessary for improved growth and yield modeling and for predicting patterns of dieback across the landscape.

The objectives of this review paper are to summarize information on current patterns of aspen dieback in North America and identify the processes that contribute to aspen stand decline. We synthesize this information within a stand-level conceptual model of the dieback process, identifying gaps in our knowledge. Finally, we consider approaches for modeling dieback at the landscape level and highlight information needs for predicting patterns of aspen dieback across the landscape.

Patterns of aspen dieback

By dieback, we are referring to the phase in the life of mature aspen stands wherein stands experience (often rapid) aboveground death of the dominant and codominant trees, a process typically leading to breakup of the stand. An important aspect of the dieback process in aspen is the recognition that rather than being individuals, stems are ramets interconnected by a common root system (DesRochers and Lieffers 2001). This clonal characteristic is a likely factor in the synchronized dieback and breakup of aspen stands. Yet, while deterioration and death are obvious in the clone's ramets, the root system typically survives (at least in part) to regenerate the clone.

Typically, stand dieback begins with the death of branches in the upper crowns and proceeds downwards. As trees (ramets) begin to die, gaps begin to form in the canopy and the remaining trees are subjected to increased wind, sunlight, and evaporative stresses, which are thought to hasten decline (Peterson and Peterson 1992). Indeed, once this process begins, deterioration typically proceeds rapidly in as little as 3–4 years in western Canada (Peterson and Peterson 1992). As an example of this rapid dieback, in fewer than 2 years over 50% of aspen stems died in some stands around Batoche, Sask., and other parts of the western aspen parkland (T. Hogg, unpublished data). Similarly, in the Great Lakes region, rapid dieback and breakup has been observed to occur over a 6-year period (Shields and Bockheim 1981). Nonetheless, dieback can be more protracted, as for example in the Rocky Mountain region, where dieback can occur over a number of decades (Shepperd and Engelby 1983).

Aboveground dieback is often associated with lower root densities (Schier 1975; Shepperd et al. 2001) and increased abundance of dead roots (DesRochers and Lieffers 2001). Root dieback is typically concentrated in openings between trees (Schier 1975), although the remaining live trees may maintain parts of the root system connected to dead trees through grafts or parental root connections (DesRochers and Lieffers 2001). Extensive root dieback is observed in Rocky Mountain stands, where death of the overstory is a more drawn-out process (Shepperd and Engelby 1983).

Patterns and timing of dieback at the landscape or regional levels are not well documented (Peterson and Peterson 1992; Pothier et al. 2004); however, the timing of dieback does appear to vary considerably across aspen's North American range. On average, decline occurs at an earlier stand age in the Great Lakes region, around 60 years (Shields and Bockheim 1982), as compared with around 80 years in eastern Canada (Kneeshaw and Bergeron 1998) and around 100 years in the western US interior (Schier 1975; Mueggler 1989). Some stands in the Rocky Mountain region are known for their great longevity (Perala 1991), with stand ages of at least 276 years in favourable high-elevation sites (McKenzie 2001). Little information is available on the longevity of stands in western Canada (Peterson and Peterson 1992). The regional variation in stand longevity across North America suggests a climatic influence over dieback. Indeed, dieback appears to occur earlier in regions with higher mean annual temperature (Shields and Bockheim 1981) or drier climates (Hogg and Hurdle 1995). While regional differences are notable, even more striking is the high degree of variability amongst stands within the same climatic region. For example, Schier and Campbell (1980) found stands in Utah as young as 49 years undergoing decline, whereas stands as old as 128 years were still healthy. Indeed, it is common to find healthy clones in proximity of clones undergoing dieback, despite being of a similar age (Schier 1975; Peterson and Peterson 1992; Hogg and Schwarz 1999). The fact that tree longevity varies considerably over its North American range and amongst adjacent stands strongly suggests factors other than age are important drivers of dieback.

Processes involved in aspen dieback

Identifying the factor(s) that drive forest decline is made difficult by the multitude of stress agents and complexity of their interactions (Auclair et al. 1992; Pedersen 1998). Describing the process of aspen dieback is made even more difficult by the weak documentation of dieback patterns (rates, timing, landscape patterns) in aspen. In the following discussion, we identify the drivers that appear to be most fundamental to the dieback process in aspen. We have organized these processes into sections based on their role as inciting, contributing, or predisposing factors, as described by Manion (1991). Briefly, inciting factors are considered the primary instigators of decline, severely weakening trees and providing opportunities for secondary, contributing factors to become established. Predisposing factors represent chronic stresses that increase vulnerability to inciting factors. This

Table 1. List of predisposing, inciting, and contribution factors (sensu Manion 1991) involved in the decline of aspen forests in North America.

Factor	Dominant spatial scale	Dominant time scale	Reference
Predisposing			
Climate	Landscape–region	Long-term	Hogg and Hurdle 1995
Successional processes	Stand	Decades	Lieffers et al. 2001; Shepperd et al. 2001
Ecosite	Stand	Long-term	Stoeckler 1960; Shields and Bockheim 1981
Forest structure	Stand	Decades	Mueller-Dombois et al. 1983
Age	Stand	Decades	Brandt et al. 2003
Local problems	Stand–landscape	Variable	Karnosky et al. 1999
Clonal aspects	Tree-stand	Long-term	Schier and Campbell 1980; Kneeshaw and Bergeron 1998
Inciting			
Drought	Landscape–region	<5 years	Hogg et al. 2002*a*
Insect defoliation	Landscape–region	<5 years	Candau et al. 2002; Hogg et al. 2002*a*
Thaw–freeze events	Landscape–region	<1 month	Cayford et al. 1959; Cox and Malcolm 1997
Wildlife stem damage	Stand–landscape	<5 years	DeByle 1985
Contributing			
Insect borers	Tree–stand	<5 years	Ives and Wong 1988
Pathogens	Tree–stand	<5 years	Hiratsuka 1987
Windthrow	Tree–landscape	<1 day	Webb 1989

organizational approach reflects our effort to identify the functional roles that different factors play in the process and how they contribute to observed patterns of dieback (see Table 1).

Inciting factors

Inciting factors exert sudden stresses on tree function and provoke a physiological response. Furthermore, they can be considered the primary instigators of decline, because their activity reduces vigour substantially, thereby enhancing opportunities for secondary agents to become established. In this way, inciting factors are critical to our understanding of the dieback problem.

Severe drought events

Severe episodes of drought have been implicated in the decline and death of aspen trees in western Canada (Zoltai et al. 1991; Hogg et al. 2002*a*; Powell 2003). The effects of severe drought on aspen are related to moisture conditions in the soil and (or) the atmosphere. Reduced stomatal conductance occurs in aspen under declining soil water availability (Dang et al. 1997; Roth et al. 1997; Wan et al. 1999), a process likely controlled by hormones produced in the roots during soil moisture stress (Lambers et al. 1998). This response typically occurs as soil water becomes depleted, as happens under conditions of extended drought. However, high soil moisture conditions with low oxygen availability (Landhäusser et al. 2003) or cold soil temperatures (Landhäusser and Lieffers 1998) can also limit water uptake (Wan et al. 1999; Landhäusser et al. 2002) and create conditions of moisture stress.

Atmospheric drought stress is driven by vapour pressure deficit (VPD, i.e., the decline in water vapour pressure from the leaf mesophyll cells to that of the air surrounding the leaves as influenced by humidity and wind-controlled boundary layer conditions (Oke 1987)). In aspen, increasing VPD also leads to reductions in stomatal conductance (Dang et al. 1997; Hogg and Hurdle 1997; Hogg et al. 2000). As a consequence, photosynthesis is reduced (Hart et al. 2000; Hogg et al. 2000), which may lead to depletion of carbohydrate reserves and could potentially increase the risk of subsequent dieback and mortality (Hogg 1999). In aspen, the observed reduction in stomatal conductance under high VPD likely represents an adaptation to maintain leaf and stem water potential above a critical threshold to avoid xylem cavitation (Hogg et al. 2000), i.e., an embolism in the xylem vessel. Under severe and prolonged water stress, cavitation can effectively destroy the ability of xylem vessels to transport water to the crown (Tyree and Sperry 1988; Sperry et al. 1991). While aspen can withstand greater amounts of xylem tension than poplars adapted to floodplain conditions (Blake et al. 1996), aspen will undergo cavitation under severe stress (Sperry et al. 1991). As water must be pulled to great heights in trees, the greatest tension will be experienced in the upper branches. It is likely for this reason that dieback is usually first observed in the upper crown of aspen trees; the dieback of upper branches of mature cottonwood during drought has been related to xylem cavitation (Rood et al. 2000). It is also possible that rapid change in crown density due to the loss of some trees might increase vulnerability to branch embolism (Cochard et al. 1999) in the remaining trees.

We expect that the intensity of drought episodes and their impacts on aspen health vary across a number of different scales. Climate conditions and the frequency of severe droughts vary widely across North America, possibly contributing to regional episodes of dieback. Further, stands with certain structure (e.g., high slenderness) or occupying certain topographic positions (e.g., exposed, xeric sites) may be more negatively impacted by episodes of severe drought, thereby driving local variation in dieback across the landscape. These factors that predispose certain regions, sites, and stands to drought are considered below under the section predisposing factors.

Other extreme weather events

Other extreme weather events such as late winter thaw–freeze (damage to buds and roots), winter Chinook winds in the west (desiccation), severe spring frost (damage to foliage), or spring snowstorms (crown breakage) can weaken aspen stands. Growing season (leaf-on) snowstorms in the southern boreal forest can cause crown breakage, especially in taller, slender trees (Gill 1974). Severe spring frosts in 1958 in southern Manitoba and Saskatchewan injured and killed buds, reducing leaf area development and radial growth in aspen (Cayford et al. 1959). Similar thaw–freeze events were associated with transient growth reductions in aspen stands in Alberta (Hogg et al. 2002*a*). Rapid freezing events such as growing season frosts can also damage the photosynthetic function of developed leaves (e.g., Lamontagne et al. 1998).

Late winter thaw–freeze events were considered important drivers of forest dieback episodes in Europe and North America (Pomerleau 1991; Auclair et al. 1992). Studies on birch have shown that thaw–freeze events can cause both xylem cavitation and root damage, rendering roots incapable of generating the pressure necessary to refill cavitated xylem (Cox and Malcolm 1997). However, while thaw–freeze events are considered a triggering mechanism in other species, their role in aspen dieback has only been speculated (Hogg et al. 2002*a*). Still, considering that most fine root growth of aspen in colder climates occurs in late summer or fall and must over winter (Landhäusser and Lieffers 2003), fine roots may be particularly susceptible to thaw–freeze events or deep frost penetration. Since snow cover helps insulate roots from extreme winter temperatures, root dieback in aspen could be a problem in years with poor snow cover or in landscape positions that do not accumulate snow.

Defoliation

Severe defoliation events reduce carbon (C) uptake (Hart et al. 2000) and were considered a significant contributor in recent episodes of dieback in western Canada (Hogg et al. 2002a) and Ontario (Candau et al. 2002). Common defoliators of aspen are tent caterpillars (*Malacosoma* spp.), large aspen tortrix (*Choristoneura conflictana* (Walker)), and to a lesser degree the bruce spanworm (*Operophtera bruceata* (Hulst)) and poplar leaf miners (*Phyllonorycter* spp.) (Jones et al. 1985; Peterson and Peterson 1992). In Canada, the forest tent caterpillar *Malacosoma disstria* Hübner is the primary defoliator of trembling aspen (Ives and Wong 1988). Cyclical outbreaks of 3–6 years duration typically occur in eastern and western Canada every 10 years on average, and widely scattered areas of defoliation can coalesce to form outbreaks as large as 350 000 km^2 (Sippell 1962; Hildahl and Campbell 1975). In the western US, the western tent caterpillar (*Malacosoma californicum* (Packard)) is the most serious defoliator, accounting for episodes of defoliation from Mexico to Washington (Jones et al. 1985). The large aspen tortrix is not generally as damaging as forest tent caterpillar; outbreaks tend only to be 2–3 years in duration, but they often precede outbreaks of forest tent caterpillar. Both the tent caterpillars and large aspen tortrix may significantly diminish radial growth during the defoliation period (Ives and Wong 1988).

While defoliation does not typically cause immediate mortality (Ghent 1958; Kulman 1971), it is clearly a stress (Kosola et al. 2001). Defoliation intensity can vary considerably from light defoliation that thins foliage to complete defoliation (Hildahl and Campbell 1975). This represents a large C cost in terms of lost photosynthesis and in energy reserves necessary to refoliate (typically in the same season) (Ives and Wong 1988). Short episodes of defoliation (1 year) or partial defoliation events have been found to have only a small influence on C reserves in *Populus deltoides* Marsh. (Reichenbacker et al. 1996). In fact, enhanced water relations of residual foliage and compensatory photosynthesis can partially offset the loss of leaf area to defoliation (Reichenbacker et al. 1996; Hart et al. 2000). Multiple defoliations, however, result in severely depressed radial growth and carbohydrate depletion (Duncan and Hodson 1958; Hildahl and Reeks 1960; Jones et al. 1985; Kosola et al. 2001; Hogg et al. 2002*a*); in years of complete defoliation very narrow and white annual rings with reduced density are produced, likely because of reduced lignin deposition (Hogg and Schwarz 1999; Hogg et al. 2002*b*). The continued allocation of reserves to regrowth of leaves instead of fine roots may not leave sufficient energy for optimum root function, thus nutrient uptake (Kosola et al. 2001) and growth increment can be diminished for years after an outbreak (Hildahl and Reeks 1960). Severe episodes of defoliation may result in branch dieback (Hildahl and Campbell 1975) or, in extreme cases, direct mortality where complete defoliation occurs for 4 or more years (Churchill et al. 1964; Hildahl and Campbell 1975; Jones et al. 1985). Under continued severe defoliation, root carbohydrate reserves are depleted (Landhäusser and Lieffers, unpublished data), impairing aspen's ability to produce new leaves and roots, repair tissues, or produce protective chemicals, thereby increasing the likelihood of stand decline. Indeed, there does appear to be a strong relationship between defoliation history and mortality at a later time, partly owing to an increased susceptibility to stem damage by insect and fungal disease following severe defoliation events (Churchill et al. 1964; Hogg et al. 2002*a*).

Landscape patterns of defoliation for tent caterpillars have not been well described, but differences in climate or forest fragmentation may contribute to outbreaks (see Predisposing factors).

Stem damage by wildlife

Aspen stands are often prone to damage by wildlife, including elk, moose, bear, porcupines, and small rodents (Debyle 1965; Peterson and Peterson 1992). Common mechanisms of damage by wildlife include chewing, clawing, and antler rubbing. In western North America, mature stands can be severely damaged by elk feeding on aspen bark, especially in wintering grounds (DeByle 1985). We suspect that wildlife damage is generally less important than drought or defoliation as an inciting factor in the boreal forest. However, wildlife damage is the most critical inciting factor of aspen dieback in the cordilleran forests of western North America, especially in areas with high elk populations. Thus, wildlife impacts may be an important consideration when making comparisons of aspen dieback patterns among different regions of North America.

Contributing Factors

Contributing factors represent secondary agents that become important following an inciting event. These predominantly consist of wood-boring insects and fungal pathogens that establish on weakened trees. Even so, some organisms typically considered secondary, for example armillaria root rot, could play a more primary role in decline (Brandt et al. 2003), as highlighted below.

Wood-boring insects

A number of stem borers affect aspen, including *Agrilus liragus* Barter & Brown, *Sthenopis* spp., and the poplar borer, *Saperda calcarata* Say. Species of *Agrilus* are particularly associated with trees already under stress by drought (Ives and Wong 1988). *Saperda calcarata* is the most common borer in the prairies provinces of Canada and is particularly troublesome in the aspen parkland. Trees are often attacked repeatedly, and while not usually killed, are structurally weakened and thus become susceptible to wind breakage (Peterson and Peterson 1992). The holes made by borers provide entry courts for diseases such as hypoxylon canker (Graham and Harrison 1954; Peterson and Peterson 1992) and further damage by woodpeckers searching for borers may increase opportunities for infection (Ives and Wong 1988).

Fungal pathogens

The frequent incidence of decay fungi in aspen stands, particularly compared with other species in northern mixedwoods (Webb 1989), suggests they have an important role in aspen stand decline. *Phellinus tremulae* (Bondartsev) Bondartsev & Borisov in Bondartsev and *Armillaria* spp. are primarily responsible for the decay of roots, butts, and trunks of aspen (Peterson and Peterson 1992). As stands age, there are large increases in incidence of heart rot and loss of volume to decay (Basham 1958; Weingartner and Basham 1985; Pothier et al. 2004), making aspen increasingly prone to wind or snow breakage (Hiratsuka 1987; Webb 1989). The mode of infection is not well understood, but stems mechanically damaged by weather events or by animal injury may facilitate infection (Hiratsuka 1987). Branch stubs resulting from natural pruning, and perhaps root damage resulting from wind, also provide entry points for infection (Basham 1958).

Armillaria root and butt rots are widespread and are associated with declining aspen stands in western North America (Hinds 1985), the Great Lakes region (e.g., Banik et al. 1995), and western Canada (Brandt et al. 2003). *Armillaria* attack the roots and butts of trees, spreading vigorously amongst trees by networks of rhizomorphs. Species of *Armillaria* are generally considered to be weakly pathogenic (Frantz et al. 1998), killing less vigorous aspen by destroying their roots (Peterson and Peterson 1992). Yet it has also been suggested that *Armillaria* may play a more primary role in aspen decline by attacking healthy trees (Brandt et al. 2003). Although they are known to cause growth reductions and mortality in other species (Mallett and Volney 1999), their role in initiating dieback remains uncertain, in part because of limited information about armillaria, particularly host–substrate relationships (Frantz et al. 1998).

Cankers represent another group of pathogens and include such species as *Cenangium*, *Ceratocystus*, and *Cryptosphaerium* in the western US. (Hinds 1985) and *Nectria*, *Cytospora*, and *Hypoxylon* in western Canada and eastern North America (Hinds 1985; Hiratsuka 1987). Canker-causing fungi produce toxins that are able to breakdown tissues, although infection typically occurs through wounds (Graham and Harrison 1954; Hinds 1985). Consequently, cankers are thought to be most common in stressed or damaged trees (Hiratsuka 1987). Hypoxylon canker (*Hypoxylon mammatum* (Wahl.) Mill.) is one of the most aggressive cankers, especially in the prairies and Great Lakes region (Hinds 1985; Hiratsuka 1987). Unlike most canker species, it can be a significant agent of aspen mortality (Hiratsuka 1987), with apparently vigorous trees being susceptible to infection (Anderson and Anderson 1968). This fungus can rapidly girdle infected trees, causing mortality within 3–7 years (Anderson and Anderson 1968; Hiratsuka 1987). Relationships between site and stand characteristics and pathogen incidence could contribute to local variation in dieback (see Predisposing factors).

Windthrow

Death of aspen trees often results from wind breakage or uprooting. Wood strength has a strong effect on susceptibility to wind damage, and aspen, because of its low wood strength, is highly susceptible relative to other species (Webb 1989). Windstorms can damage or directly kill healthy aspen trees through breakage or uprooting (Ghent 1958; Webb 1989; Kneeshaw and Bergeron 1998). However, wind damage is most commonly associated with trees already weakened by other agents. Hypoxylon canker weakens stems, which may then be broken by wind at the infection point (Anderson and Anderson 1968). Webb (1989) noted that most wind-broken stems were infected by *Phellinus* sp., whereas uprooted trees were infected by *Armillaria* sp.

Predisposing factors

The following section describes factors that may predispose (sensu Manion 1991) aspen stands to dieback. These chronic stresses and (or) stresses that gradually increase with stand development (Mueller-Dombois 1987) reduce vigour over the longer term and impair aspen's ability to withstand inciting events such as severe drought or defoliation.

Regional climate

Climatic variation, particularly moisture, across North America creates regional differences in the amount of water stress and drought. For example, the southern portions of the boreal forest in Alberta experience greater moisture stress (decreased soil water availability and higher VPD) than more northerly or high elevation sites (Hogg 1994). Chronic moisture stress is expected to be the most important climatic factor reducing C uptake, thereby increasing vulnerability to dieback in continental regions. We may therefore expect a delay in dieback in more northerly or high-elevation sites, provided that water uptake is not strongly limited by cold soil temperatures (Hogg 1994). Annual variations in weather may also affect outbreaks of defoliators. Mild winters and warm weather during spring and early summer appear to favour forest tent caterpillar, whereas cold winters and cooler summers appear more favourable to large aspen tortrix (Ives 1981). Nonetheless, efforts to identify climatic explanations for spatial patterns of defoliator outbreaks such as forest tent caterpillar have been unsuccessful (Cooke and Roland 2000).

Succession

Successional processes likely play an important role in aspen dieback (Lieffers et al. 2001). Over the course of stand development, colder soils associated with shading and increasing forest floor thickness may be expected to limit root growth and diminish resource acquisition. At the same time, declining nutrient availability associated with slower decomposition, declining pH, and retention of nutrients in stand biomass may increasingly impair the vigour of stands (Ryan et al. 1997). Furthermore, as aspen stands develop, they show a rapid increase in leaf area (Pinno et al. 2001), peaking at 15–25 years, followed by a long slow decline (Lieffers et al. 2002). This allows a gradual increase in understory shrubs or conifers (spruce or fir), which may compete for resources, reduce soil temperatures, and contribute to decline of the aspen. Indeed, understory vegetation represents an increasing proportion of total biomass in declining stands (Schier and Campbell 1980). Conifers may be especially important in the process of stand dieback (Shepperd et al. 2001), because as they grow in height, they will increasingly compete for light with the aspen. Canopies of understory spruce or fir may also contribute to increasing drought stress by interception and sublimation of precipitation before it reaches the root systems of trees (Kimmins 1997). Maturing aspen stands with understory spruce were reported to have less biomass and productivity than similar stands without understory spruce (MacPherson et al. 2001). Furthermore, the gradual dieback of aspen likely favours the establishment of conifers or tolerant shrubs (such as hazelnut or green alder) rather than sucker establishment by aspen, although some stands are self-perpetuating by suckering without major disturbance (Cumming et al. 2000). Aspen is also noted to establish by suckering in canopy gaps (Paré and Bergeron 1995). We hypothesize, however, that gradual closing of dense stands of spruce or fir will eventually kill the aspen clones, as suckers could not survive in the low light transmitted by these conifer canopies.

Ecosite characteristics

It is highly likely that the impact of inciting events such as drought will vary among different types of ecosites. Aspen appears to grow best in mesic soil moisture conditions; either too little or too much soil water greatly inhibits aspen stomatal conductance and root growth (Kozlowski 1997; Landhäusser et al. 2003). It is clear that aspen does not grow well on some sites: sandy soils with poor water-holding capacity or fine textured soils with hardpans that restrict rooting depth (and possibly impede drainage) (Stoeckler 1960); xeric sites that drain water away (Shields and Bockheim 1981); sites that accumulate excessive amounts of water (Landhäusser et al. 2003) or slopes exposed to strong light and high VPD. Thus, it is reasonable to expect that soil texture, slope–aspect, slope position and drainage will, to some degree, influence clonal vigour and vulnerability to dieback (Shields and Bockheim 1981; Candau et al. 2002).

Nutritional limitation could contribute to declines in vigour and stand productivity (Ryan et al. 1997) and thereby hasten aspen senescence on poorer sites (Loehle 1988; Yao et al. 2001). In Minnesota, life spans of aspen stands appear lower on poorer sites with low nutrient availability (Voigt et al. 1957), raising speculation that availability of exchangeable bases may affect the timing of stand dieback (Voigt et al. 1957; Stoeckler 1960). Shields and Bockheim (1981) implicated Ca and K availability in the longevity of aspen stands. Sites low in exchangeable Ca were more susceptible to early decline, likely in part because vigorous growth is dependent on a substantial supply of Ca (Alban 1982). High K availability, however, was associated with reduced longevity, perhaps because K may interfere with Ca uptake (Johansen et al. 1968 in Shields and Bockheim 1981).

Finally, Basham (1958) suggested that decay incidence might be related to site moisture conditions, although others could not find any correlations with site (Wall 1971; Kemperman et al. 1978). Relationships between pathogen incidence and site quality may be confounded by genetic differences, as the incidence of decay commonly varies amongst clones occupying the same site (Wall 1971; Kemperman et al. 1978; Weingartner and Basham 1985).

Stand structure

Stand characteristics such as stem density, slenderness coefficient, and height are also likely to affect vulnerability to inciting stresses, particularly wind events and drought. First, to lift water to their crowns, tall trees must overcome greater gravitational forces than short trees. Second, dense stands develop stems with high slenderness coefficients (stem height:diameter). These slender trees are able to remain upright and limit their bending in wind because of their mutual support and the fact that dense canopies will shield individual trees from the full force of the wind. As these stands mature, however, loss of any canopy trees will result in greater wind flow into the canopy. This will result in two factors. First, greater bending stresses exerted on the stem may cause xylem damage and reduced hydraulic conductivity, resulting in water stress in the foliage (Fredericksen et al. 1994; Liu et al. 2003). Second, there will be more light and wind penetration into canopies, reducing humidity (i.e., increasing VPD) around the foliage, which in turn will reduce photosynthesis, as described above in the section on severe drought events. Mueller-Dombois et al. (1983) noted that during dieback events in even-aged stands, the smallest diameter trees, and presumably the most slender, were most likely to die.

Stand structure can also affect the spread of pathogens, as sparse stands are more likely to be infected by hypoxylon canker (Pitt et al. 2001). Cankers appear more prevalent on stand edges than their interiors (Anderson 1964; Anderson and Anderson 1968) and cause greater tree death in thinned stands (Anderson and Anderson 1968). This is likely related to the effects of stand density on conditions affecting spore dispersal (Anderson and Anderson 1968) or the activity of dispersal agents such as insects and birds (Ostry and Anderson 1995; Pitt et al. 2001).

Local human impacts

Numerous local problems can affect stand health and increase the risk of aspen decline. For example, point sources of pollution such as emissions from smelters (e.g., Lozano and Morrison 1981) or regional ground-level ozone concentrations (e.g., Karnosky et al. 1999) may negatively affect leaf function or increase soil toxicity. Livestock grazing can damage aspen stands through soil compaction and physical injury to trees (Peterson and Peterson 1992), thereby reducing stand vigour. Also, it has been suggested that forest fragmentation can enhance defoliation episodes by disrupting regulation of tent caterpillars by parasitoids and pathogens (Roland 1993).

Clonal characteristics

Numerous studies (e.g., Hogg and Schwarz 1999; Schier 1975; Schier and Campbell 1980; Shields and Bockheim 1981) have noted the distinctly clonal nature of stand dieback, observing large differences in health among clones of similar ages. Kneeshaw and Bergeron (1998) observed that death of aspen trees tended to be a group phenomenon and speculated that group death was related to the clonal establishment of aspen. Furthermore, large differences in the incidence of damaging agents such as decay (Wall 1971) or hypoxylon canker (Copony and Barnes 1974) are observed between different clones. These studies suggest that clones, rather than individual stems, are the unit of decline and that clones may differ greatly in their susceptibility to stresses or disease. First, it must be remembered that clones share a common root system, and root reserves and hormones can be moved from one individual to another. Second, all of the ramets in a clone should respond similarly to stress. Variation in phenology may play a role in clonal susceptibility to frost, thaw–freeze events, or defoliation through its affects on timing of leaf flush, abscission, or leaf chemistry. Differences in depth of rooting between clones may create differences in clonal moisture stress. Differences in the ability to produce protective chemicals or to repair (heal) or seal off tissues damaged by mechanical abrasion or wounding may affect susceptibility to infection by decay organisms. Differences in the stomatal control of water loss may affect susceptibility to drought and cavitation. Differences in C storage between clones might ensure that some clones are better able to recover from defoliation, drought, and cavitation events. Genetic differences may act directly or indirectly to limit the damage of the killing agent. For example, genetic differences could confer resistance to hypoxylon canker directly or indirectly through resistance to the insect borers that create the entry courts for infection (Copony and Barnes 1974). If dieback is indeed a clone-level phenomenon, then the size and distribution of clones will affect the pattern of dieback across a site.

Synthesis and conceptual model of dieback

In the following discussion, we present a synthesis of the important dieback processes and propose a conceptual model of aspen dieback at the level of individual clones or stands. We then suggest an approach that could be implemented for assessing the future risk of aspen dieback at the landscape level.

Stand (clone) level model of aspen dieback

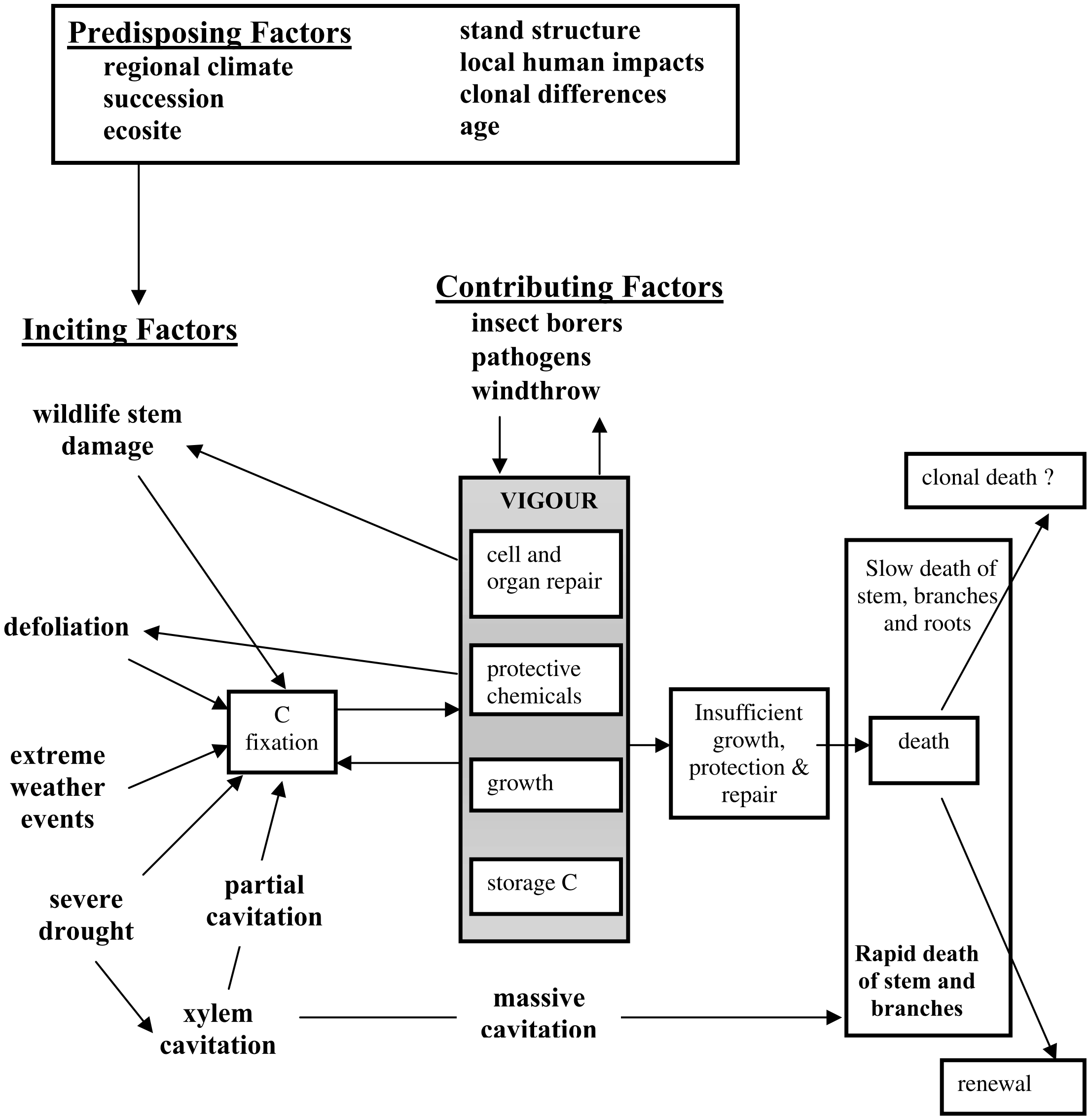
According to Waring (1987), mortality occurs when a tree is no longer capable of producing or mobilizing sufficient resources to repair damaged tissues or sustain life. Based on this definition, it is reasonable to expect that factors that reduce production and storage of C contribute to the dieback process. Furthermore, stresses that exert sudden and severe impacts on the C balance of the ramet or clone, i.e., the partial or complete loss of photosynthesis and (or) depletion of storage C, are most likely to lead to dieback. Severe depletion of C reserves will limit the clone's ability to recover, as reserves are necessary for leaf flush and root growth (Bonicel et al. 1987; Nguyen et al. 1990; Johansson 1993; Kobe 1997; Landhäusser and Lieffers 2003).

As summarized by the conceptual model (Fig. 1) and the preceding discussion, we suggest the primary factors inciting dieback are drought, defoliation, extreme weather events, and wildlife stem damage. We suspect that defoliation and drought are the most critical, as they severely impact C production and C reserves (for repair and defense), especially where they persist over several growing seasons. We expect contributing factors such as decay fungi and windthrow are in most cases secondary agents of dieback, increasing in importance as weakened trees are unable to mobilize sufficient C resources to repair damage or produce defensive chemicals. These factors in turn increasingly diminish the vigour of the clone and ultimately facilitate the death of the stand.

The speed at which decline occurs is likely dependent upon the severity (duration, intensity) of stress from the primary inciting factors. We suggest that severe episodic events (such as drought, defoliation, and perhaps thaw–freeze events) are most likely to drive rapid dieback. In other cases where the dieback process is more prolonged (e.g., Shepperd and Engelby 1983), wildlife and insect damage and fungal pathogens, which progress over longer periods of time, will play a more fundamental role. The speed at which these factors promote dieback is likely to affect the ability of clones to regenerate. We hypothesize that rapid dieback is favourable to the maintenance of a vigorous clone, since the sudden loss of apical dominance will favour sucker production and rapid redevelopment of leaf area, which can in turn support the clonal root system. Conversely, slow dieback that maintains apical dominance while gradually depleting carbohydrate reserves will inhibit sucker formation, thereby favouring establishment by other species and dieback of the clonal root system (Shepperd and Engelby 1983).

There remain fundamental gaps in our understanding of aspen's ecophysiological responses to different factors. There is little information, for example, on the degree to which rapid dieback may be caused by drought-induced cavitation. We also suspect that fine root damage resulting from extreme winter freezing could also drive rapid dieback by reducing water and nutrient uptake. This could be especially significant if the frost-induced damage of roots is followed by drought in the subsequent growing seasons. We also suspect that site and successional conditions that impact growth may affect vulnerability to other stress factors such as drought. Site factors such as soil water-holding capacity, rooting depth, and soil nutrient status may be important; however, field-based information is needed to determine whether predictions of dieback would be improved significantly through their inclusion. Similarly, information is needed on the effects of stand composition and structure on soil temperature and moisture regimes (e.g., through inter-

Fig. 1. Conceptual model of aspen dieback at the stand (clone) level as governed by carbon (C) production and allocation.



ception of rain and snow by understory conifers) and the subsequent influence on the rate of aspen growth and dieback. For older mixedwood stands, the impact of conifers on light interception by the aspen component is also a factor to be considered (Lieffers et al. 1996).

To improve our understanding of the physiological mechanisms driving dieback, we need to address the following questions:

(*i*) To what degree does cavitation occur in aspen trees under soil or atmospheric drought stress?

(*ii*) Are fine roots susceptible to severe frost or thaw–freeze events, and could this be a cause of aboveground dieback?

(*iii*) How is the incidence of damage by forest pathogens affected by climate and defoliation history?

(*iv*) Does the presence of an understory component in aspen stands lead to increased risk of aspen dieback, e.g., through changes in soil temperature and moisture?

(*v*) Is the onset and severity of aspen dieback affected by differences in soil type and ecosite conditions (e.g., texture and nutrient status) across a landscape?

(*vi*) Can tree characteristics, such as slenderness coefficient and height, be used to predict dieback?

Landscape model of stand dieback

We expect that it should be possible in the future to gain sufficient spatial information about the different dieback factors to run a landscape model. Modeling would involve assembling spatially referenced databases of defoliation history and climatic observations to create a model driven by drought and defoliation impacts within a geographic information system. The variation in climate across the landscape would be estimated through a spatial interpolation procedure that includes the influence of elevation using a digital elevation model (DEM) at the appropriate resolution (e.g., Price et al. 2000). The interpolation of climatic factors on a daily time step, in combination with the annual defoliation records, could be used as input for an ecophysiological model of aspen forest responses (e.g., Hogg 1999) that could be periodically updated to assess the areas at greatest risk of future dieback. Such a model could also be used to simulate impacts of severe frost, spring snowstorms, and thaw–freeze events. This may not be feasible in the near future, however, given the volume of input data required for simulations of many DEM grid points across large areas.

A simpler approach, limited to assessing potential moisture stress across a landscape or region, is to calculate a climatic moisture index (CMI, e.g., Hogg 1994) from spatially interpolated values of mean monthly temperature, precipitation, and solar radiation. This approach could be extended using a fine resolution DEM and a simple solar radiation model (e.g., McKenney et al. 1999) to estimate the variation in CMI that arises from differences in slope, aspect, and elevation across the landscape. The incidence of dieback across the landscape could then be related to spatial variation in the CMI in combination with a measure of defoliation frequency and severity. Alternatively, some other measure of moisture stress might be used. For example, Candau et al. (2002) used Drought Code records from the Canadian Forest Fire Weather Index to assess moisture stress spatially.

If it were found that ecosite-level differences influence susceptibility to dieback, then these aspects could be incorporated into a more sophisticated version of the landscape model. Slope position could be mapped across a landscape using the same fine resolution DEM that is used for modeling spatial variation in incident solar radiation, as described above. Variation in ecosites and (or) soil characteristics would require detailed information from field surveys that is still not available in most locations. Further refinements to the model could involve the inclusion of landscape fragmentation (to account for its role in enhancing insect defoliator activity) (Roland 1993) and stand structural aspects to account for the role of tree height, bole slenderness, and density in affecting vulnerability to drought. However, finding appropriate data to implement and validate such a model across large areas would pose a significant challenge. Also, it should be noted that some important factors, such as damage by wildlife and fungal pathogens, are difficult to include in a landscape model because they cannot be easily quantified and mapped across large scales.

Finally, one approach for evaluating our understanding of aspen dieback would be to develop and conduct field evaluations of a landscape model of aspen dieback (or index of dieback) based on the critical drivers identified above. Such a field study might be most efficiently undertaken in a complex mountainous environment, where elevation, temperature, and moisture vary over small distances.

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