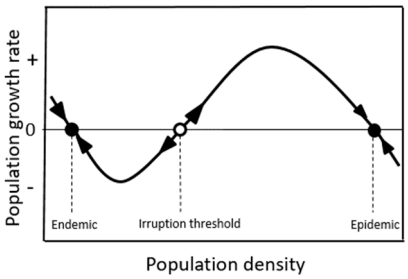
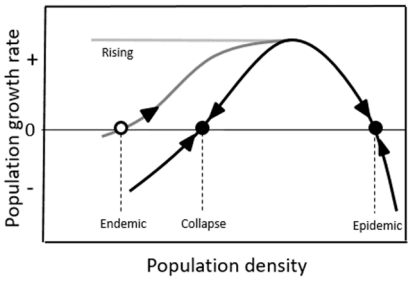
Table S1. Patterns and causes of periodic outbreaks, according to two contrasting theories, and how to optimally manage populations under each set of assumptions.

Category	Sub-Category	Contrasting Theories of Periodic Spruce Budworm Outbreaks	
		Double-Equilibrium Dynamic[1]	**Oscillatory Dynamic**
Pattern	Occurrence	Abrupt transitions from endemic to epidemic equilibrium states Outbreaks are triggered by environmental factors that drive accelerated population growth beyond the capacity of natural controls.	Smooth and gradual sinusoidal rise from endemic to epidemic (i.e., a single equilibrium). The cycle may be slightly asymmetric in rise versus collapse, but transitions are smooth.
	Recurrence	Periodicity is weakened by stochastic triggering of irruptions and stochastic vegetation dynamics. In part, outbreak frequency may be influenced by the forest renewal cycle.	Periodic with the oscillatory frequency shaped primarily by predator-prey interactions.
	Spread	Outbreaks spread contagiously in a wavelike fashion as populations along the leading outbreak edge are driven from the endemic to epidemic phase.	Asymptotic dynamic is the synchronized state. Local waves may emerge temporarily as phase differences in oscillation timing.
	Mathematical dynamics	A relaxed oscillation that goes through discrete phases of outbreak rise and collapse, followed by forest recovery in the interim between outbreaks.	Harmonic oscillations around a single equilibrium state, driven largely by top-down forces of natural enemies whose densities and impact vary in response to herbivore abundance.
Causes	Depensation (e.g., demographic Allee effects)	Strong depensation in low density populations maintains populations in the endemic phase. The type or relative contributions of biological factors driving depensatory pressure may vary amongst systems.	Depensation is non-essential, if it exists at all.
	Role of forest	Forest age and volume sets the maximum population levels at the upper epidemic equilibrium. Forest collapse helps drive herbivore outbreak collapse at the regional scale.	Forest structure has no influence on cycle amplitude, though it may influence dispersal losses during juvenile and adult stages.
	Role of natural enemies[2]	Natural enemies merely vary in response to herbivore density, but are not a key driver of herbivore population collapse.[2]	Natural enemies drive cycles through their lagged responses to prey densities.
	Role of dispersal	Density-dependent dispersal from outbreak areas to surrounding low density areas drives outbreak spread. If dispersal is directed and	Diffusive dispersal draws outbreak trends amongst neighboring populations into closer synchrony, thus promoting more synchronous oscillations at the regional scale.

	Persistence	advective then the spread may occur in "travelling waves". Populations may go locally extinct, although global extinction is prevented by endemic refugia where populations can evade extirpation.	Population densities never go extinct, and will rebound rapidly from low density once natural enemy populations decline.
	Budworm model authority	[27,1,12]	[17,18,21]
	Recruitment curves		
	Caveats	Hassell et al. [3] and Fowler [4] point out that predation is incidental in the LJH model, not a fundamental cause of qualitative dynamical behaviour. Budworm outbreaks are driven by outbreaks of foliage of high quality and quantity. Note: the multiple equilibrium model is actually a simple 2D projection of a more complex 3D "manifold", where forest volumes control the possibility for outbreaks on the upper fold of the manifold.	The single equilibrium state may be a conditional state that varies smoothly as a function of continuous changes in a slow controlling variable, such as forest condition or age.
Management approaches	Slow outbreak spread	Possible during the early rising phase of outbreak. Strong population control efforts may have an enduring effect depending on the strength and consistency of depensatory forces.	Only slight slowing is possible at the onset of the rising phase of the cycle. Population control efforts will have no lasting effect, although cycle amplitude may be diminished somewhat through early subtractions and late additions to the population.
	Prevent an outbreak	Outbreak could be completely prevented, particularly if forest is young, provided that population control is aggressively implemented as the first 'hotspots' appear.	The overarching outbreak cycle can not be prevented. Cycle amplitude may be attenuated somewhat through silvicultural policy favouring mixed-species forests.
	Stop an ongoing outbreak	Beyond a certain point, such as when population densities are near their peak, the outbreak is irreversible and will continue until forest collapse.	Onset of population collapse can be slightly accelerated through late phase control efforts (e.g., insecticides), although this risks exacerbating the next cycle by

			interrupting the natural processes driving outbreak collapse.
	Optimal risk management strategy	Mitigate damage through population control (e.g., Early Intervention Strategy) to reduce or maintain populations below the irruption threshold. Maintain young forests with higher compositions of resistant hosts (e.g., black spruce) and non-hosts (i.e., hardwoods) to reduce forest susceptibility and vulnerability.	Adapt to the inevitable damage by limiting defoliation and associated tree mortality during cycle peak (i.e., Foliage Protection strategy). Focus foliage protection efforts on keeping the highest-value at-risk trees alive until harvest. Stands not slated for imminent harvest should be sacrificed and left to regenerate.

[1] Also known as the "multiple-equilibrium hypothesis" and the "epicentre hypothesis". Aspects of this hypotheses have evolved over the past 60 years, so we have presented the most contemporary version with its core elements.

[2] Although these were the original explanations for of the role of natural enemies under each hypothesis, research in recent decades has shown that both natural enemies and host plant decline can contribute to outbreak collapse [31].

References

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4. Fowler, A.C. *Mathematical Models in the Applied Sciences*; Cambridge University Press: Cambridge, UK, 1997; p. 402.