Appendix B from S. R. Hall et al., "Selective Predation and Productivity Jointly Drive Complex Behavior in Host-Parasite Systems"

(Am. Nat., vol. 165, no. 1, p. 70)

Key Assumptions and Bifurcations in the *S-I*-Predation Model

Table B1
Key assumptions in the *S-I*-predation model, their implications for the model structure, and justification from the *Daphnia*-parasite-fish system

Model assumptions	Implications for model	Biological justification
Susceptible (S) birthrate is density dependent (through parameter c)	Susceptible population is self-regulated without predators and/or parasites	Density dependence of the birthrate is observed in many *Daphnia* systems
Infected (I) individuals do not reproduce but do deplete resources	I is not included in reproduction term (bS) but is included in the density-dependence term $[1-c(S+I)]$	Has been documented for *Daphnia*-microparasite systems (Ebert et al. 2000)
Parasite transmission is linear and depends on *S-I* contact; S and I are well mixed	Classical, "pseudo–mass action" βSI transmission term is used	Linearity is supported in *Daphnia-Pasteuria* system (Regoes et al. 2002)
Predator density (C) changes much more slowly than S and I density	A parameter, not a variable, represents predator density	Generation time of fish greatly exceeds that of bluegill
Once infected, recovery is not possible	Obviates separate equations tracking recovered and immune classes	*Daphnia* do not recover once infected (Ebert et al. 2000)

Table B2
Four bifurcations in the *S-I*-predation model, with implications for resulting dynamics

Type of bifurcation/cause	Implication for dynamics
Transcritical/a new, feasible equilibrium arises	Allee effects and alternative stable states emerge when a second boundary or a second interior equilibrium arises
Fold (saddle node)/two equilibria collide and destroy each other	Multiple equilibrial states occurring before fold are no longer possible after the fold
Hopf/sum of effects of S on dS/dt and I on dI/dt are positive	Switch from stable, damped oscillations to sustained cycles (stable limit cycles); a component of catastrophic extinctions
Homoclinic/limit cycle collides with a saddle	Quick, sudden jump from one dynamic state to another; can be catastrophic for the parasite (high selectivity) or both parasite and host (low selectivity/avoidance)