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Population cycles in voles and lemmings: state of the science and future directions

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ABSTRACT

1. Despite nearly a century of research, the causes of population cycles in Arvicoline rodents (voles and lemmings) in northern latitudes are not yet fully understood. Theory tells us that delayed density-dependent feedback mechanisms are essential for rodent population cycles, suggesting vegetation–rodent, rodent–parasite or rodent–predator interactions as the most likely drivers of population cycles.
2. However, food provisioning, carried out either indirectly through fertilisation treatments of the habitat or directly through food supplementation, has failed to alter population cycles substantially, suggesting that variation in food supply by itself is not necessary or sufficient to cause cyclic fluctuations in abundance.
3. Predator exclusion experiments conducted in Fennoscandia have succeeded in slowing population crashes and increasing autumn densities, implicating predation as the most likely cause of rodent cycles in this region. However, experimental removal of specialist predators in northern England had no discernible effect on a cyclic vole population, casting doubt on the notion that predation is a necessary explanation of rodent population cycles.
4. Population cycle research has contributed substantially to our current understanding of the dynamics, regulation and persistence of biological populations, but we do not yet know with certainty what factors or processes cause multiannual population fluctuations or if population cycles are driven by the same mechanisms everywhere. Recent theoretical and empirical studies suggest that extrinsic factors (primarily food supply and predator abundance) may interact with population intrinsic processes (e.g. dispersal, social behaviour, stress response) to cause multiannual population fluctuations and to explain biological attributes of rodent population cycles.
5. Solving the enigma of population cycles may necessitate identifying factors and processes that cause phase-specific demographic changes and performing conclusive experiments to ascertain the mechanisms that generate multiannual density fluctuations.

INTRODUCTION

Although periodic outbreaks in rodent numbers have been observed throughout recorded history, Elton (1924) was the first to scrutinise the phenomenon of multiannual population fluctuations scientifically. In this exceptionally insightful paper, Elton (1924) not only synthesised information on cyclic fluctuations in abundance of many animal species, but also extensively explored causes and consequences of population cycles with a particular focus on lemmings and voles. He suggested that the causes of population cycles in lemmings "lie either with the lemmings themselves or with their environment", but that the cause of periodicity must "lie with the environment", in light of the spatial synchrony in lemming cycles. A careful reading of his paper reveals that Elton envisioned many of the modern ideas in animal ecology, including the concept of population regulation, prey switching, trophic interactions and cascades, and climatic forcing. In essence, Elton's (1924, 1942) work on population cycles laid the foundation

of the 'Eltonian' tradition of animal ecology, and influenced generations of ecologists. Nearly a century has elapsed since the publication of Elton's seminal work, but population cycles remain enigmatic even today.

The often contentious history of population cycle research has been thoroughly discussed in the literature, and the scientific progress has been reviewed periodically (e.g. Krebs et al. 1973, Tamarin 1978, Lidicker 1988, Batzli 1992, Krebs 1996, Stenseth 1999, Berryman 2002, Hanski & Henttonen 2002, Lambin et al. 2002, Turchin 2003, Kelt et al. 2019). Here, I provide a thorough review of published research on rodent population cycles, highlight interesting features of cyclic rodent populations, review progress, discuss the possible reasons for our failure to ascertain causes of population cycles, and identify knowledge gaps that must be filled to identify necessary and sufficient causes of population cycles.

CHARACTERISING RODENT POPULATION CYCLES

The most prominent feature of cyclic populations are multiannual fluctuations in abundance with cyclic phases (increase, peak, decrease and low phases) occurring every 3–5 years (Krebs 2013). Various statistical approaches have been used to characterise population cycles, including the s-index, wavelet analysis, and time-series analysis (see Appendix S1 for details). Field evidence shows that most, if not all, cyclic rodent populations are characterised by phase-related changes in body mass, social behaviours, age structure, age at sexual maturation, survival and reproductive rates (Chitty 1952, 1960, Krebs et al. 1973, Krebs & Myers 1974, Boonstra & Krebs 1979, Boonstra 1994, Prevot-Julliard et al. 1999, Norrdahl & Korpimäki 2002); together, they constitute the 'biological definition' of population cycles (Krebs 1996). At high densities, sexual maturation is delayed, the length of breeding season shortens, juvenile survival rates and reproductive rates are reduced, the proportion of young animals in the population declines, and the mean age of reproductive females increases; these changes precede and/or accompany population crashes (Krebs et al. 1973, Boonstra 1994, Krebs 1996, Prevot-Julliard et al. 1999, Ergon et al. 2001a, b, Norrdahl & Korpimäki 2002). At low densities, these patterns are reversed. Another feature of cyclic populations is the phase-related changes in body mass: individuals are substantially heavier (up to 30% heavier) during the high-density phase, and this is often referred to as the 'Chitty effect' (Chitty & Chitty 1962, Oli 1999). Also, social behaviour may be affected (animals in high-density phases are much more aggressive than those in low-density phases; Krebs 1970, Cockburn 1988, Lidicker & Ostfeld 1991). These syndromes of population cycles are sometimes ignored or dismissed as unimportant by some theoretical ecologists for convenience (e.g. Stenseth 1999, Hanski et al. 2001, Turchin 2003), but most field studies show that these biological changes are nearly ubiquitous and indispensable components of rodent populations that exhibit cyclic or otherwise large-scale population fluctuations. Other features of cyclic populations include latitudinal gradients in the degree of cyclicity (e.g. populations in northern Europe tend to be more cyclic than southern ones) and a broad range of spatial synchrony within and among species (Stenseth 1999, Krebs 2013). Spatial patterns in cyclic dynamics range from travelling waves (e.g. Lambin et al. 1998) to highly synchronous fluctuations over large geographic areas (e.g. Angerbjörn et al. 2001). Thus, any hypothesis attempting to explain population cycles must explain phase-specific changes in population characteristics as well as broader spatial and temporal patterns in abundance that define population cycles.

THEORETICAL FOUNDATIONS

The theoretical impetus for population cycle research has been provided by two complementary ecological theories: the theory of population regulation and the theory of predator–prey (or, more broadly, consumer-resource) dynamics. The former embodies the 'density paradigm', and the latter the 'mechanistic paradigm' of population ecology proposed by Krebs (2002). The theory of population regulation provides a conceptual foundation for our understanding of how biological populations are regulated, and attempts to explain why populations fluctuate as they do. Factors or processes that prevent unlimited population growth have long been debated in the literature (Nicholson 1933, Andrewartha & Birch 1954, White 2001, Turchin 2003), but population regulation theory suggests that density-dependent feedback mechanisms that permit populations to grow at faster rates at low densities but reduce population growth rates at high density are necessary and often sufficient for preventing unlimited population growth (Royama 1992, Turchin 1999, 2003). Although the importance of abiotic (density-independent) factors in limiting populations is widely recognised, Royama (1992) argued that population regulation necessarily implies density-dependence, and that unregulated populations cannot persist. The structure of density-dependence determines the pattern of population fluctuations (Royama 1992). Direct (or first-order) density-dependence typically leads to stable equilibrium whereas delayed (or second-order) density-dependence can generate a variety of dynamical patterns, including cyclic fluctuations, depending on the strength of direct and delayed density-dependence. The primary analytical tools used within this framework are time-series models such as autoregressive or autoregressive-moving average models (Royama 1992, Turchin 1999, 2003; Appendix S1).

The theory of predator-prey dynamics was motivated by the Lotka–Volterra predation model, which showed that interaction between prey and their specialist predators can cause cyclic fluctuations in prey abundance (Lotka 1924). Motivated by the cyclic dynamics generated by predator–prey models (Lotka 1924, May 1973), the theory of predator–prey dynamics assumes that interactions between predators and their prey underlie rodent population cycles, and focuses on understanding coupled dynamics of predators and the rodent prey. Because models of trophic–interactions or consumer–resource dynamics (predator–prey, herbivore–consumer and host–parasite) often exhibit cyclic dynamics, models of predator–prey, rodent–vegetation and rodent–parasite dynamics have been studied more extensively than any other (Hanski et al. 2001, Berryman 2002, Turchin 2003). Modelling frameworks adopted in mechanistic models of rodent population cycles include differential equation models (Hanski et al. 2001, Turchin & Batzli 2001, Reynolds et al. 2013), difference equation models (Inchausti & Ginzburg 1998, 2009), age-structured or stage-structured matrix population models (Klemola et al. 2003), individual-based population models (Radchuk et al. 2016), and integral projection models (van Benthem et al. 2017).

Capture–mark–recapture modelling approaches, especially those developed by Pradel (1996), are particularly useful for the study of cyclic populations (Pradel 1996, Nichols 2016) for several reasons. First, they permit direct estimation of realised population growth rate and its components: recruitment and apparent survival. Second, recruitment, apparent survival and realised population growth rates can be directly modelled as functions of temporal covariates (e.g. rodent and/or predator abundance, season, phase of a cycle, food addition) and site covariates (e.g. habitat quality). Third, this approach provides a framework for representing competing hypotheses as statistical models, and for using information-theoretic approaches for assessing empirical support for each of the competing hypotheses (Burnham & Anderson 2002). Fourth, questions about the relationship between sets of demographic parameters (e.g. abundance and survival) can be addressed in a manner that deals adequately with sampling variances and covariances associated with the different parameters. Unfortunately, these models have seldom been applied to the cyclic rodent populations (but see Yoccoz et al. 1998, Ozgul et al. 2004, Goswami et al. 2011). Long-term capture–mark–recapture studies of fluctuating populations are challenging, and it is particularly difficult to obtain adequate sample size during the low phase.

HYPOTHESES AND EMPIRICAL EVIDENCE

Krebs (2013) placed hypotheses of rodent population cycles into five broad categories based on the causal factors invoked: food, predation, disease, self-regulation and multifactorial hypotheses. Hypotheses of rodent cycles have also been organised under the banners of various historically significant 'schools of thought' (not mutually exclusive; Batzli 1992, Krebs 1996, 2013): 1) intrinsic school, which proposed that factors internal to the population (e.g. genetics, dispersal, social behaviour, stress response) are necessary and sufficient to cause population cycles (e.g. Chitty 1960); 2) extrinsic school, which proposed that extrinsic factors such as food, predators and parasites are the cause of population cycles (e.g. Hanski et al. 2001, Berryman 2002); 3) single-factor hypotheses, which suggested that a single intrinsic or extrinsic factor is the primary cause of population cycle with additional factors playing only secondary roles (Courchamp et al. 2000, Hanski et al. 2001); and 4) multi-factorial school, which argued that rodent population cycles are too complex to be explained by a single factor and that multiple intrinsic or extrinsic factors interact to cause population cycles (e.g. Lidicker 1988). Here, I briefly review relevant hypotheses and models (when appropriate), and summarise the empirical evidence, if any.

Predator–prey dynamics and the specialist predator hypothesis

Early models of predator–prey dynamics suggested that interaction between specialist predators and their prey can lead to cyclic population dynamics (Lotka 1924, May 1973). These results motivated the specialist predator hypothesis, which posits that a delayed numerical response of resident specialist predators to changes in prey abundance leads to multiannual population cycles. Models of predator–prey dynamics are the most intensively studied mechanistic models of population cycles (Hanski et al. 2001, Turchin 2003). Predator–prey models of cyclic populations are typically formulated as two-dimensional systems of differential equations. Various versions of predator–prey models have been developed and analysed; they differ mostly in the way functional response is modelled and in whether or not the effects of generalist predators are included (Turchin & Hanski 1997, 2001, Hanski et al. 2001, Turchin 2003). A version of the predator–prey model (Appendix S1), referred to as the Hanski model, can generate prey population dynamics that are strikingly similar to cyclic dynamics exhibited by voles in Fennoscandia. The Hanski model has been extended to include seasonality and environmental stochasticity; these extensions generate more realistic dynamics (Turchin & Hanski 1997, Hanski et al. 2001).

The predation hypothesis assigns a prominent role to predators and postulates that non-migratory, specialist predators are necessary, and a combination of predation

by specialist and generalist predators are both necessary and sufficient, for causing rodent population cycles and the latitudinal gradient in cyclic amplitudes observed in Fennoscandia (Korpimäki & Norrdahl 1998, Hanski et al. 2001, Korpimäki et al. 2002, Lambin 2017). Specialist predators are thought to be the primary drivers of population cycles (hence, this idea is often referred to as the specialist predator hypothesis), because it takes time for specialist predators to respond numerically (via reproduction) to changes in prey abundance, which introduces the time-lag required to generate second-order dynamics and, consequently, population cycles. The diversity and abundance of generalist predators are thought to determine the cycle length and amplitude. Because generalist predators can affect vole populations instantaneously by increasing predation rate (and thus reducing survival) as the vole density increases (and the predators' own survival and reproduction does not depend on the abundance of a particular prey species), these generalist predators generate direct density-dependence in vole population dynamics. Together, specialist and generalist predators create a pattern of autocorrelation in prey population dynamics that is thought to be characteristic of 3–5 year rodent cycles (Bjørnstad et al. 1995, Hanski et al. 2001, Turchin 2003). The predation hypothesis has also been suggested to explain the latitudinal gradient in rodent population cycles in Fennoscandia, because the degree of cyclicity increases in northern latitudes, which generally corresponds with a decrease in the diversity and abundance of generalist predators of cyclic rodents (e.g. Hanski et al. 1991, Bjørnstad et al. 1995, Stenseth 1999).

The predation hypothesis has been tested by using observational and modelling studies, as well as field experiments (reviewed by Hanski et al. 1991, 2001). Perhaps the strongest support for this idea comes from the studies conducted by Erkki Korpimäki's research group in Finland. In a series of experiments, Korpimäki's research group found (Norrdahl & Korpimäki 1995a, b, Korpimäki & Norrdahl 1998, Sundell et al. 2000, Korpimäki et al. 2002): 1) nomadic avian predators did not substantially affect vole abundance, and these predators tracked vole abundance without time lag, leading to the conclusion that these avian predators could not cause rodent cycles although they might cause geographic synchrony in population cycles; 2) experimental reduction in densities of all main predators slowed vole population declines, whereas populations continued to decline in control areas and at sites where the abundance of only the generalist predators was reduced; and 3) reduction in the abundance of all main predators during summer and autumn increased the autumn vole density fourfold during the low phase and twofold during the peak phase, and retarded the initiation of the decline phase (Korpimäki et al. 2002). These results were taken as evidence "for the hypothesis that specialist predators drive summer decline of rodent populations in northern Europe" (Korpimäki & Norrdahl 1998). Also, these findings and results of time series analyses led Korpimäki et al. (2002) to conclude that cyclic rodent populations may be transformed to non-cyclic by manipulating predator abundance, and that predators may generate the rodent population cycles observed in Fennoscandia.

Empirical evidence for the role of specialist predators (or predators generally) outside Fennoscandia are mixed. Fauteux et al. (2016) conducted a fencing experiment in Bylot Island, Canada, to test the idea that predators limit cyclic brown lemming *Lemmus trimucronatus* populations. Their results revealed that summer density and survival of lemmings, as well as nest density during the winter, were higher in experimental grids (i.e. with reduced predation) than in control grids, providing evidence that predators may limit the lemming populations. However, these results do not show that predators regulate lemming populations in a delayed density-dependent fashion so as to cause population cycles. A similar experiment at Pearce Point, Northwest Territory, Canada (Reid et al. 1995) revealed that, although the collared lemming *Dicrostonyx groenlandicus* population protected from predators via fencing experienced higher survival, it did not grow faster than the unprotected population. These authors suggested their failure to exclude all predators and the disruption of dispersal events as explanations for these results.

The strongest evidence contradicting the specialist predator hypothesis comes from a long-term experiment conducted in the Kielder Forest, northern England (Graham & Lambin 2002). In a replicated field experiment, Graham and Lambin (2002) experimentally reduced the density of weasels *Mustela nivalis*, the specialist predator of the field vole *Microtus agrestis* in the Kielder Forest, by live-trapping and removing them; they then monitored the abundance of both voles and weasels. Results revealed that 1) weasel removal did not prevent the crash of the population during the decline phase of the cycle; 2) vole populations fluctuated similarly in control and manipulated sites, and 3) there was no evidence to suggest that the numerical response of weasels to temporal changes in vole abundance was delayed density-dependent, a condition necessary for predation to cause cyclic fluctuations in abundance. These results led Graham and Lambin (2002) to conclude that "changes in weasel predation rate were not responsible for driving the population cycles of field voles observed in Kielder Forest". However, it is important to note that Graham and Lambin's (2002) experiment reduced the abundance of the specialist predator, but not of generalist predators that prey on voles to varying degrees. Also, Kielder Forest vole cycles are thought to be somewhat

different from those in Fennoscandia (Korpimäki et al. 2005). Thus, Graham and Lambin's (2002) results only contradict the specialist predator hypothesis as a universal explanation of population cycles; they do not necessarily rule out the possibility that specialist predators interact with other intrinsic and extrinsic factors to generate population cycles in Kielder Forest or elsewhere.

Vegetation–rodent dynamics and the food hypothesis

The idea that bottom-up processes could regulate rodent numbers, either directly through changes in quantity or quality of food, or indirectly via soil nutrients, has been around for a long time. Periodic overexploitation of food resources by rodents can potentially cause populations to crash due to starvation; time required for vegetation to regrow or soil nutrients to be replenished could introduce the delayed effect needed to cause population cycles. Consequently, rodent–vegetation interactions have long been considered as an important factor causing, or contributing to, population cycles (Summerhayes 1941, Pitelka 1957, 1964).

Turchin and Batzli (2001) developed and analysed a suite of rodent-vegetation models (represented as two- or three-dimensional differential equations) with the goal of understanding quantitative characteristics of rodents and their food plants that can cause herbivore cycles. They assumed that recovery of vegetation following overgrazing can take time, which can introduce time-lags that are necessary for a consumer resource system to generate cyclic dynamics. They considered a variety of scenarios, because many aspects of rodent-vegetation dynamics are not well understood (e.g. the recovery or growth pattern of rodent food sources following overgrazing, the functional response of rodents to changes in vegetation abundance). Although some of these models generated cyclic population dynamics, none yielded periodicity of 3–5 years. Because vegetation–herbivore dynamics can be affected by rodent–predator dynamics, they also considered multitrophic models (vegetation–rodents–specialist predator dynamics), which generated cyclic rodent dynamics with realistic periodicity (Turchin & Batzli 2001). However, they advised caution in interpreting these results.

The first rigorous experiment to test the food hypothesis was conducted by Summerhayes (1941) under the supervision of Charles Elton. Some experimental plots were excluded from rodent herbivory using fences; the vegetation within and outside the exclosures was compared to test the idea that overgrazing precedes population crash. The results revealed that vegetation within and outside of exclosures did not differ substantially, and that there was no evidence of overgrazing or of starvation prior to the population crash (Chitty 1996, Krebs 2013). Exclusion of collared lemmings and brown lemmings and other herbivores by fencing actually reduced diversity and biomass of vascular plants near Barrow, Alaska, USA (Johnson et al. 2011). A study using experimental exclosures found that winter grazing by collared lemmings and brown lemmings had no discernible effect on the biomass of vascular plants or moss in the Canadian Arctic (Bilodeau et al. 2014). There has been no consistent evidence that overgrazing affects food quality, that overgrazing adversely influences rodent reproduction, or that overgrazing necessarily reduces rodent abundance. Contrary to expectations, Klemola and colleagues (Klemola et al. 2000a, b) found that voles in previously overgrazed enclosures reproduced just as well as those inhabiting control sites (i.e. areas with no previous overgrazing), and that vole abundance was higher in previously overgrazed areas than in control sites.

A variant of the food hypothesis is the nutrient–recovery hypothesis, originally proposed to explain population cycles of brown lemmings in Barrow, Alaska (Pitelka 1964). The nutrient–recovery hypothesis states that high rodent densities decimate the vegetation, leading to starvation and a population crash. Lemming populations do not experience population growth until soil nutrients are accessible to plants, plant production and accumulation of litter have increased and nutrient levels in food plants return to normal levels (Pitelka 1964, Pitelka & Batzli 2007). This idea was tested using a fertilisation experiment in Alaska, which showed that lemming populations did not respond positively to the experimental fertilisation (Pitelka & Batzli 2007). A similar fertilisation experiment failed to produce expected results in another cyclic herbivore, the snowshoe hare *Lepus americanus* in the Kluane region of Canada (Krebs et al. 1995).

A more direct approach to test the food hypothesis is to provide supplemental food to experimental populations, with the expectations that food supplementation should stop or drastically alter population cycles (Krebs 2013). Several food supplementation experiments have been conducted to test this idea (e.g. Cole & Batzli 1978, Taitt & Krebs 1981, 1983, Getz et al. 2006; for reviews, see Boutin 1990, Prevedello et al. 2013). Taken together, results of food supplementation studies show that food supply affects many aspects of rodent ecology (e.g. density, movement and spacing behaviours, and in some cases, reproduction) but food supplementation does not stop or substantially alter population cycles, suggesting that changes in food supply by itself are not necessary or sufficient for population cycles to occur. However, Johnsen et al. (2017) reported that bank voles *Myodes glareolus* went extinct in all study plots that did not receive supplemental food during the second winter of the experiment, whereas

populations persisted in plots receiving supplemental food. This result and evidence from multifactor experimental studies suggests that food supply can interact with other factors, such as predation, to affect population cycles (see below).

Many plants produce secondary compounds (e.g. silica, phenolic compounds) as a deterrent to or defence against herbivory, and these defence mechanisms may affect herbivore survival and reproduction (Huitu et al. 2014, DeAngelis et al. 2015, Hartley & DeGabriel 2016). Massey et al. (2008) found that grazing by field voles at high vole densities led to increased silica levels in a winter food plant, and eating food with high silica levels led to reduced vole body mass. Based on these findings, Massey et al. (2008) suggested that inducible, silica-based defences in grasses may drive field vole population cycles. Grazing by field voles (Reynolds et al. 2012) and root voles *Microtus oeconomus* (Wieczorek et al. 2015) has been shown to increase silica levels in their food plants; the silica levels are lagged and a nonlinear function of the grazing intensity. Using coupled grass-vole differential equation models, Reynolds et al. (2012, 2013) showed that delayed, silica-based plant defensive responses to grazing intensity can, in theory, generate population cycles. However, there exists no empirical evidence that grazing-induced changes in silica (or other measures of food quality) are necessary for, or substantially affect, rodent population cycles (Ruffino et al. 2018).

Host–parasite dynamics and the disease hypothesis

Rodents, like many animal species, are often infected by parasites (or communities of parasites) that can potentially affect survival, reproduction or both, which can in turn affect population dynamics. This possibility did not escape the notice of population cycle pioneers; indeed, recurring epidemics were one of the first possible explanations of population cycles in rodents (Chitty 1996, Krebs 2013). Although early studies based on primitive detection methods indicated that parasites such as *Toxoplasma* could be important in regulating vole populations in the UK (Elton et al. 1935), they were rejected as plausible explanations of population cycles due to lack of evidence (Elton et al. 1935, Chitty 1954). Diseases and parasites have been studied in several cyclic rodents (e.g. *Myodes* spp. and *Microtus* spp. and *Arvicola* spp.), with some studies showing that diseases can affect demographic parameters and others showing the lack of evidence for such effects (e.g. Telfer et al. 2005, Cerqueira et al. 2007, Deter et al. 2007, 2008). Smith et al. (2008) modified the classic 'susceptible, infected and recovered' model of host–pathogen dynamics to include two types of recovered hosts: hosts that have recovered from the disease and gained immunity but cannot yet reproduce; and hosts that have recovered and can reproduce, but at a rate lower than the healthy individuals (Appendix S1). Their host–parasite model was a system of four differential equations, and assumed that the disease influenced both survival and reproduction. They concluded that diseases with brief infection periods but slow recovery of reproductive function once hosts recover from the disease can generate high-amplitude, multiannual population fluctuations. Based on these modelling results and the field evidence that some diseases of field voles (or infection of voles by multiple potentially interacting parasites) show density-dependent cyclic fluctuation, the authors offered vole–parasite interactions as a potential explanation of population cycles in northern England (Smith et al. 2008, 2009).

Perhaps the most extensive study of the potential effect of diseases on the dynamics of cyclic populations was conducted by the research groups of Xavier Lambin, Sandra Tefler and Michael Begon in the UK. Studies of field voles in northern England showed that voles are infected by an interacting community of parasites, that some of these parasites show density-dependent (and sometimes cyclic) prevalence patterns, and that some of these infections can adversely affect voles' population parameters (Cavanagh et al. 2004, Telfer et al. 2005, 2011). Using models of host–parasite dynamics parameterised with field data, Smith et al. (2008, 2009) showed that diseases can potentially cause cyclic fluctuation in vole abundance. These studies also revealed complex interactions among communities of pathogens mediated by competition for resources and host immune response (Begon et al. 2009). However, most studies of the effects of disease on cyclic rodent population dynamics have been observational, and there has been no convincing experimental evidence that diseases or parasites cause rodent population cycles. Experimentally testing disease effects on cyclic populations would necessarily require manipulating a community of parasites or vaccinating voles; such experiments would be logistically challenging and have not been attempted (but see Smith et al. 2008, Turner et al. 2014).

Maternal effects model and hypothesis

Inchausti and Ginzburg (1998, 2009) suggested that maternal effects, defined as non-genetic transmission of quality from mother to offspring, can cause population cycles, and developed a model to formalise this idea. The Inchausti-Ginzburg model assumes that the 'quality' of mothers (vaguely defined) changes in a density-dependent fashion, and, in turn, influences population dynamics. The model is formulated as two systems of difference equations (each system consisting of two difference equations), one to

model the coupled dynamics of rodent population and the quality of mothers in spring, and another to model the same dynamics in autumn. The 'quality' of mothers is assumed to be transmitted from mothers to offspring, the phenotypic transmission of quality from mothers to offspring. However, the periodicity of cycles generated by this model is generally inconsistent with those observed in nature for realistic parameter values. Turchin (2003) notes that realistic parameter values lead to 1–2 year cycles, and that unrealistically high survival rates (>0.95 per month) would be needed to generate cycles with periodicity of 3–5 years.

Of several intrinsic (or self-regulation) hypotheses proposed to explain rodent population cycles, one (the Chitty hypothesis) has been rejected (Boonstra & Boag 1987), and evidence for others have been mixed or inconclusive (Appendix S2). Except for the maternal effect hypothesis, mathematical or theoretical models representing intrinsic hypotheses do not currently exist. Based on the findings of a reciprocal transplant experiment showing that past environments had no substantial effect on field vole life history variables, Ergon et al. (2001a) rejected intrinsic mechanisms as an explanation of population cycles. Because intrinsic factors are difficult to manipulate, experimental tests of intrinsic hypotheses have been rare (Krebs 2013).

Models invoking multiple factors and the multifactorial hypotheses

Rodent population dynamics are inherently complex, and highly variable over time and space. Populations of the same species exhibit multiannual cycles at some times and in some places, but seasonal or erratic fluctuations at other times or in other places. These universally accepted observations, combined with the fact that experiments in which one or two factors were manipulated have failed to explain population cycles satisfactorily, led Lidicker (1978, 1988, 2000) to conclude that rodent population cycles are too complex to be explained by one or two factors and that multiple factors interact to cause the observed population cycles. He rejected arguments regarding whether intrinsic or extrinsic factors are the primary cause of population cycles as unhelpful, proposed that four intrinsic and four extrinsic factors are involved in driving the demographic machinery underlying cyclic population dynamics in California voles *Microtus californicus*, and argued that the relative roles of these factors vary across density phases and over time and space. The multifactorial perspective is conceptually appealing, especially in light of the failure of hypotheses invoking one or two factors to explain population cycles satisfactorily. In addition, this perspective can explain annual or multiannual population fluctuations, spatio-temporal variations, and biological attributes of cycles. Experimentally manipulating many factors simultaneously can be logistically challenging; consequently, there have been few experimental tests of the multifactor hypothesis in its original form (Krebs 2013). Nonetheless, the potential influence of two or three factors thought to be the key drivers of population cycles have been tested using both mathematical models and experiments.

Klemola et al. (2003) developed and analysed demographically based, density-dependent, stage-structured matrix population models that incorporated the potential influence of vegetation, specialist predators and generalist predators on population dynamics of voles and lemmings. They showed that tri–trophic interactions can produce cyclic changes in abundance. However, the modelled dynamics did not adequately capture the shape and amplitude of cycles observed in cyclic rodent populations; the authors suggested that assumptions about phase-dependence in trophic interactions or some population-intrinsic factors would be needed to generate realistic population cycles (Klemola et al. 2003). Therefore, trophic interactions may be necessary but not sufficient to cause population cycles similar to those observed in nature. This latter observation is consistent with the idea that multiple factors may act synergistically to generate population cycles (Lidicker 1978, 1988, Andreassen et al. 2013).

Using an individual-based modelling approach (Railsback & Grimm 2011), Radchuk et al. (2016) tested models representing combinations of two intrinsic factors (sociality and dispersal) and one extrinsic factor (predation) for their potential to generate population cycles comparable to those observed in the field. They found that only the full model – including sociality, dispersal and predation – yielded population cycles with periodicity, amplitude and autumn densities comparable to those observed in North Fennoscandia. This study is the only study that used an individual-based modelling approach with empirically estimated parameters and simultaneously considered both intrinsic and extrinsic factors.

The interactive effects of two or three factors on rodent population dynamics have been tested experimentally. The most common of these experiments manipulated the two obvious potential drivers of rodent population cycles: food and predators. Results of these experiments have revealed consistently that the population-level effect of food supplementation and predator exclusion (or predator removal) was almost always greater than the singular effect of either treatment (Klemola et al. 2000a, Huitu et al. 2003, Krebs 2013, Prevedello et al. 2013). It is conceivable that primary productivity or food supply modulates the effect of predators on prey populations (e.g. Oksanen & Oksanen 2000). However, none of these experiments succeeded in stopping or substantially altering cyclic population dynamics. Forbes

et al. (2015) experimentally tested for the effect of food and a pathogen (*Bordetella bronchiseptica*) on field vole population growth rate. Contrary to expectations, they found that the vole population that received supplemental food was more severely affected by the experimentally introduced pathogen than the population that did not. Few studies have tested for effects of three or more factors on cyclic rodent populations (but see Taitt & Krebs 1983, Krebs et al. 1995, Batzli et al. 2007).

DEMOGRAPHIC MECHANISMS: THE MISSING LINK

Changes in population size are caused by changes in survival, reproduction and immigration rates, which, in turn, are functions of extrinsic and intrinsic factors (Caswell 2001, Krebs 2002, Oli & Armitage 2004). Thus, cyclic changes in abundance are caused by cyclic changes in population growth rate, which are, in turn, a consequence of cyclic changes in vital demographic rates (Caswell 2001, Krebs 2002). Indeed, it is widely accepted that population cycles are characterised by phase-related changes in several life-history parameters. Arguably, the identification of demographic mechanisms underlying population cycles can facilitate ascertainment of factors and processes that cause multi-annual fluctuations in abundance (Oli & Dobson 1999, 2001, Krebs 2013). As an example, consider a population where phase-related changes in survival are the primary determinants of population growth (e.g. Korpimäki & Norrdahl 1998, Korpimäki et al. 2002). This knowledge is sufficient to exclude factors such as social behaviour or stress response as possible determinants of population cycles, because these factors are unlikely to cause direct mortality, and to conclude that natural enemies (e.g. predators or pathogens) are the likely cause of the phase-related changes in survival, and thus, of population cycles. Consider, on the other hand, a population where phase-related changes in age at first reproduction are the primary demographic drivers of population growth (Oli & Dobson 1999, 2001, Norrdahl & Korpimäki 2002, Krebs 2013). This information is sufficient to exclude predation as the cause of population cycles, because direct predation is unlikely to influence age at sexual maturation. Indeed, phase-related changes in reproductive parameters, rather than survival rates, are thought to be the demographic drivers of vole population cycles in Kielder Forest, England and in Chize, France (e.g. Smith et al. 2006, 2008, Ergon et al. 2011, X. Lambin, personal communication). This knowledge and logical reasoning leads to the conclusion that social factors (e.g. social suppression of reproduction, kinship, spacing behaviour), stress response or food resources are the likely drivers of population cycles. Finally, consider a situation where population crashes are characterised by sharp increases in predator abundance, but rapid population growth is accompanied by early sexual maturation and a preponderance of young animals in the population (e.g. Boonstra 1994). This knowledge allows one to isolate the most likely environmental causes of population cycles and devise experiments to test them.

Unfortunately, phase-specific demographic patterns are yet to be rigorously quantified in most cyclic rodents; we do not yet know what demographic changes underlie transitions from one phase to another. Data are particularly scarce, and our knowledge is limited, for decline and low phases (Krebs 2013). A research programme that integrates the identification of demographic mechanisms of population cycles with environmental causes of demographic changes could help to devise focused experiments that may resolve the enigma of rodent population cycles (Oli & Dobson 1999, 2001).

WHAT HAVE WE LEARNED AND WHERE DO WE GO FROM HERE?

Despite the recognition of rodent population cycles as significant ecological phenomena, we have failed to determine the underlying causative factors thus far. This failure is not for the lack of trying; theoretical and empirical research has produced many hypotheses and 1000s of publications (Batzli 1992, Krebs 2013). What have we learned from nearly a century of research on population cycles, and what do we need to do to solve the enigma of population cycles? A brief summary follows.

1. We do not yet know what factors and processes are necessary and sufficient to cause population cycles in rodents, because no experiment has succeeded in stopping population cycles anywhere, although some have prevented or delayed the population crash. Given the complexity of the ecological systems of which cyclic rodents are a part, experimentally stopping population cycles (although desirable) is perhaps unrealistic; however, the hypothesised factor(s) must substantially alter cyclic dynamics for it to be a necessary cause of population cycles.
2. Modelling and experimental studies in Fennoscandia have established predation by specialist and generalist predators as the most likely cause of population cycles in northern Europe. However, tests of the predator hypothesis elsewhere have yielded mixed results, suggesting that predation is not a universal explanation of population cycles or that population cycles do not have a universal explanation.

3. A large body of theoretical work has led to a plethora of mathematical models that can generate cyclic population fluctuations. These theories and models have provided many insights, and have highlighted the role of seasonality and the potentially interacting effects of predators, parasites and intrinsic factors.
4. Although it is widely recognised that social factors and predator-induced stress responses can strongly influence rodent population processes, there have been very few experimental tests of intrinsic hypotheses of population cycles. For example, the chronic stress hypothesis, as modified by Rudy Boonstra's research group (Boonstra et al. 1998, Peers et al. 2018), is amenable to experimental testing and should be tested in cyclic rodent populations (see also Appendix S2).
5. Results of a modelling study by Radchuk et al. (2016) suggested that both intrinsic and extrinsic factors may be necessary to explain rodent population cycles adequately, yet few attempts have been made to test the multifactorial hypothesis due to logistical difficulties.
6. It is widely accepted that population cycles are accompanied by several life-history and behavioural traits that vary across cyclic phases. Whether these changes are necessary for population cycles to occur, or are simply epiphenomena associated with fluctuating populations, remains unknown and deserves further research.
7. Loss of cyclicity and/or dampening of cyclic amplitudes has been reported in some cyclic rodents in Europe. It now appears that the collapse of population cycles or dampening of cyclic amplitudes were localised and temporary phenomena (Appendix S3, Brommer et al. 2010).
8. Earth's climate is changing; climate change will inevitably affect the distribution and abundance of many plant and animal species via changes in the abiotic environment, primary productivity and species interactions (Boonstra et al. 2018). There exists some evidence that climate change may dampen the dynamics of some cyclic rodent populations (e.g. Kausrud et al. 2008, Schmidt et al. 2012, Cornulier et al. 2013). However, the direction or magnitude of climate change effects on population cycles remains unclear.
9. Despite a long history of population cycle research, many data gaps remain. Few researchers have quantified the phase-specific demography of cyclic populations, and we know very little about the decline and low phases of the cycle. Filling these data gaps, and making complete demographic descriptions of population cycles (which are essentially demographic processes), is critical for further progress in demographic cycle research.

Some theoreticians have argued that theories and models are sufficient to understand and explain population cycles, or that population cycles have been fully explained by predator-prey models (e.g. Turchin 2003). This view-point is rejected by empirical ecologists, who emphasise the importance of experiments and long-term field studies (e.g. Krebs 2013). Population cycle research would great benefit from a better integration of theoretical and empirical work: experiments guided by theories, and testable theories that explain observed facts are needed. The application of statistically rigorous modelling approaches, such as capture–mark–recapture analyses, has increased in recent years. Capture–mark–recapture methodologies now permit direct estimation and modelling of population growth rate, as well as age- and state-specific survival and reproductive rates. These approaches should be helpful in discerning demographic mechanisms underlying population cycles. Other unresolved questions in population cycle research include (also see Krebs 2013): 1) is there a universal explanation of rodent cycles? 2) Are there key experiments that can solve the enigma of population cycles once and for all? What are they? 3) Should experiments attempt to stop population cycles? Or is it enough to alter population growth rates? And 4) what constitutes the conclusive test of a hypothesis proposed to explain population cycles?

As carefully articulated by Krebs (2013), the process of explaining population cycles must begin with rigorous and quantitative characterisation of phase-specific demography. This knowledge can help exclude implausible explanations from further consideration. Population cycle research over the last 30 years has been focused almost exclusively on numerical changes, with little attention for the biological attributes of cycles. A demographically based research agenda that integrates numerical dynamics with phase-related demographic changes, and experiments that manipulate both intrinsic and extrinsic factors, will be needed to solve the enigma of population cycles, an ecological problem that fascinated Charles Elton and generations of ecologists. Population cycle research has a contentious history (Tamarin 1978, Stenseth 1999, Krebs 2013), since most leading research groups in the past seemed to subscribe to a specific idea. Rejection of data or ideas that do not support a particular viewpoint seemed to occur more often in population cycle research than in most other ecological disciplines. I concur with Krebs' (2013) suggestion that a dose of humility would benefit us all, because "no one knows where we will finish, and so much still remains to be done".

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SUPPORTING INFORMATION

Additional supporting information may be found in the online version of this article at the publisher's web-site.

Appendix S1. Details of autoregressive time series models, and models of predation and diseases.
Appendix S2. Intrinsic hypotheses of population cycles.
Appendix S3. Are population cycles dampening or collapsing?