



# Phase control of resonant systems: Interference, chaos and high periodicity

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## ABSTRACT

Much progress has been made in understanding the effect of periodic forcing on epidemiological and ecological systems when that forcing acts on just one part of the system. Much less is known about situations in which several parts of the system are affected. In this case the interaction between the impacts of the different forcing components can lead to reinforcement of system responses or to their interference. This interference phenomenon is significant if some forcing components are anthropogenic for then management might be able to exercise sufficient control to bring about suppression of undesirable aspects of the forcing, for example resonant amplification and the problems this can cause. We set out the algebraic theory when forcing is weak and illustrate by example what can happen when forcing is strong enough to create subharmonics and chaotic states. Phase is the key control variable that can bring about interference, advantageously shift nonlinear response curves and create periodic states out of chaos. The phenomenon in which high period fluctuations appear to be generated by low period forcing is examined and different mechanisms compared in a two-strain epidemiological model. The effect of noise as a source of high period fluctuations is also considered.

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## 1. Introduction

Resonance occurs when a disproportionately large response is produced through the excitation of one or more of the natural oscillatory modes of a system by external periodic forcing. Resonance is familiar in engineering systems where it can lead to catastrophic structural failure. Its effect is also apparent in the life sciences where it is thought to be the primary cause of many periodic epidemics in plant, animal and human diseases. These epidemics can have a serious negative impact on the balance of species in wildlife ecologies and on public health. This connection between epidemics and resonance was confirmed in the study of childhood diseases in the 1980s (Dietz, 1982). Since then resonance has been observed in a wide range of ecological and epidemiological situations (Altizer et al., 2006), differing in the period, strength and configuration of the external forcing and in the dynamics of the unforced system.

Seasonality is the most familiar driver of external environmental forcing, affecting both birth and death rates as well as infection transmission and predation strength. Other environmental drivers such as ENSO (i.e. El Nino Southern Oscillation) and rain patterns in Africa and Asia (Wichmann et al., 2003) have

multiannual periods. Environmental forcing is not restricted to such global or regional climate variations. For example, a species, attempting to invade a resident community or web, will see that community or web as the “environment” and will be subject to its often long period fluctuations endogenously or exogenously generated (Berryman, 1987; Dwyer et al., 2004).

The strength of forcing is also highly relevant in many forcing situations. If forcing is strong enough, subharmonics can be generated whereby the system populations oscillate with a period that is an integer multiple of the forcing period. For example, before vaccination programmes were introduced, measles epidemics occurred typically every 2 years (Dietz, 1982) even though the forcing period is thought to be seasonal, following the rhythm of the school year (Keeling et al., 2001). For even stronger forcing the population fluctuations can become chaotic (Ireland et al., 2004).

Also of importance is the configuration of the forcing, i.e. which parts of the system are directly affected by the external forcing. For childhood diseases seasonal forcing works predominantly through infection transmission but in other cases other processes can be targeted by the forcing as well. For example, for house finches infected with bacterial conjunctivitis (*Mycoplasma gallisepticum*) breeding occurs in the summer while outbreaks of the infection usually occur in the fall and winter when there is social aggregation (Altizer et al., 2006; Hosseini et al., 2004). In contrast, for harbour seals (*Phoca vitulina*) infected with the phocine distemper virus, breeding and social aggregation occur at the same time,

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when the seals haul out (Altizer et al., 2006; Swinton et al., 1998). When there are multiple components to the forcing they can interact to reinforce responses or, alternatively, to bring about interference between these responses. The spectrum of behaviour from reinforcement to interference is the result of high sensitivity of the amplitude of the population oscillations to the lags between components. There is a half cycle lag in eco-epidemiological systems if maximum predation is in the summer (e.g. the winter spent in hibernation) and maximum prey infection occurs in the winter (due to higher prey density). If instead predation is constant, as it might be in the house finch ecosystem with domestic cats as predators, then there would be a half cycle lag between breeding and infection transmission.

An important case of multiple forcing is when some of the components are the result of human activity. One example of this is the periodic harvesting of a plant or animal population seasonally driven and vulnerable to infection. Such an interaction between anthropogenic and environmental forces can have undesirable consequences if reinforcement rather than interference takes place. Harvesting, for example, can increase the incidence of infection (Choisy and Rohani, 2006). On the other hand, the ability of management to manipulate some forcing components suggests the possibility of controlling the system through “countercyclical” methods, using periodic forcing to offset the harmful effects of the environmental forcing, for example, species extinction or large scale disease epidemics. Understanding when the different components of the external forcing will lead to reinforcement and when interference will result is therefore important.

We address this problem in this paper, studying the behaviour of continuous-time models subject to multi-component periodic external forcing. For weak forcing, when the system can be linearised, the analysis can be entirely algebraic. For stronger forcing, we explore through simulation whether well chosen forcing lags are still able to switch the system from reinforcement to interference when subharmonics and chaos are present.

Throughout the discussion the response curve for a system population is used to visualise important aspects of the analysis. To construct this curve the amplitude or the maximum (over a cycle) of the chosen population is plotted against the forcing period  $p$ . This is the “natural” way of studying resonance since resonance is primarily concerned with the relationships between three different periods, the forcing period itself,  $p$ , the period  $p_1$  of the populations (in response to the forcing) and the (leading) natural period of the unforced system  $p_0$ . This natural period is the period of the damped oscillatory path taken by the (assumed) overcompensating system to bring the system back into equilibrium. This is the dynamical state that is excited by the forcing to bring about resonance. In the simplest cases this excitation shows up as a peak on the response curve when the external and natural periods coincide. Period  $p_0$  is related to and can be calculated from the imaginary part of the leading complex eigenvalue of the Jacobian of the unforced system. (In high dimensional systems there can be more than one natural state that can be excited and hence more than one complex Jacobian eigenvalue and more than one natural period. However, the impact of such secondary excitation can be quite small.)

For definiteness we focus on resonant amplification in (eco-) epidemiological systems. The problem is how to suppress the often large scale epidemics that can periodically occur in a population host to a pathogen. To study this problem we use the simplest non-trivial model available, the SI model ( $S$ =susceptible,  $I$ =infectious population). It has a simple structure without explicit built in delays (e.g. periods of latency or immunity) but is still liable to resonant amplification. Management control will be exercised mostly through host culling.

In the final part of the discussion we focus on one particular feature of the SI model behaviour, the occurrence of seemingly

high period fluctuations in system populations when the external forcing has low period. The introduction of a model describing the dynamics of a pathogen with two strains shows that the mechanisms that produce high periodicity for the SI model may also work in more complicated systems. As well, new ways of creating high periodicity are explored, including noise excitation.

## 2. Multi-component forcing

### 2.1. The fundamentals

The SI epidemiological model is defined by the equations:

$$dS/dt = aH - sH^2 - bS - \beta SI + \gamma I - c_1 PS \quad (1a)$$

$$dI/dt = \beta SI - dI - c_2 PI \quad (1b)$$

$$dH/dt = rH - sH^2 - \alpha I - c_1 PS - c_2 PI \quad (1c)$$

$S$ ;  $I$ ;  $H=S+I$  denotes the susceptible; infectious; total population while  $a$ ;  $b$ ;  $r=a-b$  measures per capita birth rate; mortality; net growth rate at low population levels. Self-regulation is introduced through the carrying capacity  $K$ , where  $s=r/K$ . Parameter  $\alpha$ ;  $\gamma$ ;  $d=b+\alpha+\gamma$  describes virulence; recovery rate; loss rate from the infectious state while  $\beta$  is the infection transmission constant. For the moment we take  $P=0$  explaining its significance later. Eqs. (1b), (1c) with  $P=0$  and (1a) redundant define for us Model 1.

To model periodic external forcing we will suppose that there are two components, working through birth rate ‘ $a$ ’ and infection transmission ‘ $\beta$ ’. Precisely

$$a = a_0(1 + \delta_1 \cos(\omega t)), \quad \beta = \beta_0(1 + \delta_2 \cos(\omega t + \phi)) \quad (2)$$

where  $a_0$ ,  $\beta_0$  are average values,  $\delta_1$ ,  $\delta_2$  the forcing strengths,  $p=2\pi/\omega$  the common forcing period and  $\phi$  the phase between components. The phase can be interpreted as a lag of  $(-\phi/2\pi)$ , so if  $\phi = -\pi/2$  then  $\beta$  is lagging ‘ $a$ ’ by a quarter cycle but if  $\phi = +\pi/2$  then ‘ $a$ ’ is lagging  $\beta$  by a quarter cycle. Sinusoidal functions will be used throughout to model periodicity because of the analytical advantages of so doing and because the qualitative features of the dynamics are usually not sensitive to the precise functional form chosen (MacDonald, 2007).

With weak forcing (i.e.  $\delta_1, \delta_2 \ll 1$ ) the model equations can be linearised with explicit formulae given for the amplitudes of the population oscillations (see Appendix B, (B9) and (B10)). These amplitudes are written as a ratio with the denominator (complex) zeros generating the resonance peaks and the numerator moderating or distorting these peaks. If the numerator for one of the populations becomes zero (for some value of the external period  $p$ ) then the oscillations for that population are eliminated entirely and for all time (for that value of  $p$ ) leaving the population at its (unforced) equilibrium value. This is an important possibility with significant policy implications.

The conditions for the numerator to be zero, when there are two forcing components, are found algebraically in Appendix B (B7). These are the conditions for the individual responses generated by the two forcing components to have the same magnitude and to be exactly out of phase, reflecting the fact that responses add in a linear system. These two conditions relate the forcing period  $p$ , the phase  $\phi$  and relative forcing strength  $\theta_0 = \delta_1/\delta_2$ . Given one of these forcing parameters, the zero amplitude conditions determine the values of the other two if there exists a feasible solution with  $p$  positive, which is not always the case.

To illustrate the zero numerator conditions (B7) we analyse Model 1 with the parameters listed as set 1 in Appendix A. In Fig. 1A with  $\theta_0=1.5$  and  $\phi = +\pi/2$ , we plot the response curve for

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