Population biology of infectious diseases: Part I

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If the host population is taken to be a dynamic variable (rather than constant, as conventionally assumed), a wider understanding of the population biology of infectious diseases emerges. In this first part of a two-part article, mathematical models are developed, shown to fit data from laboratory experiments, and used to explore the evolutionary relations among transmission parameters. In the second part of the article, to be published in next week’s issue, the models are extended to include indirectly transmitted infections, and the general implications for infectious diseases are considered.

ANY contemporary ecology text contains at least one chapter devoted to predator–prey interactions. The discussion typically embraces field and laboratory observations along with simple mathematical models, and emphasizes how the densities of both prey and predator populations may be regulated by their interaction.

In natural communities, however, an accumulating body of evidence suggests that parasites (broadly defined to include viruses, bacteria, protozoans, helminths and arthropods) are likely to play a part analogous, or at least complementary, to that of predators or resource limitation in constraining the growth of plant and animal populations. Examples from the laboratory are Park’s experiments in which the sporozoan parasite Adelina drastically reduced the population density of the flour beetle Tribolium castaneum, and in certain circumstances reversed the outcome of its competition with T. confusum, and Lancini’s studies of the way the ectoparasitic water mite Hydrophantes tenuabilis influences the population dynamics of the aquatic insect Hydrometra myrae. Various studies have indicated the importance of infectious disease as a mortality factor in populations of wild mammals, and as possibly the predominant such factor in bird populations. For example, among bighorn sheep in North America the main cause of death probably is infection by the lungworms Protostrongylus stilesi and P. rahi, which then predispose the hosts to pathogens causing pneumonia. On a grander scale, Pearsall and others have suggested that the geographical distribution of most artiodactyl species in Africa today is largely set by a pandemic of rinderpest that occurred towards the end of the nineteenth century; the numerical simulations of Hilborn and Sinclair confirm that rinderpest can have a big influence on wildebeest population levels. Several authors have argued the general case for infectious diseases as regulators of their host populations.

More broadly, it is likely that interplay between the pathogenicity of viral, bacterial, protozoan or helminth infections and the nutritional state of the host contributes importantly to the density-dependent regulation of natural populations, with the parasites greatly amplifying the effects of low levels of nutrition. Such phenomena are largely responsible for the dramatic differences between age-specific survival probabilities for people in developed and underdeveloped countries.

Indeed, McNeill and others have speculated that many of the broader patterns of human history are to be interpreted in terms of the evolving relationships between man and his diseases.

Although there does exist a large and mathematically sophisticated literature dealing with the transmission dynamics of parasitic infections of many kinds, this literature almost invariably assumes the host population to have some constant value, and then seeks to answer such questions as: Can the infection be stably maintained in the population? Is it endemic or epidemic? What is the time course (in terms of susceptibles, infectives and recovered individuals) of the infection when introduced into a virgin population? This assumption that the total host population is effectively constant derives from a history of medical interest in human diseases (predominantly in developed countries), where population densities do usually remain roughly constant on the time scale appropriate to the pathology of most diseases. On the other hand, in the ecological and parasitological literature attention has recently been given to the population dynamics of host–parasite associations, with particular emphasis on the way protozoan, helminth and arthropod parasites can depress the natural growth rate of their host populations. Our review aims to weave together these medical and ecological strands, concentrating on the way parasitic infections can influence the growth rate of their host populations.

The article is being published in two parts. This first part begins with a survey of the diverse array of infectious organisms and of their associated life cycles. We then show how a very simple dynamic model can provide a remarkably detailed explanation of a classic series of experiments on infections in laboratory populations of mice. This success gives the confidence to enable us to proceed into areas less well supported by good data, and we next discuss microparasitic infections with direct life cycles in natural populations; particular attention is given to the evolutionary relations among transmission parameters, the factors which determine the pattern of disease behaviour within populations of hosts and the population consequences of acquired immunity.
Both microparasites and macroparasites may complete their life cycles by passing from one host to the next either directly or indirectly via one or more intermediate host species. Direct transmission may be by contact between hosts (for example, venereal diseases) or by specialised or unspecialised transmission stages of the parasite that are picked up by inhalation (such as common colds), ingestion (such as pinworm) or penetration of the skin (such as hookworm). Indirect transmission can involve biting by vectors (flies, mosquitoes, ticks, and others) that serve as intermediate hosts, or penetration by free-living transmission stages that are produced by molluscan or other intermediate hosts. In other cases, the parasite is ingested when an infected intermediate host is eaten by the predatory or scavenging primary host. A special case of direct transmission arises when the infection is conveyed by a parent to its unborn offspring (egg or embryo), as can occur in syphilis and rubella and for many viral infections of arthropods, this process has been termed 'vertical transmission', in contrast to the variety of 'horizontal transmission' processes discussed above.

The natural historian's main concern is often the recondite biological details that make each parasitic infection unique. In contrast, our aim is to understand the basic similarities and differences in terms of: the number of population variables (and consequent equations) needed for a sensible characterisation of the system; the typical relations among the various rate parameters (such as birth, death and recovery rates, transmission coefficients); and the form of the expressions describing the transmission processes. In the absence of such a unified framework, each disease tends to develop its own arcane literature.

**Experimental epidemiology:** infectious diseases as regulators of laboratory populations of mice

Although there are relatively few studies of the influence of disease upon the dynamics of laboratory populations of mice, there is a remarkably detailed body of work of Greenwood et al., subsequently extended by Fenner. These experiments, on laboratory populations of mice infected with various viral and bacterial diseases, have some simplifying features which make them particularly amenable to theoretical analysis. Specifically, the space available to the mice was adjusted to keep the population density constant as absolute levels changed; in addition adult mice were introduced at specified rates, so that the basic process was an immigration–death one (removing the time lags and other complications attendant upon recruiting to the population by natural birth processes). In short, many density-dependent complications are avoided by the design of the experiments.

We now outline a simple model that captures the essentials of these experiments, and discuss its fit to the data for two microparasites: one a bacterium (Pasturella muris); the other a virus (ectromelia, a poxvirus). Both parasites multiply directly within the host and induce a long-lasting immunity to reinfection (mice show some loss of immunity to reinfection by Pasturella, but the immune response to ectromelia seems to be lifelong).

Using notation that will be standard throughout this review, we define the absolute number of susceptible (uninfected), infected and immune mice to be \( X, Y \) and \( Z \), respectively. The total number of mice, \( N = X + Y + Z \), is not assumed to be some independently-set constant, but is set by the dynamics of the infection. \( A \) is defined as the rate at which mice are introduced (\( A = 2 \) means 2 mice introduced per day), and \( b \) the natural mortality rate; in the absence of the disease, the mouse population will equilibrate at around \( N^* = A/b \). The infection is a direct one, for which the conventional assumption is that the rate at which mice acquire the infection is proportional to the number of encounters between susceptible and infected mice, being \( bXY \) where \( b \) is some 'transmission coefficient'. The mortality rate for infected mice is taken to be \( b + \alpha \), with \( \alpha \)
representing the mortality caused by the disease; there is also a recovery rate v. Recovered mice are initially immune, but this immunity can be lost at a rate y (or permanent immunity, as for ectromelia, \( y = 0 \)). These assumptions lead to the following equations for the dynamics of the infection:

\[
\begin{align*}
\frac{dX}{dt} &= A - bX - \beta XY + \gamma Z \\
\frac{dY}{dt} &= \beta XY - (b + \alpha + v) Y \\
\frac{dZ}{dt} &= v Y - (\gamma + b) Z
\end{align*}
\]

Adding all three, the equation for the total population of mice is

\[
\frac{dN}{dt} = A - bN - \alpha Y
\]

This system of equations (which is similar to that illustrated schematically by Fig. 3) differs from usual epidemiological models in that N is a dynamical variable, rather than some specified constant.

The equations have a stable equilibrium solution with the disease maintained in the population if, and only if,

\[
A/b > (\alpha + b + v)/\beta
\]

Failing this, the disease dies out, and the population settles to its immigration–death equilibrium value at \( N^* = A/b \). If equation (5) is satisfied, the disease persists, and the total population is depressed below this infection-free level to the lower value

\[
N^* = \frac{A + D(\alpha + b + v)/\beta}{b + D}
\]

Here \( D \) is defined for notational convenience as

\[
D = \frac{1}{\gamma + b}
\]

Note that the important threshold phenomena, which enter directly when \( N \) is a specified constant, appear in a more subtle form when \( N \) is itself determined by the dynamics of the disease.

In their experiments on the maintenance of pasteurellosis, \( P. muriis \), in mouse populations Greenwood et al.\(^{35,46} \) introduced new mice at rates ranging from \( A = 0.33 \) to \( A = 6 \) mice per day. The quantities \( b \), \( \alpha \) and \( v \) can be crudely estimated from life tables for uninfected populations, and from case mortality and recovery rates (we get \( b = 0.006 \), \( \alpha = 0.06 \), \( v = 0.04 \) days\(^{-1} \)). Direct estimate of the parameters \( \beta \) and \( \gamma \) is more difficult.

Using data from Greenwood et al.\(^{46,47} \) (and reanalysing to discard the transient initial population values en route to the steady state), we obtained the experimental results shown in Fig. 1a for the equilibrium mouse population \( N^* \) as a function of \( A \). These data accord well with the linear relation between \( N^* \) and \( A \) predicted by equation (6). Furthermore, the parameters \( \beta \) and \( \gamma \) may now be roughly estimated from the fit between the theoretical straight line, equation (6), and the data for \( N^* \) versus \( A \) (we estimate \( \beta = 0.0056 \), \( \gamma = 0.021 \) days\(^{-1} \)).

In Fig. 1a, the dashed line depicts the equilibrium mouse population in the absence of the disease, \( N^* = A/b \). The intercept of this line with the linear fit to the data for \( N^* \) in the presence of the disease yields the threshold immigration rate, \( A_r \), below which equation (5) is violated and the disease cannot persist; Fig. 1b magnifies this aspect of Fig. 1a. We estimate \( A_r = 0.11 \) mice per day (corresponding to an equilibrium population of about 19 mice). Greenwood et al. suggested \( P. muriis \) was always maintained in mice populations, but their lowest introduction rate was \( A = 0.33 \).

With \( \beta \) and \( \gamma \) determined from Fig. 1a, we now have a parameter-free prediction of the temporal development of the infection for any initial number of mice \( N(0) \) and introduction rate \( A \). Two such fits between theory and data are shown in Figs. 1c and 1d, for \( A = 6 \) and \( A = 0.33 \), respectively. Note the propensity to damped oscillations at relatively small \( A \) values. Bearing in mind the complete absence of adjustable parameters, both the fits are extremely encouraging, and strongly suggest that simple deterministic models can be useful even when the host population is small.

How much does the disease depress the mouse population below the level that would pertain in its absence? This general question is answered in Fig. 1e and f, which shows \( N^* \) as a function of disease pathogenicity \( \alpha \), for \( A = 6.0 \) and \( A = 0.33 \) respectively. Two significant points emerge.

First, the maximum depression of the host population is achieved by a disease of intermediate pathogenicity \( \alpha \), for \( A = 6.0 \) and \( A = 0.33 \) respectively. Two significant points emerge.

Second, note that the higher the immigration rate \( A \), the greater the degree of depression of the host population (relative...
to the disease-free equilibrium value). This suggests that diseases caused by microparasites are more likely to persist within, and cause severe reduction of, host populations with high birth (or immigration) rates; this phenomenon derives essentially from the high inflow of susceptibles.

Greenwood et al., and later Fenner, also studied the effects of the mouse pox virus, ectromelia. An analysis, akin to that just outlined, leads to similarly encouraging agreement between our simple theory and the experimental data for ectromelia in laboratory populations of mice. Some of these results are summarised in Fig. 2.

For both P. muris and ectromelia, the actual value of the pathogenicity parameter $\alpha$ (indicated by the dashed vertical lines in Figs 1e, f and 2b) lies around the value that induces maximum depression of the host population. Is this coincidence, or does it reflect evolutionary pressures? The question is intriguing, but difficult to pursue in the absence of a larger body of information about a wider range of diseases.

In brief, the theory and the facts of these experiments are in accord in showing how infectious diseases can stably regulate their host populations below disease-free levels. They also show the existence of a critical host density (directly tied to the rate at which new susceptibles are introduced, either artificially in the laboratory, or by births in the natural world), below which the infection cannot be maintained. In this sense, equation (5) replaces the threshold condition of conventional epidemiological models in which the host population is an independently determined constant.

**Microparasitic infections as regulators of natural populations**

The models discussed above are only half-way to a fully dynamic description of host–parasite interactions. Although the death rates are set by natural processes, and are influenced by the parasites, the ‘birth’ processes are determined artificially by the rate of introduction of new mice. We now consider what happens when the birth rates are also set by natural processes intrinsic to the host population.

To begin with, we focus on diseases caused by microparasites that are transmitted directly, and ask three main questions: what biological characteristics of an infection determine its impact on host population growth; what are the population consequences of immunological responses; and what conditions lead to endemic or to epidemic infections?

Consider the simple situation of an infection whose transmission processes are as described by equations (1)–(3), except that now the new individuals arise by natural births. This situation is illustrated schematically in Fig. 3. If the *per capita* birth rate is $a$, independent of whether the individual is susceptible, infected or immune, then the net birth rate term is $a(X + Y + Z)$, and the dynamical system of equations (1)–(3) is replaced by

$$
\frac{dX}{dt} = a(X + Y + Z) - bX - \beta XY + \gamma Z
$$

$$
\frac{dY}{dt} = \beta XY - (\alpha + b + v) Y
$$

$$
\frac{dZ}{dt} = vY - (b + \gamma)Z
$$

The total population of hosts, $N = X + Y + Z$, obeys

$$
\frac{dN}{dt} = (a - b)N - \alpha Y
$$

Equivalently it is useful to define the intrinsic growth rate $r = a - b$ of the disease-free population and to write $y = Y/N$ as the ‘prevalence’, or fraction of the host population that are infected. This gives

$$
\frac{dN}{dt} = (r - \alpha y)N
$$

One of two circumstances now arises. If

$$
\alpha > r \left[ 1 + \frac{v}{b + \gamma} \right]
$$

the disease regulates the host population to a stable value $N^*$. This disease-determined population level is

$$
N^* = \frac{\alpha(\alpha + b + v)}{\beta[\alpha - r(1 + v/(b + \gamma))]} \quad (14)
$$

Of this steady population, the fraction infected is given trivially from equation (12) as

$$
y^* = r/\alpha
$$

Conversely, if equation (13) is not satisfied, the system of equations (8)–(10) eventually settles to a state in which the total population grows exponentially at a rate $\rho$ given by

$$
\rho = \frac{B^2 - (b + \gamma)(\alpha - r) + rv}{(\alpha - r)^2 - B}
$$

with $B = \frac{1}{2}(\alpha + b + v + \gamma - r)$. This population growth rate is necessarily less than the disease-free one, $\rho < r$. Asymptotically, the exponentially growing total population contains a constant number of susceptibles $X$, with essentially all individuals being infected or immune. The asymptotic prevalence of infection is

$$
y = Y/N \rightarrow (r - \rho)/\alpha
$$

Note the similarities and differences between these conclusions and those for conventional models in which the total population is set at some constant value. In this crude model there are no density-dependent regulatory effects other than the
either case, if NV is initially less than a threshold value, population settles to the value N given by equation (14). In disease itself and the population 'runs away' (maintaining the

<table>
<thead>
<tr>
<th>Parasite</th>
<th>Incubation period (days)</th>
<th>Duration of infectiousness (communicability)</th>
<th>Infectiousness</th>
<th>Duration of immunity</th>
<th>Lifespan of infective stage (pathogenicity)</th>
<th>Case mortality rate (transmission coefficient H)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Measles virus</td>
<td>9–12</td>
<td>5–7</td>
<td>High</td>
<td>Lifelong</td>
<td>Very short</td>
<td>Low–high</td>
</tr>
<tr>
<td>Smallpox virus</td>
<td>12–14</td>
<td>10</td>
<td>Medium</td>
<td>Lifelong</td>
<td>High</td>
<td>H</td>
</tr>
<tr>
<td>Rubella virus</td>
<td>17–20</td>
<td>14</td>
<td>Medium</td>
<td>Lifelong</td>
<td>Very short</td>
<td>Low, V</td>
</tr>
<tr>
<td>Mumps virus</td>
<td>10–20</td>
<td>7</td>
<td>Medium</td>
<td>Lifelong</td>
<td>Short</td>
<td>Low</td>
</tr>
<tr>
<td>Bordetella pertussis (whooping cough)</td>
<td>7–10</td>
<td>14+</td>
<td>High</td>
<td>Lifelong</td>
<td>Very short</td>
<td>Medium</td>
</tr>
<tr>
<td>Polio virus</td>
<td>5–20</td>
<td>Long</td>
<td>High</td>
<td>Lifelong</td>
<td>Medium</td>
<td>Low</td>
</tr>
<tr>
<td>Varicella zoster virus (chicken pox and shingles)</td>
<td>13–17</td>
<td>20–30</td>
<td>High</td>
<td>Lifelong</td>
<td>Very short</td>
<td>Low (V)</td>
</tr>
<tr>
<td>Herpes simplex virus</td>
<td>5–8</td>
<td>Long</td>
<td>Medium</td>
<td>Lifelong</td>
<td>Very short</td>
<td>Very low</td>
</tr>
<tr>
<td>Cytomegalovirus</td>
<td>10–14</td>
<td>30</td>
<td>Long</td>
<td>Lifelong</td>
<td>Very short</td>
<td>Very low</td>
</tr>
<tr>
<td>Epstein–Barr virus</td>
<td>107</td>
<td>Long2</td>
<td>Long2</td>
<td>Short</td>
<td>Medium</td>
<td>High</td>
</tr>
<tr>
<td>Clostridium tetani (tetanus)</td>
<td>7+</td>
<td>21–30</td>
<td>Low</td>
<td>Long</td>
<td>High</td>
<td>H</td>
</tr>
<tr>
<td>Salmonella typhi (typhoid)</td>
<td>10–14</td>
<td>30+</td>
<td>Low</td>
<td>Medium</td>
<td>High</td>
<td>H</td>
</tr>
<tr>
<td>Bacillus anthracis (anthrax)</td>
<td>3–77</td>
<td>Very long</td>
<td>Very long</td>
<td>Very short</td>
<td>Very short</td>
<td>High</td>
</tr>
<tr>
<td>Corynebacterium diphtheriae (diphtheria)</td>
<td>2–6</td>
<td>20</td>
<td>Medium</td>
<td>Long</td>
<td>Medium</td>
<td>High</td>
</tr>
</tbody>
</table>

The qualitative patterns revealed in Fig. 4a are reminiscent of those shown by human population growth,55,56 between the beginning of the Agricultural Revolution (some 10,000 years ago) and the onset of the Industrial-Scientific Revolution (around 300 years ago). In the first 5,000 yr, the global population increased about 20-fold, from around 5 million to around 100 million. The next 5,000 yr saw only a roughly 5-fold increase to around 500 million in the sixteenth century. It may not be unduly fanciful to speculate that the rise of human conglomerations to levels capable of maintaining directly transmitted microparasitic diseases, and the accompanying depression of population growth rates, is at least partly responsible for the observed patterns.

### Population consequences of immune responses

Although the nature of immunological responses by individual hosts to their specific pathogens has received much attention in recent years,5,45,55, relatively little thought has been given to the population consequences of acquired immunity26,32,55 (sometimes called ‘herd immunity’ effects). The general insights just called from equations (13)–(18) can be usefully illuminated by a numerical example. Figure 4a shows the growth of a fictitious human population (from an initial size of 50,000) subject to a virus disease and under various assumptions about the duration of immunity. The vital rates and transmission parameter values are as detailed in the figure caption. In more homely terms, they represent a growth rate of the disease-free population of around 3% per annum, a case mortality of about 30% (similar to measles in malnourished human populations with no previous exposure51), duration of the infection around 4 weeks, and a transmission coefficient 7 that implies a threshold population density of N* = 380,000 people.

In all cases in Fig. 4a, the disease is not maintained and the population growth at its intrinsic 3% rate if it is below the threshold value N*. Above this point, the population’s fate depends on the nature of the immune response. If the duration of the immunity to reinfection (1/y) is of short to medium length (less than about 20 yr), the disease is able to regulate the host population at the stable level N* of equation (14). If the disease induces hardly any immunity (γ large), this equilibrium level N* will be close to the threshold N* for maintenance of the disease. Conversely, if the duration of immunity is above 20 yr, the population continues to grow exponentially at some rate lower than 3%; life-long immunity (γ = 0, as for measles) results asymptotically in 1.6% per annum growth. This example makes plain the important part immunity plays in determining the population consequences of a disease.

### Epidemic and endemic patterns of diseases

Epidemic diseases are characterised by rapid changes in the prevalence of infection. Often such infections disappear from a particular host population for short or long periods. Conversely, endemic infections persist for long times, showing relatively little fluctuation in prevalence. Note that in our dynamic models, equations (8)–(10), the disease always becomes endemic, in the sense that the host population grows to the level N > N∗, whereupon the disease is maintained. The prevalence settles to the steady value y* = r/a if the disease controls the population, and to y* = (r − ρ)/a if the population still grows.
Whether a disease will persist endemically or as recurrent epidemics, which in either case is very small. That is, if $r < a$, it is likely that $N_f > N_y$, introduction of the infection results in a classical epidemic. When $r > a$, the rate is much smaller than the case mortality rate, $r < a$. Then, if the host population grows at a rate $r$, then the introduction of the above disease into a virgin population of hosts where the equilibrium prevalence level is low. As for (b), except now the equilibrium prevalence is relatively high.

It is well known, however, that diseases which induce long-lasting immunity often exhibit periodic or episodic ‘face out’, even within relatively large host populations. In particular, the classic work of Bartlett on measles epidemics has suggested the importance of stochastic effects in determining whether a disease will persist endemically or as recurrent epidemics.

Without entering into the detailed complications of a stochastic formulation, we can use the above model to get some qualitative insights about these patterns. Of particular importance is the rate at which new susceptibles appear; hence the general correlation between endemicity and host population size, and the observation that the host birth rate is central. Specifically, consider the case where the hosts’ intrinsic growth rate is much smaller than the case mortality rate, $r < a$. Then, if $N > N_f$, introduction of the infection results in a classical epidemic (see Fig. 4b): the prevalence first rises, attains a peak, and then falls to the value given by equation (15) or equation (17), which in either case is very small. That is, if $r < a$, it is likely that prevalence settles to be so small as to give a high probability for stochastic ‘fade out’ and epidemicity. This can be true even for diseases potentially capable of regulating the host population if $r/a$ and $N^*$ are both sufficiently small. In addition, epidemics can occur even when $r > a$ if the disease does not regulate the host population but merely slows its growth rate slightly, so that $r < a$. The details of the interplay among parameters that leads to $a + r > r > a$ is complicated, and can be deduced from equation (16) for $r$. On the other hand, if neither $r$ nor $r < a$ are a lot smaller than $a$, the disease is likely to be endemic, with relatively high values of $y^*$ making stochastic extinction of the disease improbable.

The effects of microparasitic infections on the dynamics of animal populations depend on the ecology of the interactions between host and parasite. These patterns of disease behaviour involve four principal factors, namely: the host providing a habitat for the parasite; the degree to which the parasite induces immunity (or decreases the reproductive capacity of the host); the extent to which the host acquires immunity; and the necessity of transmission from one host to the next. Overlaid on these factors are many biological complications, specific to individual host–parasite associations, whose sequential action is determined by life cycle structure.

In the second part of this article, we show how a common set of factors is involved in the dynamics of all infectious diseases, whether they are caused by viral or helminth agents, and whether they are transmitted directly or indirectly between hosts.

Possible optical observation of the companion star to the binary pulsar PSR1913 + 16

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Deep R band CCD photographs of the region surrounding the binary pulsar PSR1913 + 16 have been obtained with the 4-m telescope of the Kitt Peak National Observatory. Using an accurate astrometric solution, we find a star with $M_R = 20.9$ at the precise coordinates of the pulsar. Possible interpretations of this result, including the unlikely (<3%) possibility that it is an accidental superposition of a field star, are discussed. If this object is the physical companion to the pulsar, it is probably a helium star.

The discovery of the binary pulsar PSR1913 + 16 has created substantial interest as a system in which various general relativistic effects may be measured. The ability to perform these experiments relies critically on two factors: (1) that the pulsar clock should behave in a regular way and (2) that the companion star must be treatable as a point mass. The former point seems to be satisfied so the major remaining issue for tests of relativity to proceed is how to determine the nature of the companion star. Since the optical extinction is probably modest ($A_V = 3.3$; ref. 6) we expect that a non-compact companion should be observable with a sensitive detector on a large telescope. Thus, sensitive observations could establish valuable constraints on the size of the companion star. The small observed apsidal motion in the system already limits the companion star to being either a helium star or a compact object.

The observations
We have used the JPL CCD array (400x400 pixels; 0.45" per pixel) at the prime focus of the 4-m Mayall telescope to observe the region of the binary pulsar PSR1913 + 16. Our