Chapter 17
Diseases: Old and New

The pathogens infecting a widely distributed host species must either: (i) have an alternative host; (ii) be able to survive in a dormant state; or (iii) be non-destructive to their host. For pathogens of diploid hosts that have an obligatory sexual mode of reproduction, a particularly effective strategy is that of being sexually transmitted and the movement of the host allows the pathogen to disperse. There are two major problems with this STD type of strategy: First, a sexually transmitted disease must be, at least relatively, a “gentle pathogen” or a “prudent predator” and remain as such and not evolve towards greater virulence. The second, and quite important problem is that such a pathogen must, to a large extent, be able to avoid destroying its host’s offspring. Both factors would seemingly be countermanded by the immediate selective advantage of being more virulent and producing more propagules of the pathogen. The first problem has been extensively discussed in the literature. A new proposal, presented here, is that mechanisms evolved to protect a previously infecting pathogen from other subsequent infecting pathogens and this may incidentally protect the host and its offspring. The second problem is related to the first and is a major factor with animal hosts. In large part, the “temperate” pathogens of hosts with immune systems actually depend on the host’s immune system (and other protective systems) to keep the pathogens themselves from damaging the host too quickly. The lack of an effective immune system in the fetus (even with some passive maternal immunity) and in the early life of newborns implies that the persistence of STD pathogens may be favored by the limited protective systems of the fetus’s mother to some degree. It is suggested that the mucosal immune system of the host may be able to reduce the danger of infection and of disease to the offspring. In the long run, this is favorable for the STD pathogen because then the young members of the host population can grow to become sexually mature. It is suggested here that the selection force driving both the generation of the pathogen to become “gentle” to its host and to limit the degree of infection of the fetus and neonate is the consequence of evolution of the pathogen to be able to inhibit other pathogens (even of the same species) from infecting the pathogen’s host.
INTRODUCTION

This chapter is mainly about viruses in the Eukaryote human. This is because the relationship of host and pathogen are better known or at least thoroughly speculated about. However the relationship of the host and pathogen is the same no matter what the details of their positions in the tree of life and the molecular biological abilities of either.

Much host-pathogen biology, including that relevant to human disease, can be inferred through the study of viruses that infect bacteria, such as the bacteriophages T4 and Lambda, $\lambda$. These viruses have elaborate mechanisms to deal with their host, with themselves, with each other, and with other pathogens even of their own kind. The experimental elucidation of the elaborate molecular biological mechanisms of viruses to keep out other viruses have pointed a way that counters the evolutionary tendency of the pathogen to become more virulent to the host. An analysis of the molecular biological mechanisms and how they can function for the “good” of the host for bacterial viruses is being prepared and partially presented here. Such a model is to be contrasted with earlier models based on kin and on group selection (summarized in Anderson and May (1991) and Frank (1996). The paradigm presented here may well be quite general and it will be assumed that evolution can create and maintain a “gentle” pathogen in this way. According to this generalized model developed for viruses of prokaryotes, the lysogenic pathogen persists indefinitely in a temperate state in spite of the clear short-term selective advantage of changing to become virulent. This counter-intuitive behavior is accounted for by the existence of a positive selection mechanism functioning against other pathogens. Incidentally this leads to it being more “gentle” towards its host. I will use the term “gentle” in quotes to denote that the pathogen has a quid pro quo in the interaction with the host. Although proposed and justified for host-parasite relations in the virology of bacteria, I now suggest that this mechanism could function in host-parasite interactions no matter what organisms are involved. As the primary focus in this chapter, it will be presumed to function in the interactions of primates and retroviruses with as yet unknown specifics, and in particular to humans and the HIV virus.

Two topics will be considered: First, the problems that any pathogen would have surviving on a sparse, but clustered, host population, such as cave-dwelling primitive humans. The alternatives to developing the habit of being “gentle” to its host while being an STD are either: i) acquiring and using an ability to remain dormant for long periods of time; or ii) of also being able to infect an ubiquitously and commonly occurring alternative host species. These two possibilities will not be examined further here, but they do occur in nature. The option of being “gentle” to the host is the main topic here with respect to