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Sexually transmitted diseases of insects: distribution, evolution, ecology and host behaviour

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ABSTRACT

Sexually transmitted diseases (STDs) of insects are known from the mites, nematodes, fungi, protists and viruses. In total 73 species of parasite and pathogen from approximately 182 species of host have been reported. Whereas nearly all vertebrate STDs are viruses or bacteria, the majority of insect STDs are multicellular ectoparasites, protistans or fungi. Insect STDs display a range of transmission modes, with 'pure' sexual transmission only described from ectoparasites, all of which are mites, fungi or nematodes, whereas the microparasitic endoparasites tend to show vertical as well as sexual transmission. The distribution of STDs within taxa of insect hosts appears to be related to the life histories of the hosts. In particular, STDs will not be able to persist if host adult generations do not overlap unless they are also transmitted by some alternative route. This explains the observation that the Coleoptera seem to suffer from more STDs than other insect orders, since they tend to diapause as adults and are therefore more likely to have overlapping generations of adults in temperate regions.

STDs of insects are often highly pathogenic, and are frequently responsible for sterilizing their hosts, a feature which is also found in mammalian STDs. This, combined with high prevalences indicates that STDs can be important in the evolution and ecology of their hosts. Although attempts to demonstrate mate choice for uninfected partners have so far failed it is likely that STDs have other effects on host mating behaviour, and there is evidence from a few systems that they might manipulate their hosts to cause them to mate more frequently. STDs may also play a part in sexual conflict, with males in some systems possibly gaining a selective advantage from transmitting certain STDs to females.

STDs may well be important factors in host population dynamics, and some have the potential to be useful biological control agents, but empirical studies on these subjects are lacking.

Key words: sexually transmitted disease, STD, insect, host-pathogen, parasite mediated sexual selection, parasite manipulation of host behaviour, evolution of parasitism, sexual conflict.

CONTENTS

I. INTRODUCTION

There is now an increasing realization that sexually transmitted diseases (STDs) are common throughout the animal kingdom (Sheldon, 1993; Lockhart, Thrall & Antonovics, 1996). Theoretical models have demonstrated that STDs can have considerable effects on the population dynamics of their hosts (Getz & Pickering, 1983; Thrall, Antonovics & Hall, 1993), and STDs have been implicated as important factors in determining population sizes in a number of wild species (e.g. chlamydia in koala bears, *Phascolarctos cinerus*, Smith & Dobson, 1992; Jackson *et al.*, 1999). As well as influencing host population dynamics, STDs may also have important effects on the evolution of their hosts, especially on the host's mating system (Hamilton, 1990; Sheldon, 1993; Hurst *et al.*, 1995; Loehle, 1995, 1997; Able, 1996; Thrall, Antonovics & Bever, 1997; Knell, 1999; Boots & Knell, 2002; Kokko *et al.*, 2002). Empirically, STDs are now known from a wide variety of animal taxa. Sheldon (1993) and Lombardo (1998) discussed STDs of birds, and Lockhart *et al.* (1996) reviewed 200 STDs from 27 orders of animal hosts.

It is not a surprise that the best-known STDs are found in mammalian hosts, and the important features of mammalian STDs have been reviewed by Smith & Dobson (1992) and Lockhart *et al.* (1996). Smith & Dobson (1992) emphasised the importance of transmission dynamics in the ecology of STDs (see section XI for a discussion of this). Lockhart *et al.* (1996) conducted a detailed analysis comparing mammalian STDs to conventionally transmitted diseases. They found that mammalian STDs tend to reduce host fertility rather than increasing mortality, cause more chronic infection and have narrower host ranges than conventional diseases.

STDs are thus fairly well known from both avian and mammalian systems, but what of STDs of insects? Lockhart *et al.* (1996) listed STDs from 29 insect species, but they did not discuss these further. We have expanded this list, and in this review we will discuss the various groups of STDs that infect insects, and consider their distribution and impact on hosts in terms of prevalence (% adults infected) and pathogenicity. We also discuss how they are similar to and how they differ from STDs of vertebrates and the effects they might have on the ecology and evolution of their host species. The available data are too limited in some cases for a thorough testing of our hypotheses, but we hope our discussion will inspire both future empirical and theoretical studies in this area.

II. METHODS

References to STDs infecting insects were found by performing searches of databases such as Web of Science using appropriate key words, by following references in literature already identified and by checking papers that cited ones previously identified. We defined an STD as a parasite or pathogen that is transmitted during mating, either from male to female and/or female to male and included under this definition a couple of cases where transmission occurs from the male to the progeny without directly invading the tissues of the female (e.g. the microsporidian, *Nosema fumiferanae*). In the latter cases, females mating with infected partners suffer an indirect fitness cost via offspring mortality. A number of examples where there is only circumstantial evidence that transmission occurs during mating (e.g. the virus described from the gonads of *Glossina pallidipes* by Jura *et al.*, 1988) and examples where there are no data on pathological effects, such as the phoretic nematode *Diplogaster coprophila* from *Sepsis punctum* (Kiontke, 1996) were also included. We did not include cytoplasmic incompatibility agents such as *Wolbachia* (Hoffman & Turelli, 1997) because these are not transmitted during mating, and have rather different effects on their hosts than the STDs we consider in this review. Table 1 lists the results of the literature search, which produced information on 73 species of STD infecting around 182 species of insect from a wide range of orders. This last figure is as accurate as possible, but not an absolute one because some species are reported as infecting other hosts in the same genus, for example, without specifying the

particular species (i.e. *Labidomera clivicollis*; Abbot & Dill, 2001). Where the names of host were available they are given, otherwise numbers of hosts are listed.

III. INSECT STD DIVERSITY

(1) General patterns

Fig. 1 shows the proportions of reported STDs from the various taxa of pathogens and parasites for insects, with the corresponding data for STDs of mammals from the review by Lockhart *et al.* (1996) also plotted for comparison. It is immediately apparent that whereas the vast majority of STDs of mammals are viruses and bacteria (89%), STDs reported from insects are often fungi or multicellular parasites such as nematodes or mites (62% of agents in Table 1). Why should this be? Part of this pattern is probably due to reporting bias, and in particular it is likely that microparasitic STDs of insects are seriously underreported. However, at least for bacterial agents, a paucity of observations may reflect a paucity of incidences. Although bacteria have been recorded in the testes of male insects, which might at first sight suggest sexual transmission, all of those that have been intensively studied so far are agents of cytoplasmic incompatibility (see Hoffman & Turelli, 1997 for a review). These are exclusively vertically transmitted via egg cytoplasm and are not transmitted to females during sex. The absence of bacterial STDs may be explained by the discovery of a peptide with anti-microbial properties in *Drosophila melanogaster*, termed andropin, manufactured only in testes (Samakovlis *et al.*, 1991) and passed to females during mating (Lung, Kuo & Wolfner, 2001). The protein may be secreted into accessory fluid to guard sperm stored by the females against degradation. A by-product of this would be the prevention of passage of bacteria in the accessory fluid, thereby excluding them from male to female sexual transmission. Fungal pathogens are more common in insects than in vertebrates, so it is not surprising that some of these fungi have evolved the ability to be transmitted between hosts during mating. Nematodes and mites on the other hand are common parasites of both vertebrates and invertebrates, but mostly seem to have failed to utilise the STD niche in vertebrates. As we will discuss later, the reason that there are so many STD mites and nematodes of insects may be that they have evolved from phoretic ancestors that used the genitalia of their hosts as refuges during transport, and this preadaptation to sexual transmission does not seem to be available to nematodes or mites living in or on vertebrate hosts. Alternatively, the difference may be due to the difference in duration in mating in vertebrates and insects, with the longer couplings in insects allowing more opportunity for transmission of ectoparasites.

(2) Viruses

Most of the viruses that are transmitted between insects by mating, such as the Dengue Fever viruses and St Louis Encephalitis Virus, are normally vectored between mammalian hosts by biting insects (mosquitoes and sand-flies). Sexual transmission in these viruses is presumably an adaptation that enables horizontal spread within the vector population, and may be an important factor in maintaining these viruses in their vector populations if mammalian hosts are absent or vertical transmission is inefficient (Tesh, 1981; Shroyer, 1990). It has even been suggested that some of the arboviruses may have evolved from mosquito viruses maintained in mosquito–mosquito cycles by sexual and vertical transmission (Turrell, 1988). Whatever their evolutionary history, it is surprising that the sexual transmission of these diseases between vectors has not been studied more, given their obvious importance in human health terms. A similar case is that of the tomato virus, TYLCV, which is sexually transmitted in its whitefly vector, *Bernisia tabaci* (Ghanim & Czosnek, 2000).

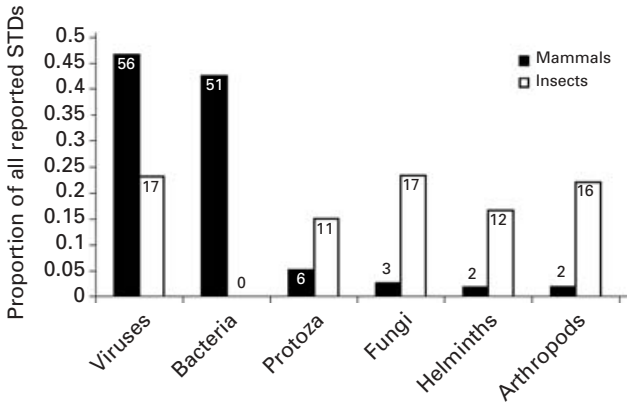

Fig. 1. Proportions of sexually transmitted diseases (STDs) from different taxa from mammals and insects. Mammal data from Lockhart *et al.* (1996). Numbers on or above bars indicate the total numbers for each taxon, totals are mammalian STDs 120, insects 73.

Other sexually transmitted viruses do not have a horizontal transmission component in a mammalian or plant host. These viruses are more virulent than the arboviruses and horizontal transmission occurs through the death of larval forms, which releases infective particles into the environment, which subsequently infect larvae (and to a lesser extent adults) that ingest them. These larvae carry the infections to the adult phase, from which vertical and venereal transmission occurs. In three such cases, transmission during mating has been demonstrated experimentally, namely the baculovirus infecting rhinocerous beetles (*Oryctes rhinocerus*) reported by Zelazny (1976), a parvovirus in *Aedes albopictus* (Barreau, Jousset & Bergon, 1997) and two nuclear polyhedrosis viruses (NPVs) in lepidoptera (Hamm & Young, 1974; Kunimi, 1982).

Sexual transmission has also been demonstrated for the gonad specific virus (GSV) Hz-2V (Hamm, Carpenter & Styer, 1996; Rallis & Burand, 2002) and for the *Drosophila* S virus (DSV) (Ferber *et al.*, 1997). Because of their location within the host gonads a further two viruses are considered likely to be transmitted during mating, but no data exist yet to support this.

Table 1. Reported Sexually transmitted diseases (STDs) of insects. 'Status' refers to whether the STD is definitely transmitted during mating (D) or whether this is inferred from its position in or on the host or from other evidence (P). No entry under the 'pathology' column indicates no data available. 'Transmission' refers to whether the disease is predominantly sexually transmitted (S), both sexually and horizontally transmitted (SH), sexually and vertically transmitted (SV), sexually, horizontally and vertically transmitted (SHV) or whether the important forms of transmission are unknown and sexual transmission is inferred from infection of the gonads (?)

Pathogen taxon	Pathogen species	Host order	Host species	Status	Transmission	Pathology	References
Virus: Flaviviridae	Japanese encephalitis virus	Diptera	*Culex tritaeniorhynchus* (sexual transmission demonstrated) + six other) hosts	D	SHV	No obvious fertility or fecundity effects in females.	Rosen *et al.* (1989)
Virus: Flaviviridae	St Louis encephalitis virus	Diptera	*Aedes taeniorhyncus* (sexual transmisison demonstrated), *Culex quinquefasciatus* + approx. 20 other natural hosts	D	SHV	No obvious fertility or fecundity effects in females.	Shroyer (1990); Nayar *et al.* (1986)
Virus: Flaviviridae	Dengue fever viruses	Diptera	*Aedes albopictus* (sexual transmission demonstrated) + at least three other hosts	D	SHV	Increased larval development time. No obvious fertility or fecundity effects in females.	Rosen (1987); Kuno (1997)
Virus: Togaviridae	Sindbis virus	Diptera	*Aedes australis* (sexual transmission demonstrated) + at least four other hosts	D	SHV		Ovenden & Mahon (1984)
Virus: Bunyaviridae	La Crosse virus	Diptera	*Aedes triseriatus*	D	SHV	Salivary gland damage and reducing feeding effectiveness. No effects on larval development, sex ratio, hatching success, fecundity or adult survival for vertically infected individuals.	Thompson & Beaty (1977); Thompson (1979); Grimstad *et al.* (1980)
Virus: Bunyaviridae	Snowshoe hare virus	Diptera	*Aedes triseriatus*	P	SHV		Schopen *et al.* (1991)
Virus: Bunyaviridae	Tahyna virus	Diptera	*Aedes triseriatus*	P	SHV		Schopen *et al.* (1991)
Virus: Bunyaviridae	Toscana virus	Diptera	*Phlebotomus perniciosus*	P	SHV	No obvious fertility or fecundity effects in females.	Tesh *et al.* (1992)
Virus: Baculoviridae	*Rhabdionvirus oryctes*	Coleoptera	*Oryctes rhinoceros*	D	SHV	Reduced fertility and adult survival. Increased mortality of vertically infected larvae allows horizontal transmission.	Zelazny (1976)
Virus: Picornaviridae	Unnamed	Coleoptera	*Diabrotica virgifera*	P	?	Damage to sperm, decreased hatch rate.	Degrugillier & Degrugillier (1991)
Virus: Geminiviridae	Tomato Yellow Leaf Curl Geminivirus (TYLCV-Is)	Hemiptera	*Bemisia tabaci*	D	SHV	Decreased longevity and fecundity.	Rubenstein & Czosnek (1997); Ghanim & Czosnek (2000)
Virus: Nuclear polyhedrosis virus	HzNPV	Lepidoptera	*Heliothis zea*	D	SHV	Reduced survival of larval offspring. No effect on mating, female fertility or fecundity.	Hamm & Young (1974)
Virus: Nuclear polyhedrosis virus	HcNPV	Lepidoptera	*Hyphantria cunea*	D	SHV	Reduced survival of larval offspring. Little or no effect on mating behaviour. Reduced female fertility.	Kunimi (1982)

Virus: parvovirus	AaPV	Diptera	*Aedes albopictus*	D	SHV	No effect on female fecundity. Reduced female survivorship and offspring survival.	Barreau *et al.* (1997)
Reoviridae	*Drosophila* S virus (DSV)	Diptera	*Drosophila simulans*	D	SV	Developmental malformation.	Ferber *et al.* (1997)
Virus	Gonad Specific Virus (Hz-2V)	Lepidoptera	*Helicoverpa zea* (sexual transmission demonstrated) + *Helicoverpa armigera*	D	SHV	Gonadal hypertrophy and sterility.	Hamm *et al.* (1996); Rallis & Burand (2002)
Virus	Unnamed	Diptera	*Glossina pallidipes*	P	SVH	Eventual female and male sterility and hypertrophy of the salivary glands.	Jura *et al.* (1988)
Protista: Neogregarine	*Ophryocystis elektroscirrha*	Lepidoptera	*Danaus plexippus* + *D. gilippus berenice*	P	SHV	Reduced survival at low humidity, minor effect under less harsh conditions.	Leong *et al.* (1992); Altizer (2001)
Protista: Microsporidia	*Nosema calcarati*	Coleoptera	*Pityogenes calcaratus*	P	SV	Gonads destroyed when males heavily infested.	Purrini & Halpern (1982)
Protista: Microsporidia	*Nosema epilachnae*	Coleoptera	*Epilachna varivestris*	P	SHV	Infection particularly heavy in reproductive tissues of adults. Damage to tissues unknown.	Brooks *et al.* (1980, 1985)
Protista: Microsporidia	*Nosema fumiferanae*	Lepidoptera	*Christneura fumifera*	D	SHV	Reduced female fecundity. Decreased size and survivorship of offspring.	Thomson (1958)
Protista: Microsporidia	*Nosema heliothidis*	Lepidoptera	*Heliothis zea*	P	SVH		Brooks (1968)
Protista: Microsporidia	*Nosema henosepilachna*	Coleoptera	*Henosepilachna elaterii*	P	SHV	Reduced female fecundity and male fertility. No obvious effect on host survival. Tissue damage.	Toguebaye & Marchand (1984)
Protista: Microsporidia	*Nosema kingi*	Diptera	*Drosophila willistoni*, *D. melanogaster* + *D. simulans*	P	SHV	Reduced female fecundity and fertility and survival in both sexes.	Armstrong (1976, 1977); Armstrong & Bass (1989); Armstrong *et al.* (1986); Burnett & King (1962)
Protista: Microsporidia	*Nosema plodiae*	Lepidoptera	*Plodia interpunctella*	D	SHV	Reduced female fecundity and fertility and reduced male fertility. Invades many other tissues. Increased mortality of orally infected larvae.	Kellen & Lindegren (1968, 1971)
Protista: Microsporidia	*Nosema varivestris*	Coleoptera	*Epilachna varivestris*	P	SHV	Invades reproductive tissues of adults. 'Not very virulent'. Damage to tissues unknown.	Brooks *et al.* (1985)
Protista: Microsporidia	*Vairimorpha antheraeae*	Lepidoptera	*Mamestra brassicae*	D	SHV		Yefimenko *et al.* (1990)
Protista: Microsporidia	Unnamed	Orthoptera	*Acrida turrita*	P	?	Little or no effect on sperm development.	Moutairou *et al.* (1993)
Fungi: Laboulbeniales	*Hesperomyces virescens*	Coleoptera	*Adalia bipunctata*, *Chilocorus bipustulatus* + several other coccinellids	P	S	No obvious fertility and mortality effects or effects on O_2 consumption in *C. bipustulatus*.	Welch *et al.* (2001); Applebaum *et al.* (1971)
Fungi: Laboulbeniales	*Canthadomyces platystethi*	Coleoptera	*Platystethus cornutus*	D	S		Weir (1997)

Table 1 (*cont.*)

Pathogen taxon	Pathogen species	Host order	Host species	Status	Transmission	Pathology	References
Fungi: Laboulbeniales	*Laboulbenia odobena*	Coleoptera	*Bembidion picipes*	P	S		Benjamin & Shanor (1952)
Fungi: Laboulbeniales	*Eusynaptomyces benjaminii*	Coleoptera	*Enochrus testaceus*	D	S		Scheloske (1976*a*)
Fungi: Laboulbeniales	*Misgomyces coneglanensis*	Coleoptera	*Laccobius minutus* (sexual transmission demonstrated) + other *Laccobius* spp.	D	S		Scheloske (1976*b*)
Fungi: Laboulbeniales	*Ceratomyces rostratus*	Coleoptera	*Hydrocombus fimbriatus*	P	S		Thaxter (1896, in Lockhart *et al.* 1996)
Fungi: Laboulbeniales	*Filariomyces forficulae*	Dermaptera	*Labidura riparia* (sexual transmission demonstrated) + *Prolabia pulchella* (position highly indicative)	D	SH on *L. r* and S on *P. p*	Reduced survival and possible reduced egg viability in *L. riparia*.	Strandberg & Tucker (1974); Benjamin & Shanor (1952)
Fungi: Laboulbeniales	*Stigmatomyces baeri*	Diptera	*Musca domestica*	D	S	No significant effect on life-span.	Peyritsch (1875 in Whisler 1968) and Benjamin & Shanor (1952)
Fungi: Laboulbeniales	*Stigmatomyces ceratophorus*	Diptera	*Fannia canicularis*	D	S	No significant effects on mortality. Makes holes in cuticle.	Whisler (1968)
Fungi: Laboulbeniales	*Stigmatomyces aciurae*	Diptera	*Anastrepha striata*	P	S		Hedström & Monge-Nájera (1998)
Fungi: Laboulbeniales	*Stigmatomyces verruculosus*	Diptera	*Anastrepha striata*	P	S		Hedström & Monge-Nájera (1998)
Fungi: Entomophthorales	*Massospora cicadina*	Hemiptera	*M. septendecim* (sexual transmission demonstrated), *Magicada cassini*, *M. septendecula*, *M. tredecula*, *M. tredecim*, *M. tredecassini*	D	SH	Damage to the abdomen of adults leads to reduced reproductive success of males and females and eventual death. Hence, minor effects on mortality. Heavily infected individuals remain active and attempt matings, despite losong abdominal segments.	Lloyd *et al.* (1982); Speare (1921); Soper (1963); White & Lloyd (1983); Williams & Simon (1995)
Fungi: Entomophthorales	*Massospora levispora*	Hemiptera	*Okanagana rimosa*	D	SH	Damage to the abdomen of adults leading to infertility (males and females) and eventual death.	Soper (1963); Soper *et al.* (1976)
Fungi: Entomophthorales	*Massospora* sp.	Hemiptera	*Meimuna boniensis*	P	SH	Reduces fertilty and survival. Infected males observed attempting to mate.	Ohbayashi *et al.* (1999)
Fungi: Entomophthorales	*Massospora pahariae*	Hemiptera	*Paharia casyapae*	P	SH		Soper (1981)
Fungi: Entomophthorales	*Massospora cicadettae*	Hemiptera	*Cicadetta murrayiensis*, *Cicadetta puer* + *Cicadetta* sp.	P	SH		Soper (1981)
Fungi: Entomophthorales	*Entomophthora muscae*	Diptera	*Musca domestica*	D	SH	Lethal.	Møller (1993)

Nematoda: Acugutturidae	*Noctuidema guyanense* + at least 2 other spp.	Lepidoptera	*Spodoptera frugiperda* (sexual transmission demonstrated) + > 30 other lepidopteran species	D	S	Reduced female fertility. Infected females shorter lived.	Simmons & Rogers (1990*a*, *b*, 1994)
Nematoda: Aphelenchoididae	*Bursaphelenchus* sp.	Coleoptera	*Urophorus humeralis* (sexual transmission demonstrated) + four other spp	D	SH	No obvious effects on survival or fertility. Possible that large infestations may block the genitalia.	Giblin (1985)
Nematoda: Diplogasteridae	*Diplogaster coprophila*	Diptera	*Sepsis punctum* (sexual transmission demonstrated) and others	D	SH	No obvious effect on fertility or fecundity.	Kiontke (1996)
Nematoda: Rhabditidae	*Rhabditis stammeri*	Coleoptera	*Nicrophorus vespilloides*	D	SH	No obvious effects on survival or fertility. Possible that large infestations may block the genitalia.	Richter (1993)
Nematoda: Rhabditidae	*Oryctonema genitalis*	Coleoptera	*Oryctes monocerus* + five other spp?	P	S	No effects on mortality or fecundity. Possible that large infestation may block the genitalia.	Poinar (1970)
Nematoda: Rhabditidae	*Oryctonema pentodonis*	Coleoptera	*Pentodon punctatus*	P	S		Poinar & Triggiani (1979)
Nematoda: Rhabditidae	*Rhabditis adenobia*	Coleoptera	*Oryctes monocerus* (sexual transmission demonstrated) + 14 other dynastid spp.	D	S	No obvious effects on fertility or survival. In females occurs in colleterial glands.	Poinar (1971)
Nematoda: Aphelenchoididae	*Huntaphelenchoides* sp.	Hymenoptera	*Anthophora bomboides*	P	SH	No obvious deleterious effects.	Giblin *et al.* (1981)
Nematoda: Mehdinematidae	*Mehdinema alii*	Orthoptera	*Gryllodes sigillatus* (sexual transmission demonstrated) + *G. domesticus*	D	S	The adult stage is parasitic. Almost without exception adults are only found in males. Low pathogenicity.	Farooqui (1967); Luong *et al.* (2000)
Nematoda: Allantonematidae	*Parasitilenchus coccinellae*	Coleoptera	*Propylea quatuordecimpunctata*, *Synharmonia conglobata*, *Adalia bipunctata*, *Adonia variegata*, *Menochilus sexmaculatus* + *Ileis indica*	P	S	Reduced fecundity and fertility in females.	Hodek & Honek (1996) and references therein
Arthropoda: Acarina	*Coccipolipus hippodamiae*	Coleoptera	*A. bipunctata* (sexual transmission demonstrated), *A. decempunctata* (sexual transmission demonstrated), *Synharmonia conglobata* (sexual transmission demonstrated), *Calvia quatuordecemguttata*, *Hippodamia convergens*, *Exochomus fulvimanus* + *E. concavus*	D	S	Fecundity and egg viability reduced. No effect on mating success. No effect on survival under laboratory conditions, but overwintering mortality increased, especially in males.	Husband (1981); Hurst *et al.* (1995); Webberley & Hurst (2002); Webberley *et al.* (2002); Webberley *et al.* (2003)
Arthropoda: Acarina	*Coccipolipus epilachnae*	Coleoptera	*Epilachna varivestris*	D	S	Possible reductions in fertility and survival but results are mixed.	Schroder (1982); Cantwell *et al.* (1985); Hochmuth *et al.* (1987)

Table 1 (*cont.*)

Pathogen taxon	Pathogen species	Host order	Host species	Status	Transmission	Pathology	References
Arthropoda: Acarina	*Coccipolipus* sp. probably *macfarlenei*	Coleoptera	*Coccinella septempunctata*, *C. transversalis*, *Coleophora bissellata*, *Menochilus sexmaculatus*, *Harmonia octomaculata*	D	S	Heavy infestation leads to reduced reproductive potential.	Rhamhalinghan (1989)
Arthropoda: Acarina	*Chrysomelobia labidomerae*	Coleoptera	*Labidomera clivicollis* (sexual transmission demonstrated) + other *Labidomera* spp. + *Leptinotarsa* spp.	D	S	Increased female mortality, increased mortality of males and females when starved.	Abbot & Dill (2001)
Arthropoda: Acarina	*Dorsipes dorsipes*	Coleoptera	*Carabus granulatus*	P	S		Regenfuss (1968)
Arthropoda: Acarina	*Eutarsopolipus lagaenaeformis*	Coleoptera	*Scarites buparis*	D	S		Regenfuss (1968)
Arthropoda: Acarina	*Eutarsopolipus acanthomus*	Coleoptera	*Broscus cephalotes*	D	S		Regenfuss (1968)
Arthropoda: Acarina	*Eutarsopolipus pterostichi*	Coleoptera	*Pterostichus melanarius*	D	S		Regenfuss (1968)
Arthropoda: Acarina	*Eutarsopolipus vernalis*	Coleoptera	*Pterostichus nigrita* + *P. anthracinus*	P	S		Regenfuss (1968)
Arthropoda: Acarina	*Ovacarus clivinae*	Coleoptera	*Clivina impressifrons*	P	S	Reduced fecundity.	Stannard & Vaishampayan (1971)
Arthropoda: Acarina	*Ovacarus peelei*	Coleoptera	*Pasimachus elongates*	P	S		Husband (1974)
Arthropoda: Acarina	*Parasitellus fucorum*	Hymenoptera	*Bombus* sp.	D	SH		
Arthropoda: Acarina	*Podapolipoides grassi*	Orthoptera	*Chortoicetes terminifera* + *Locusta migratoria* (sexual transmission demonstrated in both)	D	S on *C. t*, SH on *L. m*	Reduced activity levels in Australian *C. terminifera*.	Volkonsky (1946); Gauchat (1972)
Arthropoda: Acarina	*Podapolipoides patangae*	Orthoptera	*Patanga succincta* + *Oedaleus* sp.	P	S		Lo (1990)
Arthropoda: Acarina	*Podapolipus lahillei*	Orthoptera	*Eyprepocnemis smaragdipes*	P	S		Naudo (1967)
Arthropoda: Acarina	*Unionicola ypsilophora*	Diptera	*Paratrichocladius rufiventris*	D	SH	Mites feed on the host, but increase mating success of host.	McLachlan (1999)
Arthropoda: Acarina	*Kennethiella trisetosa*	Hymenoptera	*Ancistrocerus antilope*	D	SH	Mite protonymphs feed on haemolymph of host pupae.	Cowan (1984)

(3) Protists

There are only five protistans infecting insects for which sexual transmission has been demonstrated experimentally and six cases where infection of the testes suggests sexual transmission. This is surprising, since protistans cause many diseases of insects, with the Microsporidia being an obvious large taxon of specialist arthropod parasites. It is likely that this low number is more a consequence of a lack of attention rather than a real rarity, and with further investigation many more examples may come to light. Alternatively, it could be that this pattern reflects biological reality, and protistan STDs of insects might truly be rare, but it is not currently possible to assess which of these two options is correct.

(4) Fungi

The majority of fungi that have been reported as being STDs come from the order Laboulbeniales. These fungi are all ectoparasites of arthropods, and usually exhibit a high degree of specificity in the parts of the insect that they will grow on (Weir & Beakes, 1995). This high degree of specificity has led to a number of them being identified as being transmitted during mating because they are found on the parts of the male and female insects that come into contact with each other during mating. That this corresponds with transmission during mating has been confirmed experimentally several times, originally by Peyritsch (1875, cited in Whisler, 1968; arguably the first description of an STD of an insect), and several times since (Whisler, 1968; Strandberg & Tucker, 1974). Both of these latter studies also found that the fungus can also be transmitted by other contact between conspecifics. It is often thought that the Laboulbeniales do little harm to their hosts (Weir & Beakes, 1995), but Strandberg & Tucker (1974) found a significant increase in mortality in earwigs (*Labidura riparia*) infected with *Filariomyces forficulae*.

A second group of sexually transmitted fungus is the *Massospora* species. Their periodical cicada hosts continue to fly and mate even when heavily infected and abdominal segments have broken away to expose the mass of infectious conidia left in their place (Soper, Delyzer & Smith, 1976; White & Lloyd, 1983). A final and rather unusual example of an STD fungus is that of *Entomophthora muscae* infecting *Musca domestica*. Møller (1993) described how this fungus causes swelling of the abdomen of freshly killed cadavers of its host, making them resemble gravid females. Male flies attempt to mate with these cadavers and in doing so become infected.

(5) Nematodes

As mentioned above, although nematodes have frequently been reported from the genitalia of insects, there seems to be a continuum from nematodes that are essentially phoretic, spending a part of their life-cycle in the genitals of a host insect to achieve dispersal, to nematodes that are 'true' STDs. The latter, which complete their entire life-cycle in the host, include *Oryctonema genitalis* and *Rhabditis adenobia*, described from the genitalia and associated structures of tropical dynastid beetles (Poinar, 1970, 1971) and *Mehdinema alii* from the cricket *Gryllodes sigillatus* (Luong *et al.*, 2000).

The best-known nematode STD does not live in the genital region of its host. This is the ectoparasitic *Noctuidema guyanese*, first reported from noctuid moths in French Guiana and now known from 41 host species from five families of the Lepidoptera in tropical and subtropical North and South America (Simmons & Rogers, 1996), and from four noctuid species in Fiji (Rogers, Marti & Clayton, 1997). *Noctuidonema guyanese* has been studied extensively as a potential control agent for *Spodoptera frugiperda* and a considerable amount is known about its transmission, reproduction, ecophysiology and pathology (reviewed in Simmons & Rogers, 1996). Two more ectoparasitic species of *Noctuidema* have since been described (*N. daptria* and *N. dibolia*; Simmons & Rogers, 1996). It is likely that many other moth species in the Neotropics and elsewhere are also infected with *Noctuidema*, and also that further examination of the genus *Noctuidema* will reveal more ectoparasitic nematode species (Simmons & Rogers, 1996). A related species, *Acugutturus parasiticus* has also been reported from the cockroach, *Periplaneta americana* in the West Indies (Hunt, 1980), implying that similar sexually transmitted nematodes may be found in taxa other than the Lepidoptera.

(6) Mites

Ectoparasitic mites from the family Podapolipidae have been reported to be transmitted during mating a number of times, with the best known species being *Coccipolipus hippodamiae*, which infects the two-spot ladybird beetle *Adalia bipunctata* in Central and Eastern Europe (Hurst *et al.*, 1995). It is likely that the majority of mites from this family are transmitted during mating to some extent, and as there are currently 203 described species, from five orders of hosts, with perhaps 10 times that number yet to be described (R. Husband, personal communication) this is undoubtedly a most important taxon of insect STDs.

Within the family, and even within genera, there is a range of reliance on sexual transmission. Some, such as *Podapolipoides grassi* on North African *Locusta migratoria*, are transmitted both sexually and socially (Volkonsky, 1946), whereas others such as *Coccipolipus hippodamiae* are almost totally sexually transmitted (Hurst *et al.*, 1995; K. M. Webberley, unpublished data). The most extreme examples of adaptation to sexual transmission in this taxon are *Ovacarus peelei* (Husband, 1974) and *Ovacarus clivinae* (Stannard & Vaishampayan, 1971), both of which are endoparasites that live in the genitalia of male and female carabid beetles from the Midwestern USA.

There are a few other examples of sexually transmitted mites known, the first being *Unionicola ypsilophora*, of the family Hydracharinidae. This species is essentially a phoretic mite, using its chironomid midge hosts (*Paratrichocladius rufiventris*) to disperse to new areas of fresh water and mussel hosts. Only female midges return to water after mating to oviposit, and mites transfer from male to female midges during mating (McLachlan, 1999). A similar example of phoretic mites transferring from males to females during

mating is found in bumblebees (Huck, Schwarz & Schmid-Hempel, 1998). Specialised phoretic deutonymphs of *Parasitellus fucorum* will transfer to female bumblebees from males in order to ensure that they are transported to a new nest. A final, fascinating, sexually transmitted mite is *Kennethiella trisetosa*, which infests the eumenid wasp *Ancistrocerus antilope* (Cowan, 1984); this particular case is discussed in more detail in section IX.

IV. HOST DIVERSITY AND RANGE

(1) Distribution of STDs across host taxa

Fig. 2 shows the distribution of insect hosts of both purely sexually transmitted diseases and STDs with additional forms of transmission compared to the distribution of all insects across the different orders. Clearly, the incidence of STD cases in the different orders does not simply reflect how speciose the orders are (pure STDs: $\chi^2 = 15.2$, d.f. $= 5$, $P < 0.01$, all STDs: $\chi^2 = 16.2$, d.f. $= 5$, $P < 0.01$), nor does it appear to be related simply to the amount of research effort directed at each order (research effort quantified by the number of hits reported from a key word search on the name of the order on the ISI 'Web of Science' online database, correlation with number of reported STDs: $r = 0.49$, d.f. $= 6$, $P = 0.262$), and several features stand out. First, if one considers all STDs and not just those that are purely sexually transmitted, there appears to be an excess of cases affecting the Diptera. However, these are mainly arboviruses, i.e. diseases of medical importance to man, indicating a possible study bias in this particular case. A more interesting observation is the comparatively low number of reported STDs from the Hymenoptera, which has also been noted by Schmid-Hempel (1998) when considering the social Hymenoptera. There are no examples of 'pure' STDs to be found in Hymenoptera and only three examples of STDs with additional forms of transmission. Given the analysis of research effort detailed above, and considering that this is a large and intensively studied order of insects, it seems unlikely that this is simply an artefact of sampling effort, and so this lack of STDs may be a real phenomenon. It may be related to the high level of reproductive skew found in the social insects, which make up a large subset of this order. Here, many individuals never mate and hence spread of an STD through a population is precluded. Indeed, when hosts live together in close contact in a colony, parasites using other forms of transmission are likely to be favoured. Similarly, the low number of pure STDs found amongst the Hemiptera may reflect the unusual reproductive systems found amongst some homopterans. Specifically, it is likely that homopteran species with regular parthenogenesis are unable to maintain purely sexually transmitted parasites. In the one homopteran case reported, that of tomato leaf curl geminivirus in the whitefly *Bemisia tabaci* (Ghanim & Czosnek, 2000), the majority of transmission between hosts is horizontal via infection of host plants, and sexual transmission may only play a minor role in disease dynamics.

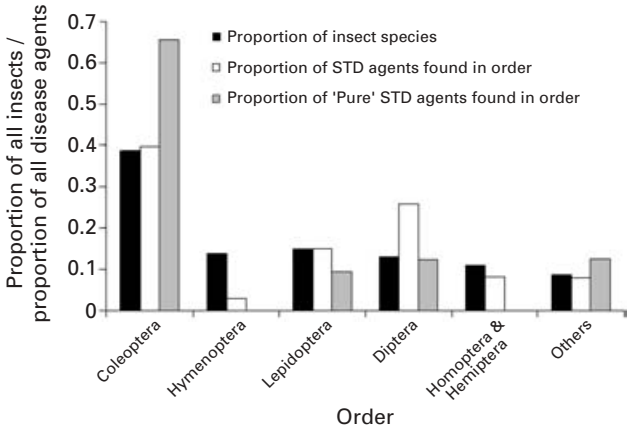

Fig. 2. The distribution of sexually transmitted disease (STD) agents across host orders and the distribution of insect species across host orders. The percentages of insects in the different orders was calculated from data in Wilson (1992), and the proportions of all STDs and 'pure' STDs (i.e. STDs with no additional forms of transmission) are from the data in Table 1. It is clear that the distribution of STDs across the Insecta does not simply depend on how speciose each order is.

In contrast to the Hymenoptera and Hemiptera, the Coleoptera are particularly well represented and this may be a consequence of two factors. First, many Coleoptera exhibit a promiscuous mating system based on scramble competition, where most individuals enjoy some or even high mating success (Ridley, 1990). Such behaviour is clearly conducive to STD maintenance (see below). Second, most Coleoptera from temperate latitudes seem to overwinter as adults (see chapter 7 of Beck, 1980, for example), whereas many other taxa are more likely to overwinter as eggs, larvae or pupae. This habit is likely to be strongly associated, at least in temperate climates, with whether the host species has overlapping adult generations, and sexual transmission is only feasible as a primary transmission route when this is the case, with at least a few adults from one generation surviving to mate with and therefore infect adults of the next generation. Those listed in Table 1 as having transmission of type 'S' have, or probably have, no alternative transmission route that would allow the STD to be transmitted between non-overlapping generations of adults (i.e. vertical transmission). The majority of these 'pure' STDs (21 of 32) are reported from Coleoptera. Of those STDs that do not have alternative means of transmission between generations and do not infect Coleoptera, many are reported from tropical and subtropical species, which are likely to be continuously breeding, such as *Anastrepha striata* from Costa Rica and the various Lepidoptera infected by *Noctuidonema guyanese* from tropical and subtropical America. Of the remaining few temperate species, the host populations of most are known to have overlapping, long-lived adult generations, such as the cricket *Gryllodes sigillatus* (California; Marlene Zuk, personal communication).

The extent of overlap of host generations can also be important in determining STD distribution. Welch *et al.*

(2001) describe how the prevalence of the fungus *Hesperomyces virescens* on *Adalia bipunctata* increases with closeness to the centre of London, and they suggest that this may be because more adults are able to survive the winter in the city centre, meaning that there is a greater proportion of adults infected with the fungus in the spring.

If the sexually transmitted parasite or pathogen has vertical transmission between parents and offspring, the need for overlapping adult generations is circumvented.

For example, *Helicoverpa zea* undergoes a period of diapause as a pupa (Pullen, Meola & Lopez, 1992), but its gonad specific virus, Hz-2V is transmitted vertically (in the egg) to offspring as well as horizontally during mating (Hamm *et al.*, 1996). In systems where there are multiple hosts of the same STD, periodic reinfection from alternative hosts may also maintain the STD on hosts that do not show reproductive continuity between generations (Webberley *et al.*, 2003). Similarly, migration can regularly reintroduce STDs to populations that have unsuitable phenology, as is the case with populations of *Spodoptera frugiperda* in the Southern USA. These are reinfected with the nematode *Noctuidonema guyanese* each spring by infected moths which have migrated from continuously breeding populations in Central America (Simmons *et al.*, 1991; Simmons & Rogers, 1996).

The mating system of insects also affects the distribution of STDs. Promiscuous insects are more likely to harbour STDs than those that mate infrequently. As mentioned above this may explain the rarity of STDs in the Hymenoptera and Homoptera. The prevalence of STDs is also likely to be higher in promiscuous insects. Strong evidence for this relationship comes from study of the prevalence of the sexually transmitted mite *Coccipolipus hippodamiae* on three European ladybird hosts. Here, prevalence across host samples correlates positively with mating rates, with the highest prevalence occuring in the most promiscuous species, *A. bipunctata* (Webberley *et al.*, 2003). Differences in mating rate can also explain variation in STD prevalence between the two sexes when the sex ratio deviates from 50:50. Such variation is recorded in six of the pure STD cases in Table 2a, and in two of these cases, *Eusynaptomyces benjaminii* on *Enochrus testaceus* Scheloske (1976*a*) and *Coccipolipus hippodamiae* on *Adalia bipunctata* (K. M. Webberley, personal observation), the cause is assumed to be that the less common sex has a higher mating rate and hence is more likely to be infected. Differing degrees of skew in mating success between the two sexes, differing susceptibilities to infection or a different age structure of male and female populations are additional possible explanations for the patterns observed in other systems.

One final factor that is likely to be important in the distribution of STDs in insects is the lifespan of the host species. Insects with short lifespans or those that spend a very short period of their lives as adults, are unlikely to maintain purely sexually transmitted diseases. For example, an insect as ephemeral as a mayfly is unlikely to become infected with an STD, incubate the disease and then pass it onto another mating partner in the short time it is an adult. Short host lifespan becomes especially limiting to STD maintenance when the disease agents are multicellular parasites rather than fast-replicating microparasites. This leads us to predict that STD occurrence may be correlated with adult lifespan, as in longer-lived insects the latent period of any sexually transmitted disease will be short relative to the lifespan of the host, and consequently, the infectious period much longer.

As we have seen, STD distribution varies with host taxon and probably also with geography, with STDs possibly being more common in continuously breeding tropical insects, but just how common are STDs within the insects? The fact that we found 73 examples suggests that STDs are not rare. Further, four studies where data on the incidence of STDs in a group of insects have been gathered indicate that STDs are widespread, particularly in the Coleoptera and Lepidoptera. The nematode *Noctuidonema guyanense* infects 15% of noctuid moths in French Guiana (Rogers *et al.*, 1990). Eleven species of dynastid beetles from the genus *Oryctes* occur in Madagascar and Bedford (1968) found at least one species of sexually transmitted nematode in the genitalia of six of the eleven. Examination of Eastern European coccinellid beetle species for the presence of just one type of STD, sexually transmitted podapolipid mites, revealed that 17% ($N=18$) were infected. The parasite was *Coccipolipus hippodamiae* in all cases (Webberley *et al.*, 2003). Similarly, Regenfuss (1968) examined 78 species of German carabid beetles and found 50% to be infected with podapolipid mites from two genera, *Eutarsopolipus* and *Dorsipes*, which he viewed as sexually transmitted species. The podapolipid mites are a tantalizing group for anyone considering the incidence of STDs. They are very widespread, having been found on four insect orders across several continents. In most cases only the very basic biology has been studied, but where transmission mode has been assessed there is commonly a sexual component.

(2) Host range

Specialisation of a parasite to one host is an expected side-effect of specialisation to sexual transmission, because this probably presents few opportunities for transmission to members of different host species. Where the ancestral parasite resides on many different host species, sexual transmission will produce strong population division, leading to decreased gene flow between parasites on the different host species. The parasite may be divided into virtually separate populations and sympatric speciation may then follow. This mechanism of speciation may not require selection for host specialization *per se*; the barrier to gene flow may come purely from selection for sexual transmission. Notably, Lockhart *et al.* (1996) found that STDs of mammals had narrower host ranges than conventional infectious diseases.

Fig. 3 shows the number of reported STDs and hosts for each parasite taxon. In all parasite taxa there are more reported species of host than species of STD, demonstrating that insect STDs are often reported from more than one host species. This contrasts with the situation for vertebrate host taxa: Sheldon (1993) found 15 STDs from five bird species and Lockhart *et al.* (1996) listed 123 STD species

Table 2. The peak prevalence recorded in the literature for four types of insect STDs, (a) 'pure' STDs, i.e., those for which the majority of transmission is during host copulation and there is no requirement for any other form of transmission for the maintenance of the parasite in the host population, (b) sexually and horizontally transmitted diseases, (c) sexually and vertically transmitted diseases and (d) sexually, horizontally and vertically transmitted diseases. If only one prevalence value was found, that is given. Where male and female prevalence differs, overall peak prevalence is used if given. If the sex ratio is not given, a sex ratio of 1:1 is assumed for calculation of overall peak prevalence. Only cases where there are data on prevalence are included, see Table 1 for additional cases. References given are for the sources of the data on peak prevalence and prevalence variation, where relevant

Pathogen species	Host species	Site	Peak prevalence	Notes	Reference
(a) Pure STDs					
i) Fungi					
Hesperomyces virescens	*Adalia bipunctata*	London, UK	50 %	Seasonal variation.	Welch *et al.* (2001)
Hesperomyces virescens	*Chilocorus bipustulatus*	Israel	> 75 %	Seasonal variation.	Applebaum *et al.* (1971)
Laboulbenia odobena	*Bembidion picipes*	Illinois, USA	35 %	Variation with host sex.	Benjamin & Shanor (1952)
Eusynaptomyces benjaminii	*Enochrus testaceus*	Germany	28.6 % on males, 13.3 % on females. 18.8 % overall	Variation with host sex.	Scheloske (1976*a*)
Misgomyces coneglanensis	*Laccobius minutus*	Germany	12.5 %		Scheloske (1976*b*)
Stigmatomyces baeri	*Musca domestica*		'Common'		Whisler (1968)
Stigmatomyces ceratophorus	*Fannia canicularis*	San Francisco, USA		Heaviest in late summer.	Whisler (1968)
Stigmatomyces aciurae	*Anastrepha striata*		Approx. 30 %		Hedström & Monge-Nájera (1998)
ii) Nematodes					
Noctuidema guyanense	*Spodoptera frugiperda*		Peak 100 %	Average in Grenada 77 %. Variation with season, latitude and host sex (Silvain & Remillet (1993).	Simmons *et al.* (1991)
Oryctonema genitalis	*Oryctes monoceros* and five other spp.	Ivory Coast, West Africa	Unclear	92 % of older females infected. 0 % of virgin females infected.	Poinar (1970)
Oryctonema pentodonis	*Pentodon punctatus*	Italy	92 %		Poinar & Triggiani (1979)
Rhabditis adenobia	*Oryctes monocerus*	Malaysia and Ivory Coast	70 % females, 50 % males (estimate 60 % overall)	Variation with host sex.	Poinar (1971)
Mehdinema alii	*Gryllodes sigillatus*	California, USA	36.3 % males, 20 % females (estimate 28 % overall)	Variation with host sex.	Luong *et al.* (2000)
Parasitilenchus coccinellae	*Propylea quatuordecimpunctata*	Southern France	> 70 %		Hodek & Honek (1996)
Parasitilenchus coccinellae	*Synharmonia conglobata*	Southern France	20 %		Hodek & Honek (1996)
Parasitilenchus coccinellae	*Adalia bipunctata*	Southern France	< 10 %		Hodek & Honek (1996)
Parasitilenchus coccinellae	*Adonia variegata*		< 10 %		Hodek & Honek (1996)
iii) Mites					
Coccipolipus hippodamiae	*A. bipunctata*	Poland	> 80 %	Variation with host sex.	Webberley *et al.* (2003)
Coccipolipus hippodamiae	*A. decempunctata*	Poland	31 %		Webberley *et al.* (2003)
Coccipolipus hippodamaie	*S. conglobata*	Poland	6 %		Webberley *et al.* (2003)
Chrysomelobia labidomerae	*Labidomera clivicollis*	Mid-west USA	> 90 %	Seasonal variation.	Abbot & Dill (2001)
Dorsipes dorsipes	*Carabus granulatus*	Germany	3 %		Regenfuss (1968)
Eutarsopolipus pterostichi	*Pterostichus melanarius*	Germany	> 60 %	Seasonal variation.	Regenfuss (1968)
Eutarsopolipus vernalis	*Pterostichus nigrita*	Germany	> 50 %	Seasonal variation.	Regenfuss (1968)
Ovacarus clivinae	*Clivina impressifrons*	Mid-west USA	25 %		Stannard & Vaishampayan (1971)
Podapolipoides grassi	*Chortoicetes terminifera*	Australia		'Distribution widespread and rapid'.	Gauchat (1972)
Podapolipoides patangae	*Patanga succincta* + *Oedaleus* sp.	Taiwan	60–100 %.	Seasonal variation Found at lower prevalence on *Oedaleus* sp.	Lo (1990)

(b) Sexually transmitted diseases with additional horizontal transmission					
i) Fungi					
Filariomyces forficulae	*Labidura riparia*	Florida, USA	37 %	Seasonal variation.	Strandberg & Tucker (1974)
Massospora cicadina	*Magicada septendecim*	Kansas, USA	50–90 % males infected with sexually transmitted stage late in season (estimate 50 % overall)	Variation with host sex, else where, 65 % on males, 1.2 % on females. Prevalence of horizontally transmitted stage much lower (Lloyd *et al.*, 1982).	Speare (1921)
Massospora levispora	*Okanagana rimosa*	Ontario, Canada	18 % (sexually transmitted stage)		Soper (1963)
Massospora sp.	*Meimuna boniensis*	Japan	34.4 % (sexually transmitted stage)		Ohbayashi *et al.* (1999)
ii) Nematodes					
Diplogaster coprophila	*Sepsis punctum*	Berlin, Germany	40 %		Kiontke (1996)
Rhabditis stammeri	*Nicrophorus vespilloides*	Germany	5–28 %		Völk (1950)
Huntaphelenchoides sp.	*Anthophora bomboides*	California, USA	14 % females, 7 % males (estimate 10.5 % overall)		Giblin *et al.* (1981)
iii) Mites					
Unionicola ypsilophora	*Paratrichocladius rufiventris*	UK	16 %	Variation with sex and varies with behaviour.	McLachlan (1999)
(c) Sexually and vertically transmitted diseases					
Protista					
Nosema calcarati	*Pityogenes calcaratus*	Israel	50 %		Purrini & Halpern (1982)
(d) Sexually transmitted diseases with additional horizontal and vertical transmission					
i) Viruses – arboviruses					
Dengue fever viruses	*Aedes aegypti* and *A. albopictus*	India	5.9 %	Seasonal and geographic variation. Lower in Singapore (0.051 %) and Thailand (0.059 %).	Kuno (1997)
Japanese encephalitis virus	*Culex tritaeniorhynchus*		2 %	Seasonal variation.	Acha & Szyfres (1980)
St Louis encephalitis virus	*Aedes taeniorhyncus*		0.1 %		Nayar *et al.* (1986)
– other viruses					
Rhabdionvirus oryctes	*Oryctes rhinocerous*	W. Samoa	35 %	Variation with host sex and season.	Zelazny (1973)
Unnamed	*Glossina pallidipes*	Kenya	15.6 %		Odindo (1982)
ii) Protista					
Ophryocystis elektroscirrha	*Danaus plexippus*	Florida	> 70 %	Variation depends on degree of migration.	Altizer *et al.* (2000)
Nosema epilachnae	*Epilachna varivestris*	North & South Carolina, USA	28 %	Seasonal variation. Highest late in season. Lower in overwintering adults.	Brooks *et al.* (1980)
Nosema fumiferanae	*Choristoneura fumifera*	Ontario, Canada	40 %		Thomson (1958)
Nosema heliothidis	*Heliothis zea*			High prevalence in laboratory cultures.	Brooks (1968)
Nosema henosepilachna	*Henosepilachna elaterii*	Dakar, Senegal	90 % males, 72 % (estimate 81 % overall)	Variable prevalence.	Toguebaye & Marchand (1984)
Nosema plodiae	*Plodia interpunctella*	California, USA	7 %		Kellen & Lindegren (1968)
Nosema varivestris	*Epilachna varivestris*	North & South Carolina, USA	4 %	Seasonal variation. Epidemics of 85 % prevalence in laboratory populations.	Brooks *et al.* (1980)

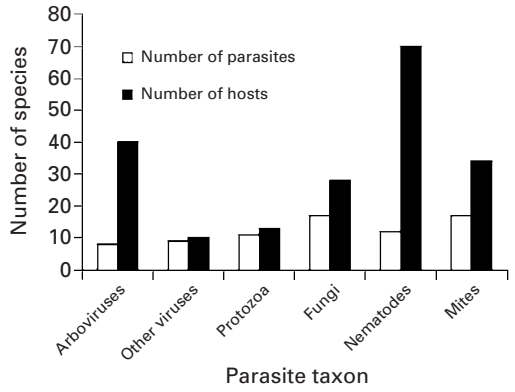

Fig. 3. Numbers of reported sexually transmitted disease (STD) species from different parasite taxa (open bars) and the number of host species infected by parasites from each taxon (filled bars).

from 23 species of mammalian host. The majority of the vertebrate species are domesticated species that are intensively studied, no doubt accounting for some of this pattern, and we should also note that the data presented by these authors mentioned above was not intended to give exact details of host range, and so some hosts may have been missed out. Nonetheless, there are no reported vertebrate STDs with host ranges approaching those of, for example, *Noctuidonema guyanese* (>30 host species) or *Coccipolipus hippodamiae* (seven host species). Two factors may be important here. First, it is likely that interspecific matings tend to occur more often between natural populations of insect species than in domesticated vertebrate animals (Arnold, 1997), removing the barrier to gene flow and allowing STDs more readily to colonise new insect hosts. Second, the differences between species may be smaller in the more speciose insects, where lots of sibling species occur, than in the higher taxa (Price, 1980). Hence, once transfer to a new host has occurred, the parasite may be more likely to colonise the new 'environment' successfully.

A closer examination of the data reveals that there is a taxonomic element to host range: microbial STDs such as viruses (other than the arboviruses) or protists tend only to have been reported from a single host species, but the sexually transmitted nematodes, and to a lesser extent the mites, are likely to have been reported from more than one host species (Fig. 3). However, this difference between taxa is not quite statistically significant (Kruskal–Wallis test, $H=10.36$, d.f. $=5$, $P=0.066$).

Whether these parasites enjoy a wider or narrower host range than similar conventionally transmitted parasites of insects remains a hard question to answer with current knowledge. There has been limited synthesis of the host range of parasites, although Price (1980) gives data for parasitoids: studies of 514 Ichneumonidae found that 53% had only one host, whilst 60% of 214 Braconidae were specialised to one host. These figures are similar to the 58% of parasites ($N=81$) in Table 1 with only one known host. The figure is even lower when only pure STDs are considered; only 43.5% ($N=39$) have only one known host. On first consideration, these findings appear to contrast strongly with Lockhart *et al.* (1996)'s finding of smaller host ranges for STDs compared to other diseases in mammalian hosts. The high rate of hybrid matings in insects may be important here, but caution is needed in interpreting the data as they stand. Synthesis of data on other insect parasite groups is needed and furthermore, it is not known whether some of the parasites in Table 1 with wide host ranges are really species complexes (as is the case with, for example, *Entomophthera muscae*; Keller, Kalsbreek & Eilenberg, 1999). We consider this question to be a potentially fruitful avenue for future research, and suggest that parasite transfer experiments and investigation of the genetic differentiation between parasites on different host species would be a useful way to investigate this problem.

All the parasites and pathogens we consider, despite their range of reliance on sexual transmission, will have the same effect: to make sex more risky for their hosts. Thus, all will probably have important effects on the evolution of the mating systems of their hosts. Clearly, differences in transmission modes are reflected in the parasites' distributions; additional transmission by other routes widens the range of suitable hosts. As we shall describe below, virulence patterns are also likely to differ between those that are purely sexually transmitted and those that are also transmitted in other ways.

V. PATTERNS OF TRANSMISSION

We found a range of parasites and pathogens from those that are purely sexually transmitted through those that are mostly sexually transmitted, but also use one or more additional forms of transmission, to ones that are predominately transmitted by other routes and are only rarely transmitted during mating (see Table 1). Those that are 'pure STDs', i.e. those for which all, or almost all, transmission is sexual, constitute 45% (32 of 71, two excluded due to lack of data) of the total number of agents. The degree of dependence on sexual transmission and the combination of transmission modes displayed appears to depend on whether the agent is an endo- or an ectoparasite. The majority of the ectoparasites listed (32 of 46), all of which are fungi, nematodes or arthropods, are predominately sexually transmitted. Of the recorded fungal parasites, 10 were purely sexually transmitted and seven were both sexually and horizontally transmitted. Similar patterns were observed for nematodes (eight purely or predominantly sexually transmitted, four with additional horizontal transmission) and mites (14 sexually transmitted and three with sexual and non-sexual horizontal transmission). Those ectoparasitic STDs that are transmitted by non-sexual routes tend to be horizontally, rather than vertically transmitted. For example, *Massospora* fungi on cicadas are sexually transmitted at high rates between adults during the mating season (Soper *et al.*, 1976), but horizontal transmission of resting spores via the environment to last instar nymphs also occurs and allows transmission between generations separated by up to 17 years. Other ectoparasites

exhibit both sexual and other forms of horizontal transmission, even though host adults are present all year round, e.g. the mite *Podapolipoides grassi* in North Africa, which is found on both the adults and hoppers of *Locusta migratoria* (Volkonsky, 1946).

By contrast, we were unable to find any examples of purely sexually transmitted endoparasites (where transmission has been fully investigated), and here the dominant additional form of transmission is vertical transmission. The protista, microsporidia, arboviruses and the majority of other viruses listed in Table 1 all show sexual and vertical transmission and, perhaps surprisingly, most (nine out of 10 protists and 15 out of 16 viruses for which there are good data on transmission) also show some additional horizontal transmission.

VI. PREVALENCE OF STDs

A notable and general characteristic uncovered by our research was the high prevalence reached by STD epidemics in insect populations. There are several examples of purely sexually transmitted parasites periodically reaching in excess of 70% prevalence. These regular epidemics seem to be set up by the inherent time delays in recruitment to the adult generation found, at least, in temperate species. In addition, there is a trend for prevalence to be higher in systems with purely sexual transmission than in those with additional forms of transmission, although this is not statistically significant (Mann–Whitney U test: $z=1.69$, $P=0.09$). We collated reports giving data on prevalence for insect STDs (Table 2). Prevalences were recorded in a variety of ways by different authors, and over varying periods of time, but some patterns are discernable. Mean peak prevalence of purely sexually transmitted diseases in natural insect populations was found to be 46% ($N=23$, Table 2a). Those that are both sexually and horizontally transmitted were found to have a mean peak of 29% ($N=8$, Table 2b) and those that exhibit both additional horizontal and vertical transmission had a mean peak prevalence of 26% ($N=11$, Table 2d). The one sexually and vertically, but not horizontally, transmitted pathogen for which there are prevalence data infected up to 50% of the adult population (Table 2c). Further data are needed to assess whether the association is real and truly due to the degree of dependence on sexual transmission or to other confounding factors, such as agent taxon (see above). One case that strongly supports the hypothesis that sexual transmission is key in producing high prevalence, comes from the *Massospora* fungi infecting cicadas. Infection with the horizontally transmitted resting spores that develop into conidia reaches less than 10% of the newly emerged adults, whereas later in the season sexual transmission leads to 65% of adult males becoming infected (Lloyd, White & Stanton, 1982). High prevalences may also be a feature of some STDs of vertebrates. Most of the available data on vertebrate STDs comes from domesticated animals, and in these cases prevalence may be influenced by, for example, the breeding regime imposed by the keepers. One study of wild koala bears, however, reported prevalences of *Chlamydia pecorum* of up to 73% and *C. pneumoniae* at up to 23% (Jackson *et al.*, 1999).

VII. PATHOLOGY FROM INSECT STDs

Table 1 includes information on the pathology caused by the insect STDs listed. Of the purely sexually transmitted diseases that have been tested for pathology, the majority have some deleterious effects on the host, the most common of which are reduced fertility, either a reduction in the number of eggs laid or a reduction in the probability of those eggs hatching, and an increase in host mortality. There are 12 cases where only a negative effect on fertility has been recorded, four cases where only a negative effect on longevity has been recorded and 10 cases where there is a negative effect on both. Fertility can be reduced effectively to zero, meaning that the STD sterilizes the host. This is seen in, for example, *Coccipolipus hippodamiae* infecting *Adalia bipunctata*, which causes egg viability to drop to less than 20% when the female has been infected for more than ten days and complete female infertility approximately 17 days after infection (Hurst *et al.*, 1995). A similar effect was reported for *Spodoptera frugiperda* females infected with *Noctuidema guyanese*, although a second set of experiments showed less of a reduction in egg viability (Simmons & Rogers, 1994). This reduction in host fertility was also found in mammalian STDs by Lockhart *et al.* (1996) who demonstrated that STDs of mammals cause reductions in fertility more often than do conventionally transmitted pathogens.

Reductions in host fertility associated with infection are known as 'parasitic castration', and may be an adaptation on the part of the parasite to divert resources to the parasite without reducing host longevity (Poulin, 1998*b*; Hurd, 1998, 2001). In the case of STDs it is important that the host does not become obviously diseased or morbid, as it will then be less likely to obtain a mating and transmit the STD (Hurst *et al.*, 1995; Knell, 1999). By sterilising its host, the STD may free up resources that it can use to reproduce without diminishing the host's abilities to attract mates or reducing longevity, thereby increasing the likelihood of transmission. This assumes, of course, that the parasite is able to sequester resources originally destined for host reproduction without causing a reduction in mating frequency. Alternatively, rather than being host manipulation by the STD, reductions in fertility may be a host response to infection whereby resources which would be used in reproduction are released for investment in defences against the parasite (Hurd, 1998, 2001). To differentiate between these two hypotheses we can compare the patterns of pathology observed in hosts of purely sexually transmitted diseases and in the adult hosts of those diseases in Table 1 that have additional forms of transmission. We find that negative effects on fertility are more common for pure STDs [occurs in 73% (eight of 11) of cases where effects on fertility were tested for] than for STDs with additional forms of transmission, where 56% (15 of 27) of cases report effects on fertility. This difference is not, however, statistically

significant (Fisher exact test: $P=0.47$) making it difficult to assess the effect of transmission mode on virulence. Effects on survival are less common for pure STDs (44 %, or four of nine cases where mortality was examined) than for the other diseases (67 %, or 10 of 15), but again this difference is not statistically significant (Fisher exact test: $P=0.40$). It is also worth noting that in two of the three pure STD cases with mortality effects, the effects show up only when the host is very stressed, i.e., when it is overwintering or starved (Abbot & Dill, 2001; Webberley & Hurst, 2002). In the third case, only a 'possible effect' is described (Schroder, 1982; Cantwell, Cantelo & Cantwell, 1985; Hochmuth *et al.*, 1987). A final possibility is that reductions in host fertility may arise simply because of parasite replication in the reproductive organs, allowing easy transmission during host mating. In the cases of parasites such as *C. hippodamiae* and *N. guyanense*, however, which are both ectoparasites that neither live nor feed in the host's reproductive organs, this seems unlikely.

The evolution of virulence is a more complicated affair for parasites that use more than one transmission strategy. Vertical transmission is usually assumed to lead to selection for reduced virulence, but theoretical studies have revealed that this is not always the case (Lipsitch, Siller & Nowak, 1996), and if a parasite is transmitted both during mating and also vertically then the optimal virulence can depend on the relative importance of sexual and vertical transmission. STDs that induce host sterility can enhance their transmission rate if sterilised hosts make more mating attempts than others (Lockhart *et al.*, 1996), but this will reduce the opportunities for vertical transmission available to the parasite. Reducing sterility may be selected against mostly in hosts with high birth rates, and hence more opportunities for vertical transmission (Altizer & Augustine, 1997). In these cases parasites are expected to produce mainly virulence effects other than reduced host fecundity, fertility and mating rate.

The virulence effects on adult hosts of the arboviruses are low and effects on host fertility are negligible. For example, the harmful effects of La Crosse virus on its mosquito hosts appear to be limited to salivary gland pathology (Lam & Marshall, 1968; Patrician & DeFoliart, 1985). These decrease mosquito biting effectiveness (Grimstad, Ross & Craig, 1980), making feeding more risky and less effective, but increasing the number of mammalian hosts bitten and hence parasite dissemination. The vertically transmitted protists, microsporidians and baculoviruses such as OrBV and NPVs tend to be more virulent. They may eventually harm their adult hosts, either reducing fertility or longevity, but they have much more deleterious effects on their progeny: killing them during larval stages to produce horizontal transmission (Zelazny, 1972; Hamm & Young, 1974). It appears that in these cases horizontal rather than vertical transmission plays a more important role in disease dissemination.

Finally, the high prevalence observed in insect STD systems may have implications for the evolution of parasite virulence. Multiple infections of single hosts are likely to occur in such systems and this may lead to selection for increased virulence in competing strains (van Baalen & Sabelis, 1995). This may explain why mortality effects, although limited, are found in some of the pure STD cases in which prevalence can reach extremely high levels.

VIII. EVOLUTION OF INSECT STDs

The continuum of parasites with varying reliance on sexual transmission provides insight on the evolution of specialisation to sexual transmission. Three routes are immediately apparent.

The first is evolution from a phoretic ancestor. The most likely candidates for this route are found in the nematodes. Non-parasitic nematodes are frequently phoretic, attaching themselves to larger organisms to disperse, and since the phoretic lifestyle gives some important pre-adaptations to parasitism it seems that many parasitic nematodes have evolved from phoretic ancestors (Anderson, 1984; Athias-Binche & Morand, 1993; Poulin, 1998*b*). Sexually transmitted nematodes seem to be a good example of this. Nematodes have frequently been reported from the genitalia of insects, and there seems to be a continuum from nematodes that are essentially phoretic, using the insects as transport between patches of food, to nematodes that are 'true' STDs, with their entire life cycle being carried out in or on the body of the host and transmission between hosts being achieved largely or entirely during mating. Phoretic nematodes that produce larvae that live in the genitalia of their hosts have been reported from a wide range of insects including soil-nesting bees (*Anthophora bomboides* and others; Giblin, Kaya & Brooks, 1981), nitidulid beetles (*Urophorus humeralis* and others; Giblin, 1985), burying beetles (*Nicrophorus vespilloides*; Richter, 1993), dung beetles (*Geotrupes* spp.; Kuhne, 1996; *Onthophagus* spp.; R. J. Knell, unpublished observations) and sepsid flies (*Sepsis punctum* and others; Kiontke, 1996). The degree to which phoresy is obligatory for these nematodes varies. The various species of *Diplogaster* which live in the brood balls of dung beetles undergo between six and 16 generations in an individual brood ball before the beetle larva pupates and second-stage juveniles develop into dauer larvae and enter the genital region of the newly hatched beetle (Kuhne, 1994, 1996). By contrast, the dauer larvae of *Diplogaster coprophila* are unable to complete their development unless they have spent some time in the reproductive system of a sepsid fly host, and transmission of these larvae during mating has been observed (Kiontke, 1996). The evolutionary step from this to the nematode completing its entire life cycle in the host is a short one, and as we have seen there are a number of examples of nematodes that do exactly this.

Amongst mite STDs, it appears that the podapolipid family also evolved from a phoretic ancestor. The Podapolipidae are believed to have evolved from tarsonemids, the majority of which are now phytophagous (Eickwort, 1975). The evolutionary trajectory may have been via a parasitic ancestor that transferred whenever hosts made close contact. Amongst the extant species some, such as *Podapolipoides grassi*, which is found on both adults and hoppers of its host, North African *Locusta migratoria*, employ a mixed strategy of both sexual and social transmission (Volkonsky, 1946). They

may represent intermediate points on the evolutionary trajectories towards purely sexually transmitted parasites. Undoubtedly, the best examples of adaptation to sexual transmission in the podapolipid mites come from one of the most derived genera, *Ovacarus*, which are endoparasites in the genitalia of hosts (Stannard & Vaishampayan, 1971).

Secondly, a few STDs appear to have evolved from gut parasites. HzNPV virus is transmitted from males to females through faecal contamination of the tip of the abdomen (Hamm & Young, 1974). Direct tissue infection of adults does not occur, but infected females suffer an indirect fitness cost when their eggs become contaminated with virus during oviposition. The hatching larvae eat the eggshells, are infected and die as a result. A similar process occurs in a microsporidian system, *Nosema fumiferanae* on *Choristoneura fumifera* (Thomson, 1958), which is perhaps unsurprising as the majority of non-sexually transmitted microsporidian parasites of insects enter through the gut. Other sexually transmitted Microsporidia invade the reproductive organs of both sexes, e.g. *Nosema plodiae* (Kellen & Lindegren, 1968).

Finally, as mentioned above many of the sexually transmitted micro-parasites of insects are also vertically transmitted. It is likely that the site of infection for vertical transmission, the reproductive organs, predisposes the parasite to sexual transmission (or *vice versa*). Furthermore, a theoretical study of host-pathogen dynamics that considered pathogens with both frequency-dependent horizontal transmission (thought to be typical for STDs) and vertical transmission found that pathogen strains capable of both types of transmission were usually able to invade and exclude pathogen strains that were only capable of horizontal transmission (Altizer & Augustine, 1997). It is not surprising, then, that many STDs also show vertical transmission, and we note that all of the 'pure' STDs we have described are multicellular ectoparasites, and may be constrained from vertical transmission by their size and location on the host.

IX. STDs AND INSECT BEHAVIOUR

(1) STDs and mate choice

The most obvious aspect of host behaviour that might be affected by the presence of an STD is mating behaviour. Most attention has focussed on the idea that STDs could drive sexual selection via female choice for males that are unlikely to infect them (Clayton, 1991; Able, 1996; Loehle, 1997). Knell (1999), however, argued that avoidance of STDs was unlikely to be important in female choice, since there would be strong selection on STDs to become cryptic in such circumstances.

Two empirical studies have tested these ideas. In both *Adalia bipunctata* infected with the mite *Coccipolipus hippodamiae* (Webberley *et al.*, 2002) and in *Labidomera clivicollis* infected with *Chrysomelobia labidomera* (Abbot & Dill, 2001) females appear to be completely unable to discriminate between males with and without the sexually transmitted mite, so mating occurs essentially at random with regards to a male's infection status. This is especially surprising in *A. bipunctata*, since the mite is highly pathogenic and very common, meaning that the fitness benefits to a female of being able to detect and avoid it would be considerable. These results are consistent with Knell's (1999) prediction, that STDs will be cryptic to potential mates and so the presence of an STD will be unlikely to select for mate choice.

Graves & Duvall (1995) discussed an alternative effect of STDs on mate choice, that STDs could cause selection against choosy females because popular males will be more likely to infect them during mating. Rather than providing a selective advantage for mate choice, therefore, STDs could be acting as a cost of choosing a mate *per se*. Such selection against choosy females has recently been modelled by Boots & Knell (2002) and by Kokko *et al.* (2002). Both studies found that an STD was unlikely to eradicate female choice, but that polymorphisms could arise, with the evolutionarily stable strategy often being a mixed one as a result of frequency-dependent selection.

(2) STDs and promiscuity

A second way in which the presence of an STD could affect the evolution of host behaviour is by causing selection against hosts that mate frequently (Hamilton, 1990; Sheldon, 1993; Loehle, 1995; Lockhart *et al.*, 1996). Thrall *et al.* (1997) and Thrall, Antonovics & Dobson (2000) modelled the effects of STDs on promiscuity, as did Boots & Knell (2002). These studies found that STDs can select for reduced promiscuity, but if there is a fitness benefit to promiscuity then once again the outcome can be polymorphism, with some individuals being promiscuous and others less so (Boots & Knell, 2002).

To date, we only have data on the relationship between promiscuity and STD infection from one system (*Coccipolipus hippodamiae* on coccinellids; Webberley *et al.*, 2003), which suggests a positive correlation between mating rate and STD prevalence. However, it is likely that any evolutionary effects of STD presence are obscured by the fact that the ecology of promiscuous species is better suited to STD spread. Selection experiments may be more useful for examining the evolution of reduced promiscuity in response to STD presence, and insect systems are ideal for such a method.

(3) STD manipulation of host behaviour

STDs could influence host behaviour if the parasites themselves manipulate the behaviour of the host. Behavioural changes associated with parasitic infection are well known, and at least some of these appear to be adaptations on the part of the parasite to increase transmission (Moore, 1993, 2001; Poulin, 1994*a*, *b*, 1998*a*, 2000). Four recent studies of insect STDs are relevant here. McLachlan (1999) showed that male midges (*Paratrichocladius rufiventris*) infected with the mite *Unionicola ypsilophora* were more likely to be in mating pairs than uninfected males. As discussed earlier, the mites rely on female midges to return them to water to complete

their life cycle. If they find themselves on a male midge, therefore, they are effectively dead unless their host mates with a female, allowing the mites to transfer to the female midge, giving a clear selective advantage to mites that somehow increase the probability of their male hosts mating. Whether this qualifies as a true adaptation by the parasite is not clear yet because not enough is known of the mechanism by which the increase in infected males in mating pairs occurs.

Raina *et al.* (2000) found that Hz-2V infected female corn earworm moths *Helicoverpa zea* produce two to three times more sex pheromone than uninfected female moths, possibly enhancing their ability to attract male moths, although they also reported that these animals vigourously resisted mating. Abbot & Dill (2001) found that male *Labidomera clivicollis* beetles infected with the mite *Chrysomelobia labidomera* were more likely to displace other males from mating pairs, which again could be interpreted as being adaptive manipulation of the host by the parasite to increase transmission. Webberley *et al.* (2002), by contrast, found that infection of *Adalia bipunctata* with *Coccipolipus hippodamiae* did not have any effect on the mating behaviour of the host. Taken together, these studies suggest that STDs are capable of manipulating their host's behaviour to increase transmission. More detailed studies are needed, however, to determine whether these behavioural changes are real adaptations by the parasite or whether they are simply by-products of the pathology the parasite causes (Poulin, 2000; Moore, 2001).

(4) STDs and sexual conflict

When considering the selective forces that are acting in a host-STD relationship, we must bear in mind that the evolutionary interests of males and females are often not the same, and there is frequent conflict over, for example, control of fertilisation and oviposition rate. We know that male insects can attempt to influence female behaviour by, for example, producing large numbers of anucleate sperm which seem to act as a 'filler' and reduce the female's remating rate (Cook & Wedell, 1999) or by producing proteins in their ejaculate which increase the female's oviposition rate at the expense of her longevity (Chapman *et al.*, 1995). When viewed in this light, we can see that there are some circumstances under which transmitting an STD to a female with whom he mates may actually increase a male's fitness. If, for example, contracting an STD reduced a female's remating rate without affecting her fecundity over the short term then a male who gave the female this STD might fertilise more eggs than one who did not, and in this case transmission of the STD might be regarded as an adaptation to avoid sperm competition. Similarly, if females react to infection by increasing their oviposition rate then a male may benefit from transmitting an STD to a female because the number of eggs that his sperm fertilise will be increased. There are two examples of increased oviposition following STD infection in the literature. Female *Spodoptera frugiperda* infected with the sterilising nematode *Noctuidonema guyanense* produce more eggs over a short period following infection (Simmons & Rogers, 1994). Similarly, infection of the earwig *Labidura riparia* with the fungus *Filariomyces forficulae* leads to more eggs being laid at the first oviposition after mating (Strandberg & Tucker, 1974). It should be noted here that increasing the oviposition rate following infection may also be adaptive behaviour on the female's part, especially when the pathogen in question is highly pathogenic (Adamo, 1999) and teasing apart the evolutionary causes and implications of this behaviour may well prove difficult.

A more unusual system also provides circumstantial evidence for the use of STDs by males to manipulate their mating partners. Cowan (1984) discussed the life cycle of the mite *Kennethiella trisetosa*, which parasitises the Eumenid wasp *Ancistrocerus antilope* in North America. Female wasps lay single eggs in holes in wood, provision them with paralysed caterpillars and seal the nest. When they oviposit, they may also leave nymphs of the mite in the nest. These immature mites feed on the caterpillars, and when the wasp larva becomes a quiescent prepupa the now adult mites feed on its haemolymph. Eggs are laid on the pupal wasp and the freshly hatched mite nymphs feed on haemolymph from the pupa. When the adult wasp emerges the mite nymphs attach themselves to the wasp's propodeum and are carried away with it. Emerging female wasps are rarely infested, however, because they kill most of the mites in their nests when they are still larvae. Males show no sign of such behaviour, and often emerge with heavy infestations of mites. When a male mates with a female, roughly half of the mites on his body are transferred to the genital chambers of the female. The obvious question here is why do the females, but not males, kill the mites in their nests? There are two possible answers to this. First, males may be somehow constrained and unable to kill mites. This seems unlikely given that females of the same species can do so very efficiently. Second, the selective consequences of mite infestation may be different for male and female wasps, with the presence of mites reducing female fitness but enhancing male fitness, and this could arise if the presence of mites in the female's genitalia reduces her likelihood of remating. As Cowan (1984) points out, the selective advantage of this to the male would be boosted by the fact that due to the haplodiploid sex-determination system of the Hymenoptera, few of the offspring of a particular male will be infested with mites that are derived from him. Male offspring, which are infested on emergence and presumably pay a higher price in terms of pathology from the mites, only carry genes from their mothers, and female offspring, with a set of chromosomes from their fathers, are able to destroy mites while they are still larvae.

These examples are all rather extreme and similar systems are likely to be rare, but other interactions between STDs and sexual conflict in the host may occur more widely. As mentioned above, presence of an STD may select for a reduction in promiscuity, but other selection pressures on mating rate may differ in the two sexes, so that the evolutionary outcome is disadvantageous for one sex (Thrall *et al.*, 1997). Transmission efficiency, susceptibility, virulence effects, and hence STD impact, frequently differ for the sexes and it is likely that this further exacerbates conflict over mating rate.

X. OTHER HOST ADAPTATIONS

In addition to behavioural changes in response to STD presence, other possible host adaptations include immunological and chemical resistance and also life-history changes. The presence of a sterilising or lethal parasite in a population is expected to select for an increase in early reproductive effort, to compensate for the future loss of reproductive success (Minchella, 1985; Shykoff & Kaltz, 1997). This is possible in many female insects because they often store large amounts of sperm and hence could increase reproduction without increasing exposure to sexually transmitted parasites. Alternatively, STD infection might trigger increased reproductive effort by individual hosts (as already discussed in Sections VII and IX). In either case, reduced host longevity is likely to result as a trade-off.

XI. STDs AND INSECT POPULATION ECOLOGY

As discussed in the Introduction, theoretical studies have demonstrated that STDs have the potential to have important effects on the population dynamics of their hosts, and in a few cases it has been suggested that this might be happening in animal species. In the case of STDs of insects, however, there are currently no available data to evaluate how often this might be the case. Some systems do seem likely candidates for population regulation by STDs, however. Both *Coccipolipus hippodamiae* and *Chrysomelobia labidomerae* have been found to reach high prevalences in their host populations (Table 2; Abbot & Dill, 2001; Webberley *et al.*, 2002, 2003), and *C. hippodamiae* certainly has important pathological effects including sterilization of females, so the presence of the mite may be having important effects on recruitment rates in the host population.

Some other STDs are both common and pathological; the nematode *Parasitilenchus coccinellae* reaches over 70% prevalence in the ladybird, *Propylea quatuordecimpunctata* and infection leads to reduced female fecundity and fertility (Hodek & Honek, 1996). Similarly, prevalence of the nematode *Noctuidonema guyanese* is often around 35% and can be as high as 90% in the fall armyworm, *Spodoptera frugiperda* (Simmons & Rogers, 1990*a*) and the parasite reduces the fertility of the host's eggs and increases its mortality rate (Simmons & Rogers, 1994). Since high temperatures and humidity seem to favour the growth of this parasite (Simmons & Rogers, 1990*b*) it is possible that infection with *N. guyanese* may interact with climate to affect the distribution of its hosts. In addition, the parasite is reported from a number of species, with varying infection rates (Simmons & Rogers, 1996). This may have an effect on the outcome of intraspecific competition if some species suffer from a greater reduction in their intrinsic rate of population increase from parasitism than others.

One very important aspect of host-STD interactions arises from the way in which transmission rate depends on host density. STDs are assumed to be transmitted in a 'frequency-dependent' manner, whereby the rate of transmission is equal to the proportion of infectious hosts in the population, whereas conventional directly transmitted diseases are thought to show 'density-dependent' transmission, the transmission rate being proportional to the densities of infectious and susceptible hosts (Getz & Pickering, 1983; Smith & Dobson, 1992; Thrall *et al.*, 1993; McCallum, Barlow & Hone, 2001). This means that the STD does not have a host threshold density for transmission, below which it will become extinct (Getz & Pickering, 1983). STDs are therefore able to persist in low-density populations. This leads to the prediction that STDs might be present in rare species of insect which do not support other specialist pathogens or parasites, although the data currently available do not allow us to test this.

It is worth noting that host adaptations in response to STD presence, such as reduced promiscuity or host life-history changes, will affect the STD population dynamics. A decrease in mating rate or reductions in overlap of generations produced by shortened host life-span may limit STD spread. Theoretical models of the adaptive dynamics of insect-STD systems would be useful in elucidating likely outcomes.

XII. STDs AS BIOLOGICAL CONTROL AGENTS

Most interest in insect pathogens arises from their possible use as pest control agents, and some STDs of insects have been investigated for this reason, although to date there has really only been one attempt to use a sexually transmitted parasite for biological control. Introductions of the baculovirus ORBV, into disease-free islands has lowered the population density of the pest species, *Oryctes rhinoceros* to 10–20% of pre-release levels (Zelazny, Lolong & Crawford, 1990). Use has been suggested in at least one other case: Jura & Davies-Cole (1992) proposed the release of male Tsetse flies, *Glossina morsitans* sterilised through sexually transmitted viral infection, as a control measure of this serious pest. More recent work has found that this is unlikely to be effective because of high mortality experienced by males as a consequence of infection (Sang *et al.*, 1997).

From a consideration of the pathology that STDs cause it seems unlikely that they will provide a useful source of 'biological insecticides' because they tend not to produce the kind of rapid mortality that is needed for such products. However, some STDs, such as the two referred to above, do show promise as agents for longer term control. As mentioned in the introduction, theoretical studies have shown that STDs have the potential to affect host population dynamics (Getz & Pickering, 1983; Thrall *et al.*, 1993, 1997) although the circumstances under which useful control of a pest population might be achieved by an STD have not been considered by theoreticians.

One aspect of STD-host population dynamics that might be important in some circumstances is the ability of STDs to persist in low-density host populations when other pathogens might become extinct. STDs may therefore be useful in situations where a pest insect is normally only present at low densities but occasionally outbreaks; if the STD could keep the population density of the pest suppressed even when rare

this might reduce the frequency and/or the severity of the outbreaks.

XIII. CONCLUSIONS

This review represents the first serious collection of reports of STDs from insects. There are a surprising number of such diseases known, and it is likely that there are a great many more waiting to be discovered. Most of the STDs described so far are conspicuous and come from well-studied systems, and it is probable that the microbial STDs of insects are especially poorly described. This makes generalising about insect STDs a hazardous undertaking. Nonetheless, there are some conclusions that it is possible to be reasonably sure of.

(1) Insects seem to suffer from many more multicellular STDs than do mammals, with nematodes and mites both featuring heavily in the list of insect STDs presented here, but being very rare in the list of mammalian STDs presented by Lockhart *et al.* (1996). These nematode and mite STDs of insects probably evolved from phoretic ancestors, and the lack of phoretic use of mammals by similar animals may explain this difference.

(2) We found a continuum of parasites with varying reliance on sexual transmission. Sexually transmitted ectoparasites were either purely sexually transmitted or had additional routes of horizontal transmission. All the sexually transmitted endoparasites were also vertically transmitted and also tended to show horizontal transmission. The degree to which parasites use sexual transmission is reflected in distribution and virulence patterns.

(3) Whether or not the host has overlapping adult generations is an important factor controlling the distribution and evolution of purely sexually transmitted diseases of insects. It is likely that many more STDs remain to be described from tropical systems for this reason. Other important aspects of host life-history in determining STD incidence and prevalence are mating rate and probably adult lifespan.

(4) Insect STDs are often highly pathogenic. While this may manifest itself as increased host mortality, a common effect is reduced fecundity or sterility of the host. This is a similar pattern to that reported for mammalian STDs by Lockhart *et al.* (1996), and suggests that similar factors are important in the evolution of virulence in both mammalian and insect STDs.

(5) Insect STDs can be very common in their host populations, and prevalences of $>90\,\%$ have been reported in a number of cases.

(6) This combination of high pathogenicity and high prevalence means that STDs have the potential to be important in the evolution and ecology of their hosts. In particular, the implications for the evolution of host mating systems are potentially very important.

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