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Sexually Transmitted Disease in Birds: Occurrence and Evolutionary Significance

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Sexually transmitted disease in birds: occurrence and evolutionary significance

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SUMMARY

Sexually transmitted diseases (STDs) span two current areas of sexual selection theory, namely the roles of multiple mating in determining individual reproductive success, and of parasites in mate choice, yet have been relatively neglected in the ecological literature. I reviewed the occurrence of STDs in populations of commercially kept birds and found widespread evidence for the existence of pathogenic STDs in such populations. STDs may have important consequences for the evolution of behaviour, reproductive physiology and some secondary sexual characteristics. Where STDs are costly they are hypothesized to affect the evolution of mating systems, and, via selection for hostility in the female reproductive tract, to explain high levels of sperm mortality after insemination. The potential for coevolutionary cycling is large, as some STDs may coevolve with female and male reproductive physiology, which may themselves coevolve. Although little information currently exists concerning the occurrence of STDs in wild birds, techniques for their identification are well established. This study raises a number of testable predictions about the consequences of STDs for avian reproductive biology, and I suggest that STDs should be considered as a potentially powerful factor in future studies of mate choice and sperm competition.

1. INTRODUCTION

The past decade has seen a large amount of interest among evolutionary biologists in two aspects of sexual selection. First, there has been considerable debate concerning the role of parasites in the evolution of secondary sexual characters (Hamilton & Zuk 1982; Møller 1990; Loye & Zuk 1991). Variation in parasite resistance by hosts is seen as a possible source of continuous genetic variation that intersexual selection ('mate choice') can act upon. As parasites and hosts coevolve, optimal resistance genotypes of hosts are not fixed over time but keep changing as parasites quickly overcome new host defenses. Secondary sexual characteristics are interpreted as 'revealing handicaps' (Harvey & Bradbury 1991) which have evolved to give information about the ability of an individual to resist parasites. By choosing individuals with such handicaps, females can select those males most resistant to the current parasite fauna. Although in theoretical work, 'parasite' has had a broad definition (Price 1980; Anderson & May 1982; Hamilton & Zuk 1982), in most empirical work, the attention of biologists has, for practical reasons, usually been restricted to relatively large endo- and ectoparasites (Møller 1990; Loye & Zuk 1991).

Second, there has been increased interest in the extent to which sperm competition and multiple mating can affect variance in reproductive success among individuals (Smith 1984; Birkhead & Møller 1992). Improved biochemical and molecular techniques (Jeffreys *et al.* 1985; Burke 1989) have revealed that multiple mating by both males and females probably occurs in the majority of bird species (Westneat *et al.* 1990; Birkhead & Møller 1992), and may be responsible for a large part of the variance in individual reproductive success (Gibbs *et al.* 1990; Westneat 1990; Birkhead & Møller 1992).

At the interface between the two areas lie sexually transmitted diseases (STDs), where I define an STD as any pathogen that is transmitted during the act of copulation. (Clearly, this definition includes ectoparasites which might be transferred during copulation, but for most of the arguments advanced here the effects of ectoparasites will be similar to other forms of STD). STDs have received little attention in the ecological literature to date, with, for instance, little mention in Loye & Zuk's recent (1991) review of bird-parasite interactions or Birkhead & Møller's (1992) review of sperm competition in birds, although Hamilton (1990) raises some thought-provoking ideas concerning STDs and mating systems, and Freeland (1976) considers that they may have been important in the evolution of primate sociality and sexual behaviour. However, the existence of STDs suggests a number of unique implications for the evolution of mating behaviour, reproductive physiology and the evolution of secondary sexual characteristics not considered by more general parasite models. In addition, avoidance of STDs has been identified as one of the direct benefits that might drive female choice in the face of the apparent 'lek paradox' (Reynolds & Gross 1990).

Unfortunately the prevalence and ecology of STDs has been largely ignored, with perhaps the exception

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of cloacally transmitted endo- or ectoparasites (Hamilton 1990). However, modern practices in poultry farming, where the sperm from a single male may (artificially) inseminate hundreds of females, or large numbers of breeding individuals may be kept in close proximity, mean that STDs are potentially of considerable economic importance (e.g. Stipkovits *et al.* 1986). As a result, the prevalence of STDs in commercial populations of chickens *Gallus*, turkeys *Meleagris* and geese *Anser* is better documented, and may give some indication of likely patterns of STDs in wild birds. Although housing conditions may facilitate the occurrence and spread of parasites among commercial stock (Calnek *et al.* 1991), in wild bird populations, infectious diseases can cause mortality at levels comparable to that usually attributed to predators or scarce resources (Anderson & May 1979: Hudson & Dobson 1991), so the potential for STDs to be a potent force in evolution should not be discounted. In humans, the world incidence of STDs is probably only surpassed by diseases such as malaria and roundworm infections (Bailey 1979).

2. STDs IN BIRDS

To assess the importance and occurrence of STDs in birds, I searched the biological abstracting services under related key-words and subject areas. Literature references to STDs in birds (table 1) came mainly from Calnek *et al.* (1991) and from *Biological Abstracts* 1970–1991 and *The Zoological Record* 1970–1990.

Evidence demonstrating that the main means of pathogen transmission is sexual is rather scant in most cases (table 1), but strong supporting evidence indicating that sexual transmission is possible, and could be important (e.g. the presence of pathogens in semen, transmission via artificial insemination) exists for other diseases. Accordingly, I have divided 'STDs' into classes based upon the strength of evidence that they are sexually transmitted (table 1). The weakest category of evidence includes macroparasites such as nematodes and trematodes isolated from the cloaca; whether or not these could be transmitted during copulation is open to question. In addition to the parasite species detailed in part 4 of table 1, a large number of other bacterial species have been isolated from the cloaca (e.g. Cooper *et al.* 1980) but many of these may be non-pathogenic.

The pathogenicity of those diseases identified as being transmitted sexually varies considerably. Goose venereal disease can cause up to 10% mortality in affected flocks, together with reduced weight, fertility and egg production in other flock members (Stipkovits *et al.* 1986; Marius-Jestin *et al.* 1987). Lymphoid leukosis virus is somewhat intermediate in its pathogenicity (Spencer *et al.* 1980), but is known to cause delayed sexual maturity and decreased egg weight, production and fertility. At the other end of the scale, an outbreak of a venereally transmitted pox had no observed effect on egg-production and mortality (Metz *et al.* 1985). Thus, at least some STDs are likely to fulfil the pathogenicity criteria identified by Hamilton & Zuk (1982) as likely to play a role in the evolution of secondary sexual characteristics.

There have been few controlled studies of the transmission dynamics of STDs in birds; the little that is known results largely from anecdotal observations such as those of Marjankova *et al.* (1978) who noted that within a week of first detecting goose venereal disease in a large flock ($n = 700$), 94% of the ganders

Table 1. *Summary of literature references to pathogens either known to be sexually transmitted or with potential to be*

(Species code: (host species) 1 = turkey; 2 = chicken; 3 = domestic goose; 4 = domestic duck *Anas* sp.; 5 = Smith's longspur *Calcarius pictus*.)

	species code	Reference
1. Unquestionably STD		
Mycoplasma meleagridis	1	Mohamed & Bohl (1967)
Mycoplasmosis iowae	2	Calnek *et al.* (1991)
Mycoplasmosis cloacale (goose venereal disease)	3	Stipkovits *et al.* (1986)
lymphoid leukosis virus	2	Crittenden (1981); Spencer *et al.* (1981)
2. Agent isolated from semen		
arizonosis	2	Perek *et al.* (1969)
Ureaplasma spp.	1	Stipkovits *et al.* (1978)
reticuloendotheliosis	1	McDougall *et al.* (1980)
Mycoplasma gallisepticum	2	Perek *et al.* (1969)
Herpes virus	1	Thurston *et al.* (1975)
3. Agent transmitted via AI		
Pox	1	Metz *et al.* (1985)
4. Isolated from cloaca		
campylobacteriosis	2	Shanker *et al.* (1988)
infectious bronchitis	2	Calnek *et al.* (1991)
nematode: *Capillaria anatis*	4	Calnek *et al.* (1991)
trematode spp.	5	J. V. Briskie (pers. comm. 1991)
avian paramyxovirus-2	2	Goodman & Hanson (1988)
Salmonella gallinarum	2	Jordan (1956)

had become infected. Mohamed & Bohl (1967) investigated the rate at which hens became infected with mycoplasmosis by (artificially) inseminating a test group from the same pool of infected semen. Within a week of receiving a single insemination, all 33 of their test hens were positive for mycoplasmosis.

A number of the pathogens which appear in table 1 are more typically gut parasites (Perek *et al.* 1969), but here the physiology of the avian urogenital system, with the cloaca serving a common function for gamete transfer and excretion is such that it may increase the number of pathogens which could be sexually transmitted compared with mammals. Thus, the possession of a cloaca, as distinct from separate anal and urogenital openings, may in fact predispose birds to be exposed to STDs in comparison with mammals (for example). If this is the case then the definition of STD would expand to include parasites that could also be transmitted via other routes. Presumably, any gut pathogen could become incorporated into an ejaculate, particularly those the size of bacteria or viruses, but this might also apply to the eggs of parasitic worms.

The evolutionary implications of STDs can be divided into two classes: (i) those involving direct selection on a particular trait; and (ii) those involving indirect selection via coevolutionary cycling. The distinction between the two is similar to that discussed by Clayton (1991), with the first class relating to the transmission-avoidance model of parasite-mediated sexual selection (PMSS), and the second relating to 'good genes' models of PMSS. Although I shall be specifically discussing birds here, many of my conclusions are equally applicable to other taxonomic groups.

3. DIRECT SELECTION

(*a*) *Effects on behaviour and mating systems*

If STDs inflict a cost in terms of survival or reproductive success, individuals are expected to be 'choosy' about partners for copulation. This should be particularly true for extra-pair copulations, where participants may have had less time to assess the 'health' of each other. Evidence from work on chickens suggests that the probability of an individual female contracting a particular STD depends on the number of inseminations that she receives (e.g. Perek *et al.* 1969). In addition, the spread of a disease has been shown to be facilitated by the number of copulatory partners, as with goose venereal disease, which is much more prevalent in large breeding flocks than in small ones (Stipkovits *et al.* 1986). Wagner (1991) reports that female razorbills *Alca torda* engaged in extra-pair copulations for the purpose of mate appraisal tend to avoid direct cloacal contact, possibly in order to avoid the risk of contracting an STD. However later in the breeding season, when males are probably no longer producing sperm (and therefore much less likely to transmit an STD) females allow males to have cloacal contact with them.

Exposure of the cloaca is widespread in precopulatory displays, both from the male to the female and vice-versa. It is tempting to suggest that this behaviour may have evolved in response to the existence of STDs, and that by exposing the cloaca to potential partners, birds are in effect displaying a 'clean bill of health'. As Hamilton (1990) has suggested, the evolution of the extraordinary cloaca-pecking display in the dunnock *Prunella modularis* (Davies 1983) could conceivably represent investigation by the male of the cloaca of a potential copulatory partner, in order to determine the presence or absence of any STDs. Two other species known to perform cloaca-pecking or female cloaca-inspection (alpine accentor *Prunella collaris*; (Nakamura 1990); house sparrow *Passer domesticus*; (Møller 1987)) are characterized, in the former species by an extreme degree of polygynandry and in the latter by a high level of extra-pair copulation; in both cases a copulating male is likely to copulate with a female that has already copulated with several other males. As some STDs result in conspicuous lesions around the cloaca (e.g. venereal pox in the turkey; Metz *et al.* 1985), visual inspection of external genitalia may have evolved as a means of avoiding infected partners. After copulation, birds frequently respond with vigorous preening, which Read (1990) has suggested may be adaptive, in that it may remove ectoparasites that have been transferred during copulation. In addition, Hart *et al.* (1987) have shown that rats prevented from genital grooming after copulation are more likely to contract an STD.

If the prevalence of STDs varies between species or populations, then STDs have the potential to influence the evolution of avian mating strategies, particularly the extent to which a species is polygynous or polyandrous, both 'socially' and genetically. This conclusion results from a consideration of the effect that sexually transmitted parasites, as defined above, have on the host. I use the terms parasite 'intensity' to refer to the number of parasites on a particular host, and parasite 'prevalence' to refer to the proportion of the host population that is parasitized (following Clayton *et al.* 1992). Macroparasites, such as arthropods and helminths (Anderson & May 1979), tend to have non-lethal, chronic effects, where the debilitatory effect of the parasite is a function of parasite intensity. In contrast, microparasites (bacteria and viruses; Anderson & May 1979) have rather different effects, typically either resulting in host mortality or complete recovery to a state of immunity; here the important consideration is whether or not the host comes into contact with the parasite at all. The division is a generalization, since some parasites show characteristics of microparasites whilst at the same time, their effects on host fitness are dependent on intensity of infection (Clayton *et al.* 1992).

If we first consider the effects of sexually transmitted macroparasites on the behaviour of hosts, those with increasingly severe effects on their hosts will tend to select for increased monogamy in the host species. Typically, for females the costs of exposure to a virulent macroparasite will be more severe than for

males, because the rate at which females can reproduce is less than for males, but this will not affect the basic conclusion, that where a species is exposed to a relatively more virulent STD, selection favours those individuals having fewer copulatory partners than when STDs are less costly.

The effect of virulent microparasites on host behaviour is a little more difficult to determine; although Clayton *et al.* (1992) have shown that increasingly virulent microparasites provide increasing opportunities for selection, prevalence rather than intensity may be a more important factor in host evolution. If the parasite is exceptionally virulent, an application from foraging theory, the concept of risk-sensitivity (Stephens 1990), may prove more appropriate. In this scenario, where the risk of host mortality is very high if exposed to a particular parasite species, selection will favour those individuals that reduce risk, compared to the situation for a parasite with a lower risk of host mortality. Of course, for the first potential partner, under any circumstances, individuals must be risk-prone. It is likely that the effects of both macro- and microparasites will act in tandem in the evolution of host behaviour, and the likely effect is that host reproductive behaviour will be pushed further towards monogamy than is the case when considering either macro- or microparasites in isolation.

As Ewald (1983) has stressed, a parasite should show 'adaptive severity', in that its effects on a host should evolve so as to balance parasite reproduction within the host with dispersal to new hosts. If this is the case, then as an STD drives a host species' mating system towards monogamy, its virulence should decrease, increasing again if the host is evolving towards polygamy. Such cycles might evolve to an equilibrium point, but in any case will tend to decrease the level of polygamy or multiple mating within the population. Equally, it is possible that the evolution of monogamy could drive lowered incidence or virulence of an STD; the direction of causality could be in either direction. Although other factors are certainly important in determining mating systems (such as female dispersion, resource dispersion (Emlen & Oring 1977)), this model suggests that, other things being equal, species with high levels of STDs are less likely to mate multiply compared with species with low levels of STDs.

(*b*) *Sexual asymmetry*

In species exhibiting internal fertilization, the sex receiving the gametes will be under stronger selection to avoid exposure to STDs. This asymmetry results from the length of time that the ejaculate remains in the reproductive tract of the female compared with the length of time that the male cloaca is in contact with that of the female. This asymmetry is particularly marked in most birds, where males do not possess an intromittent organ, and copulation is usually very brief (of the order of a second for most passerine species). Although copulation duration is likely to be influenced by a large number of factors (including predation risk and the likelihood of sperm competition (Birkhead *et al.* 1987)) selection to avoid infection by STDs may have played some part in determining male copulation duration. For females, the chance of contracting an STD is probably little affected by copulation duration, except that the likelihood of ectoparasite transferral (Hamilton 1990) may be time-dependent. Although in most bird species females are thus hypothesized to be much more choosy' than males about copulation partners, in those species where males possess an intromittent organ (approximately 3% of extant species (Briskie 1990)) the selection pressure on males to avoid STDs may be closer to that experienced by females. Unfortunately, most qualitative predictions arising from the asymmetry discussed above will be difficult to distinguish from those arising as a consequence of differential investment in gametes by the sexes (Trivers 1972). A related asymmetry concerns the optimum number of copulatory partners predicted for each sex, in the presence of a given STD. As females benefit less than males do for each extra copulatory partner (Trivers 1972), for females the optimum number of copulatory partners for a given risk is lower than that for males; this of course follows from the fact that a female is usually physiologically constrained in the amount that she can reproduce. The possible extra benefits to a female of each subsequent copulatory partner are probably relatively small compared to males (increased offspring fitness might result from increased investment by males, or through 'good genes'; Madsen *et al.* (1992) present evidence that female adders *Vipera berus* enjoy increased offspring viability through multiple copulations).

The degree of asymmetry in risk of infection may, however, be reduced by the effects of testosterone in males. Folstad & Karter (1992) have proposed that reproducing males experience a trade-off, in that whereas testosterone stimulates development of the characters used in sexual selection, it suppresses the immune system, therefore increasing susceptibility to parasites (the immunocompetence-handicap hypothesis). Given this, at the time when male birds copulate, and are exposed to STDs, they may be most at risk of contracting them.

(*c*) *Physiological responses*

As females are exposed to male ejaculates for longer than males, they will be under strong selection to evolve measures to counteract STDs within the reproductive tract. One consequence may be selection for females to make their reproductive tracts 'hostile' to foreign bodies (Austin 1975). As sperm may also be inadvertently destroyed by female defences, this may in turn provide an explanation of why males have to produce so many sperm in order to fertilize an egg (Birkhead *et al.* (1993) also discuss this question). It is presumably of little consequence to a copulating female whether a male expends relatively little or a great deal on an ejaculate, provided that her fecundity is not decreased. Supporting evidence for the suggestion that the hostility of the female reproductive tract may influence ejaculate size comes from the observation (in mammals) that the degree of hostility in the

female reproductive tract is reduced at the time of ovulation (Austin 1975). At this time the risks of contracting a potentially costly STD may be weighed against the risk of failing to reproduce resulting from an 'over-enthusiastic' immune system.

As Møller (1988, 1991) has demonstrated, there is a strong relationship between ejaculate size and testes size in birds, and the extent of sperm competition, as judged by a species' mating system. However, part of the increase may be explained by the effect of STDs selecting for a hostile reproductive tract in the female. Where multiple mating is relatively frequent, conditions will favour the existence and proliferation of STDs. This in turn will select for greater hostility in the female tract, with the result that more sperm are inadvertently destroyed by the immune responses of the female, hence leading to selection for larger ejaculates and larger testes in males. Although one possible female response might be to be hostile towards only the 'non-sperm' part of the ejaculate, this would select for STDs to mimic the characteristics of the sperm that females were using to discriminate.

Although males will be under strong selection to evolve means for their sperm to overcome female hostility, the same will apply equally, if not more strongly, to sexually transmitted pathogens, and their much shorter generation time presumably facilitates the evolution of counteradaptations. The composition of the bacterial flora on the goose phallus shows consistent fluctuations over time, with certain species occurring more frequently during the period in which copulations occur (Marius-Jestin *et al.* 1987), although whether these particular species rely on sexual transmission is unclear. It might be argued that it would be better for females to avoid copulations with diseased or infective males altogether rather than develop defences at the physiological level. However, any parasite that depends solely upon copulation as its means of transmission will be under very severe selection to prevent hosts from developing indicators of infection, or means of detecting infection in potential partners, and selection will thus surely favour females with multiple defences.

A class of parasite not so far considered, is that consisting of cellular organelles and cytoplasmic plasmids, and these may have very important consequences for the evolution of some aspects of reproductive physiology. Hurst (1990) argues convincingly that such parasites may have triggered the evolution of anisogamy, because the fittest zygotes will be those with the smallest parasite diversity and zygotes with the smallest parasite diversity will be formed from the most disparately sized gametes. Anisogamy would reduce the number of such parasites in the smaller (male) gametes, and the long term consequence of this selection for small gametes may be male sperm which have very little cytoplasm. Such parasitic elements are, of course, by definition STDs, but I will not be considering them further.

4. INDIRECT SELECTION

The nature of host-parasite interactions and their power to explain the evolution of secondary sexual characteristics have received a great deal of attention in the last ten years (Loye & Zuk 1991; McLennan & Brooks 1991). Sexually transmitted diseases form a smaller system than that usually considered under the umbrella of 'parasites', and although they presumably have potential to drive the evolution of complex or extravagant traits in similar ways to other parasites (Hamilton & Zuk 1982), they also have the potential to affect several unique aspects of reproduction associated with copulation itself.

As discussed above, STDs are a potent force selecting for hostility to foreign bodies in the female reproductive tract. The resulting 'arms race' between STDs and female hostility is likely to result in high mortality of sperm that enter the reproductive tract ('caught in the crossfire' to extend the metaphor). As a result there will be selection on males to develop 'anti-hostility' measures to overcome female hostility, and then selection on STDs to mimic any successful adaptations of male sperm. The combination of the three systems (female hostility, male anti-hostility and STDs) has the potential to yield complicated coevolutionary cycles, which are unlikely to follow predictable patterns, and could result in behavioural, anatomical and physiological adaptations to both male and female reproductive biology. The potential complexity generated could provide the genetic substrate on which a subtle means of female choice – choice for males on the basis of their ability to be successful at sperm competition (Harvey & May 1989; Curtsinger 1991) – could operate.

In addition, and distinct from the situation described above, is the possibility that STDs and characteristics of the male ejaculate or copulatory behaviour may co-evolve. A possible example is provided by Crittenden's (1981) description of the means by which lymphoid leukosis virus propagates in chickens: this retrovirus incorporates itself into gametic DNA and is therefore transmitted vertically to offspring, as well as horizontally as with more conventional STDs (Crittenden 1981). Once incorporated into germ line cells, it is easy to imagine possible conflicts of interest between parts of the virus genome and parts of the host genome (perhaps in ejaculate size; paternity assurance is of less importance to a virus which can also be passed horizontally, for example). A tantalising example is provided by the work of Segura *et al.* (1988), which suggests that male chickens positive for lymphoid leukosis virus produce larger ejaculates than non-infected males.

5. FURTHER WORK

What has preceded is to a large extent speculation, although it is difficult to believe that STDs are either absent or unimportant as evolutionary forces in wild bird populations. To assess their importance, good empirical data revealing both their occurrence and pathogenicity are required. The first objective is probably achievable; certainly swabs from the cloaca, or semen (easily collectable from male birds during the breeding season: Wolfson 1952; Quay 1985) can be cultured to determine their associated bacterial

flora (e.g. Flammer & Drewes 1988). Similar methods may also provide records of macroparasites. The second objective, the assessment of the pathogenicity of any STDs in wild birds, may be much more difficult. As Toft (1991) has pointed out, one of the problems with many of the interspecific tests of the Hamilton–Zuk hypothesis is that they have merely quantified the frequency of occurrence of parasite species. Two problems with this are, firstly, that parasite–host interactions are highly dynamic, and secondly that parasite burden and parasite-induced host mortality may be inversely related (Anderson & May 1978). In this case, surveying a species and finding relatively few diseased individuals or parasites does not imply that parasites do not contribute importantly to host mortality.

However, a number of predictions can be made on the basis of the hypotheses advanced above. At present the data to test them do not exist, but it should be possible to collect the necessary information using methods already available. If selection on females has been so strong because of their greater exposure to STDs, then the reproductive tract of the female should be more 'hostile' than that of the male. There should be a relationship across species, with female reproductive tract hostility being higher in species where there are many STDs, or where potential STDs are highly pathogenic. If cloacal inspection has evolved, at least in part as a result of STDs, then cloacal inspection should also play a role in mate choice. There should be a positive relationship across species between the frequency of cloacal inspection and the prevalence and costs of STDs. Making predictions about differences in behaviour between males and females is difficult because the direction in which the prediction is made depends crucially upon the transmission dynamics of the disease, any differences in pathogenicity between the sexes and on the relative benefit of multiple mating to the sexes. Where STDs are relatively more costly to males than to females (as a consequence of the immunocompetence handicap for instance (Folstad & Karter 1992)), and where the extra benefits gained by males from mating multiply, relative to those gained by females, are small, conditions may favour females having more mating partners than males. As the average number of partners must be the same (given equal sex ratios), this suggests that these conditions may favour the evolution of polyandry, and that males might therefore evolve to become the sex providing parental care (see also Hamilton 1990).

Finally, in species where STDs play an important role, birds will tend to have evolved structures that facilitate detection of a sexually transmitted infection (e.g. conspicuous 'lips' to cloaca, or white feathers surrounding it: see Nakamura 1990); these will, of course, merely be a specialized case of a revealing handicap in the sense suggested by Hamilton & Zuk (1982).

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