RELATIONSHIPS OF BIRDS AND ARBOVIRUSES[1]

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SEVERAL epidemics of disease in man have recently attracted wide public attention due to their spectacular characteristics, of particular interest here being the involvement of birds. Ornithologists have been bombarded with questions and have lacked answers. This paper reviews the relationships of birds and arboviruses, illustrates the impact of epidemics of arbovirus disease on human communities, and outlines some ornithological information needed by virologists and epidemiologists.

The term arboviruses refers to viruses that infect hemophagous arthropods when they ingest infected vertebrate blood. The viruses multiply in the tissues of the arthropod and are transmitted by bite to susceptible vertebrates.

About 150 different arboviruses are now recognized and they occur in practically all regions of the world. About 50 are known to produce disease in man or domestic animals, and 8 of these have been isolated from wild birds. These latter are eastern (EE), western (WE), St. Louis (SLE), and Japanese B (JBE) encephalitis viruses; West Nile, Sindbis, and Ilheus viruses; and at least one member of the Russian tick-borne complex of viruses.

Undoubtedly other arboviruses will be discovered; the geographic distribution of those now known may be much greater than recognized, and knowledge of the ecology of most of them is too incomplete to assess adequately their host ranges or their importance in causing disease (Scherer, 1963; Hammon, 1961–62; Work, 1963). The following summarizes information available on the relationships of birds and those arboviruses known to produce disease in man.

MOSQUITO-BORNE ARBOVIRUSES

Geographic distribution.—Although EE virus only occasionally produces

[1] This paper was prepared by the late Donald Stamm as chairman of the subcommittee on Relation of Birds to Arboviruses, which is a part of the American Committee on Arboviruses. The manuscript was revised by the members of the subcommittee (Robert J. Newman, Joseph J. Hickey, C. Brooke Worth, and Maurice W. Provost) in 1963. After Dr. Stamm's death the manuscript was again revised by the committee and edited by David E. Davis. The present members are Richard O. Hayes (chairman), David E. Davis, Robert W. Dickerman, Robert J. Lechleitner, Robert J. Newman, Maurice W. Provost, and C. Brooke Worth. Part of the subcommittee's review was carried out with the encouragement of William C. Reeves and financial aid from the Rockefeller Foundation through the School of Public Health, University of California. Reprints should be requested from the Communicable Disease Center, Atlanta, Georgia.

disease in man and domestic animals, it occurs frequently in those states bordering the Atlantic and Gulf coasts of the United States, from Massachusetts to Texas, and occasionally as far inland as Michigan and Wisconsin (Beadle, 1959; Bryne *et al.*, 1961; Hayes *et al.*, 1962b; Henderson *et al.*, 1962). It has also been isolated in Panama, Brazil, Trinidad, British Guiana, the Dominican Republic, Jamaica (Belle *et al.*, 1964), Czechoslovakia, and the Philippines.

WE virus occurs throughout the United States (Thomas and Smith, 1959) and in southern Canada, Brazil, Uruguay, Argentina, Mexico, Panama (Galindo *et al.*, 1964), and British Guiana.

SLE virus is generally distributed west of the Mississippi in the United States and has been active in Missouri, Ohio, Indiana, Kentucky, and Florida. It has also been isolated in Trinidad, Panama (de Rodaniche and Galindo, 1961), and Haiti. Serological surveys suggest that it has been widely active in the Caribbean area and in Ecuador.

JBE virus probably has the widest distribution of any of this group of viruses (Scherer, 1963). It has occurred in the far eastern USSR, Korea, China, Taiwan, India, Burma, Malaya, Java, Sumatra, the Philippines, and Guam. Sindbis virus has been isolated in Egypt, India, South Africa, and Malaya. West Nile virus isolations have been made in Egypt, Uganda, South Africa, Israel, and India. Ilheus virus has been isolated in Brazil, Trinidad (Downs, 1963), Honduras, and Panama. In addition to these mosquito-borne viruses that have been isolated from wild birds, there is evidence that Murray Valley encephalitis virus also exists in a bird-mosquito cycle. This virus has caused important epidemics of disease in man in Australia and was recently isolated from a fatal human case in New Guinea.

Bird species and population susceptibility.—Susceptibility of a bird species to infection with these viruses is best established by demonstrating viremia (presence of virus in peripheral blood) resulting from natural or laboratory infection. On this basis, at least 52 species of birds are known to be susceptible to EE virus, 51 to WE virus, and 24 to SLE virus (Karstad *et al.*, 1959, 1960; Reeves and Hammon, 1962; Stamm, 1963). These species are members of 26 of the 75 families and 10 of the 20 orders of North American birds. No avian species has been demonstrated to be refractory to infection with any of these viruses and additional species should be found susceptible as our study continues. Application of this information to a specific bird population in Alabama resulted in the deduction that over 85 per cent of the individual birds present were of species already known to be susceptible to one or more of these three viruses (Stamm, 1963).

Transmission and population involvement.—Numerous studies show

that many mosquitoes feed avidly on birds (Reeves *et al.*, 1963), although various species differ greatly in their preferences among vertebrates.

A distinction must be made between susceptibility to infection of a vertebrate species and its potential for serving as a source of virus to infect mosquitoes. Different mosquito species vary in regard to the concentration of virus, in a blood meal, necessary to infect them. The concentration of virus necessary to infect a given mosquito species also varies with different viruses.

Generally, small passerine birds have a greater potential for infecting mosquitoes with EE virus than do larger birds. Domestic fowl (*Gallus domesticus*), however, do circulate SLE virus at concentrations adequate to infect the usual vector mosquito species (Sudia and Chamberlain, 1959), and domestic pigeons (*Columba livia*) may be especially important since they may die while infected (Gainer *et al.*, 1964). In the case of JBE virus, herons and egrets (Ardeidae) appear to be especially important in Japan (Scherer, 1963; McClure, 1963), but the role of smaller birds in the epidemiology of this disease has been inadequately assessed.

An efficient transmission cycle, therefore, requires susceptible hosts that produce blood virus concentrations sufficiently high to infect mosquitoes. In addition, a proper ecological association of hosts and vectors is required. They must coincide properly in time and space, and their behavior and abundance must be adjusted so as to allow a rate of transmission that will assure the long-time survival of the virus. Host-vector relationships have been studied to some extent by assessing relative attractiveness of different host species to mosquitoes (Hayes, 1961). Infection and transmission rates in mosquito populations have been examined (Reeves *et al.*, 1961). Another approach has been to determine the source of blood in engorged mosquitoes by serological tests. Recently, considerable progress has been made in refining techniques for the latter approach. Species of mosquitoes vary in their spatial distribution, and different biting rates at varying elevations above the ground have been reported. Also, their abundance varies in different years according to weather conditions (Hayes *et al.*, 1964).

Some progress has been made in developing quantitative methods of studying virus activity in wild bird populations (Stamm *et al.*, 1960, 1962). Additional evaluation and new methods are needed since findings by similar techniques may vary in different habitats (Anderson and Maxfield, 1962). The use of captive birds as sentinels (or indicators) of virus activity has yielded helpful information in some areas (Hayes *et al.*, 1962).

It has been demonstrated repeatedly in a number of places during the peak period of transmission that seven per cent or more of the birds present were circulating EE or WE virus. Since viremia in individual birds is

detectable for only three to six days, this indicates very rapid spread of virus through bird populations. Antibody surveys have shown that by the end of the transmission season up to 70 per cent of the birds in local populations have been involved (Stamm, 1963).

Course of infection in individual birds.—After the bite of an infected mosquito, or laboratory exposure, an individual bird develops viremia, which usually remains at levels sufficiently high to infect mosquitoes for only three to six days. Techniques are not yet available to assess adequately the mortality produced by these infections in wild birds under natural conditions. It appears that mortality produced by EE virus infection in native North American species is negligible, at least in birds past the fledgling age. Introduced species such as Ring-necked Pheasants, *Phasianus colchicus*, Chukar, *Alectoris graeca*, House Sparrows, *Passer domesticus*, young peking ducks, *Anas platyrhynchos*, and chickens are, however, known to suffer high mortality from EE infection (Dougherty and Price, 1960; Herman, 1962; Locke *et al.*, 1962). Nothing is known about possible effects of infection on the physiology of birds, their mobility, their susceptibility to predators, or their parental functions. Within a few days after the termination of viremia, an antibody specific to the infecting virus appears in the blood serum. Antibody concentration reaches a peak after several weeks, maintains a plateau for several months, and by the sixth month in some individuals declines below levels considered positive (Stamm *et al.*, 1962). Re-exposure to the same virus usually again raises antibody levels for a time. Most birds probably are completely immune to reinfection after one exposure.

Overwintering and interepidemic survival of mosquito-borne viruses.—Epidemics of these diseases occur irregularly in time and location. In temperate areas, where most epidemics occur, mosquito populations are greatly reduced or absent during winter months. A number of different mechanisms of virus survival have been postulated.

Virus transmission between birds and mosquitoes may persist in small areas of especially favorable habitat where some mosquitoes are active throughout the year. A continual supply of susceptible birds may move into such areas for food or water or by ordinary movement of migratory birds. Migrant birds may then transport viruses from these areas to others favorable for transmission during spring and summer months. Investigations of this mechanism have yielded negative results to date but have been conducted on a scale too small to make an evaluation (Stamm and Newman, 1963). Banding studies linking wintering birds to specific breeding areas (and vice versa) are of considerable interest to virologists and studies to enlarge our knowledge of this phenomenon (e.g.,

Ali, 1963) should be encouraged. Summaries of regional migration patterns are very useful to virologists.

Survival of these viruses through winter months in hibernating mosquitoes has been considered. Again, this mechanism has not been proved to exist (Rush *et al.*, 1963), but much more work is necessary before the hypothesis can be discarded.

Survival of mosquito-borne viruses in bird mites or in ticks has been studied thoroughly. The possibility of these mechanisms being important is very remote.

Small mammals, including bats, have been investigated to some extent as hosts of overwintering virus. Laboratory studies have confirmed the possibility that this mechanism can operate, but the occurrence of such a cycle in nature has not been demonstrated (Sulkin *et al.*, 1963). Recently, some evidence has appeared in Colorado and New Jersey that small rodents may be hosts.

The persistence of virus in some birds, perhaps in a latent form, has been demonstrated by isolation of virus from tissues, including blood, between 55 and 306 days after the initial infection. It has not been demonstrated, however, that sufficient concentration of the virus circulates a second time in birds to infect mosquitoes and re-initiate the natural cycle (Reeves and Hammon, 1962).

WE virus infection has been shown to persist, through winter hibernation, in garter snakes, *Thamnophis* spp., the entire cycle having been carried through experimentally (Gebhart and Hill, 1960; Thomas and Eklund, 1962). EE virus-neutralizing antibody has been detected in snakes, a turtle, and an alligator at the time of capture. Snakes, lizards, alligators, and turtles have been shown to respond to inoculation of EE virus with high titers of viremia or circulating antibody, or both (Karstad, 1961). No virus has been isolated from a naturally infected reptile, although considerable numbers have been tested (Hayes *et al.*, 1964). It has been demonstrated, therefore, that such a cycle can occur, but it has not been demonstrated that it does occur in nature.

The possibility that mechanisms of transmission other than by mosquitoes occur in nature cannot be ruled out (Burton *et al.*, 1961; Winn and Palmer, 1961). It has been shown that direct transmission can occur in captive Ring-necked Pheasants and young peking ducks (Dougherty and Price, 1960). While transmission in nature is usually by means of mosquitoes, the possibility of transmission by contact exists because it has been demonstrated in captive birds (Bourke, 1964).

TICK-BORNE ARBOVIRUSES

Small wild rodents are considered to be the primary vertebrate hosts

of the Russian tick-borne complex of viruses in Eurasia, but the vector ticks are known to feed on over 100 species of birds and mammals. Russian and European workers have been actively investigating the role of birds, and of arthropods that occur in bird nests, in the life cycles of this group of agents (Libikova, 1960; van Tongeren, 1960). The virus has been isolated at least five times from birds, and many species of birds have been shown to possess antibodies. Chicks are susceptible to at least some agents of the group and can serve as a source of virus for other ticks.

Although birds have not been shown to be associated with tick-borne viruses in North America, at least one tick-borne virus is known to be present (McLean and Larke, 1963). Ticks are known to occur on birds in the United States (Nibley, 1962), and migrating birds are known to transport ticks for long distances in the Mediterranean region (Hoogstraal and Kaiser, 1961).

Overwintering of tick-borne viruses is relatively simple; ticks infected as larvae or nymphs can carry virus through the winter and then infect hosts on which they feed during the following year.

HUMAN EPIDEMICS OF DISEASE CAUSED BY ARBOVIRUSES

Activity of EE and WE viruses in bird and mosquito populations has frequently been demonstrated during summer and fall months in wooded freshwater swamps along the Atlantic and Gulf coasts of the United States (Stamm, 1958; Stamm *et al.*, 1962). In fact, EE virus has been found on almost every occasion that it has been sought in these localities and seasons. EE virus has very frequently produced epidemics of disease in horses and Ring-necked Pheasants, but relatively few human epidemics have occurred. WE and SLE epidemics have been important public health problems in the western United States, especially California, for at least 20 years. Excellent accounts of long-term epidemiological studies conducted in Kern County, California, have been published (Reeves and Hammon, 1962; Reeves *et al.*, 1963) and the problems of immunization of humans summarized (Smadel, 1963).

We lack understanding of the ecological factors that usually confine activity of these viruses to a bird-mosquito cycle in natural habitats but periodically allow them to produce disease in man and domestic animals. Annual variations in temperature and other weather conditions appear to be important (Hess *et al.*, 1963). Spectacular epidemics of encephalitis in man occurred in New Jersey in 1959 and in Florida in 1962. Their impacts on the communities involved received attention throughout the country.

The 1959 epidemic of EE in New Jersey had a forceful economic and emotional impact in addition to the public health aspects of such epidemics.

In all, 33 human cases were reported, 21 persons died, and others were left with severe after effects. The economic effects of the epidemic were felt primarily in the resort and recreational areas in the three counties involved. Dollar volumes of resort business fell off 30 to 75 per cent. The hotel industry in Atlantic City alone suffered a loss estimated at two million dollars. Rumor and irrational thinking produced anxiety approaching panic in many parents. Releases by news media spread fear and hysteria throughout the state. Many persons considered evacuation. The possibility of complete school closure was discussed, and pupil absenteeism at times exceeded 50 per cent.

In the fall of 1959, an epidemic, presumed to be St. Louis encephalitis, occurred in Pinellas County, Florida. The 68 human clinical cases recorded involved 5 deaths. In October, November, and December, 1961, another epidemic of 25 human cases with 7 deaths occurred in Pinellas, Manatee, and Sarasota counties. Serological evidence again indicated infection with SLE virus or a closely related agent. Between 1 July and 27 September 1962, 350 suspected cases of encephalitis were observed in Pinellas, Manatee, Sarasota, and Hillsborough counties. SLE virus was isolated from some of the 20 fatal cases, and serological evidence of SLE infection obtained in many other cases.

The general reaction of the community to these epidemics was quite analogous to the 1959 experience in New Jersey. In addition, a reaction of considerable interest to ornithologists occurred in 1962. The news media repeatedly circulated opinions that feeding wild birds in the city of St. Petersburg produced concentrations of birds that were responsible for the epidemic. The city passed an ordinance making it unlawful to "place or cause to be placed in the open any bird seed, bird food, or other substance that is edible by [wild] birds." The rationale of this action cannot be evaluated until much more is known about the complex interrelationships between birds and mosquitoes. Information obtained by systematic bird counts showed a considerably greater density of certain birds (notably Mourning Doves, *Zenaidura macroura*, and Cardinals, *Richmondena cardinalis*) in St. Petersburg than in neighboring Tampa (which had a much lower SLE case rate). The Florida State Board of Health reviewed the entire situation in December and decided not to press for any bird control beyond that which had been undertaken during the epidemic.

During the late summer of 1964, epidemics of St. Louis encephalitis occurred in a variety of places, producing about 1,000 cases. The outbreak in Houston, Texas, with about 300 cases and 34 deaths, was the largest, while the Philadelphia-Camden, Pennsylvania, New Jersey, area in September had numerous cases and 8 deaths. In some small towns (McLeansboro, Illinois, and Danville, Kentucky) locally spectacular

epidemics occurred. In some places a few cases were attributed to eastern or western encephalitis virus by various tests. Rather intensive studies of birds in Houston were initiated, but the results are not yet available. Although the viruses in the Tampa Bay region were inconspicuous in 1964, well-planned studies of bird populations are underway. The total number of cases for the summer of 1964 cannot yet be determined, nor can the role of birds be assessed for several years. However, the large number and wide distribution of cases have stimulated extensive ornithological research.

The emergency measures proposed to stop epidemics of this type include the suggestion that the vector population of birds be wiped out by heavy application of a powerful insecticide like aldrin, dieldrin, or heptachlor. It is true that application rates of two or more lbs/acre of these compounds will reduce local bird populations on the order of 80 or more per cent (Scott *et al.*, 1959; Clawson and Baker, 1959) and that even the spraying of DDT can wipe out 90 per cent of the bird population of a residential area (Hunt, 1960). It is equally true that the bird population of a treated area will seemingly recover after the insecticide has disappeared (Mills, 1959); but it is also true that research has shown that the chlorinated hydrocarbons are now subjected to striking methods of biological concentration and that they are capable of selective (i.e., varying from species to species) destruction of the upper layers of animal communities for as long as five years after application. Heavy application rates in aquatic environments will, of course, kill off not only birds but large numbers of fish and fish-food organisms as well.

The really critical causative mechanism is juxtaposition of donor and vector, and later of vector and recipient; it is not the absolute numbers of any of the three entities. In the case of SLE in St. Petersburg, for instance, the critical dimensions are amount of contact (= juxtaposition) between birds (= donor) and *Culex nigripalpus*, and later, the amount of contact between the same vector and susceptible man (= recipient). Obviously, the chances of juxtaposition increase with increases in populations of either birds, vectors, or susceptible men. But it must not be overlooked that juxtapositions may increase through ecological and behavioral modifications in host, vector, or recipient without changes in their absolute numbers. Better synchronization between bird nesting and mosquito production in a particular year could greatly increase the bird-vector juxtaposition. Weather conditions extending the average life span of mosquitoes by only a few days could materially increase the critical vector-man juxtaposition. In attempting to explain epidemics, one could postulate such factor permutations *ad infinitum* without, in any case, assuming greater numbers of any of the three pathogen-carrying animals in the

cycle. The fact that such poorly understood and for the most part unmeasurable variables are superimposed on densities of birds, mosquitoes, and susceptible men is almost certainly why epidemics of mosquito-borne encephalitides are so often unexplainable. Once an epidemic is afoot, however, it certainly can be stopped cold by removing all birds, or all mosquitoes, or all men. This nevertheless, is not proof that unusual numbers *per se* of birds, mosquitoes, or men caused the epidemic. For cause we must fall back on juxtaposition as the only sure thing.

Interposing barriers between mosquitoes and man can break the critical juxtaposition. Thus, malaria control authorities recommend that domestic animals be quartered around the perimeter of villages so that anopheline mosquitoes feed on the animals which they encountered first and thus stay away from man (Brumpt, 1944). Recent recurrences of malaria in parts of British Guiana have been attributed to a drastically reduced domestic animal : man ratio (Giglioli, 1963). This suggests one more explanation (out of hundreds possible) for the SLE epidemic in the Tampa Bay area. There is no question but what *Culex nigripalpus* is essentially a bird feeder. If birds are not available it feeds on mammals. It is therefore admirably suited to keep the SLE virus circulating in the bird population. If it feeds appreciably on man, which it must do to cause SLE epidemics, it must be because there are not enough birds being interposed between mosquito and man. The ecological complexity of SLE is so vast that an explanation such as this can be advanced with as much logic as the assumption that an overabundance of birds causes epidemics.

ORNITHOLOGICAL INFORMATION NEEDED BY VIROLOGISTS

A number of virologists who have conducted long-term investigations on birds and arboviruses were asked for statements summarizing the ornithological information that would be most helpful to them. The replies from E. M. Buescher, C. M. Eklund, R. O. Hayes, A. D. Hess, and W. C. Reeves were very helpful in assembling the following synthesis:

1. A great deal of specific information is needed in the general field of bird population dynamics. Techniques for such studies need further development and evaluation. The laboratory tests employed in virologic studies are expensive and time consuming, and knowledge of the age of individual birds adds greatly to the value of data obtained. There is a need for a comprehensive review of existing information on methods of aging and sexing all species of North American birds, especially small passerines. This will reveal gaps existing in present knowledge and guide additional research. Census techniques need further development and critical evaluation. Methods are needed that will efficiently assess bird abundance at all seasons in a variety of habitats and especially in mix-

tures of habitat such as urban and agricultural areas. Virologists need simple, efficient, standardized methods for following changes in bird abundance and population age structure. Such methods will permit them to obtain meaningful annual infection and antibody rates in birds in a variety of habitats and seasons and to correlate these with parallel information on arthropod populations. There is strong suspicion that human alteration of natural habitat with its consequent upsetting of natural controls on the transmission of disease-producing agents is a large factor in the occurrence of arbovirus epidemics. Careful studies of bird population abundance, dynamics, and mobility (all species present) are needed in a variety of habitats in their natural state and after human exploitation. These studies must be correlated with dependable assessment of virus activity in the bird and mosquito populations. Information on changes in geographic distribution of bird populations needs to be assembled and correlated with available knowledge on the history of arbovirus epidemics.

2. Virologists are greatly interested in bird migration and its potential for local and long-distance dispersion of viruses. The chief deficiency in existing information is that it relates to bird species and groups and general regions. The virologist needs information relating to individual birds and specific localities. More information is needed on timing, routing, and length of stopover in the migration of individual birds. The factors that determine arrival and departure dates and their annual variations should be examined. Again, there is a shortage of basic techniques for obtaining such information. Coordinated banding studies on migratory species in their breeding and winter locations have produced promising results and should be expanded. The examination of gastroliths, seeds, and pollen, in the gizzards of migrant birds may prove to be an important technique and critically needs development. Identification of subspecies may prove to be a very useful tool and existing information permitting this should be assembled and analyzed. The intermingling of migrant species and subspecies and their mixing with permanent resident birds along migration routes with resultant opportunity for virus exchange should be examined.

3. Collaborative studies between virologists and ornithologists may reveal that some species of birds are more important hosts than others. Such findings would permit concentration of virologic effort on individual species and habitats and result in increased efficiency.

4. Detailed studies of bird die-offs should be made. These studies will result in increased accuracy of differential diagnosis and add to information on general disease ecology in birds.

5. Virologists make extensive use of wild bird species as experimental

hosts for arboviruses. More information is needed on methods of maintaining wild birds in the laboratory. This knowledge will facilitate studies on physiologic activities that fluctuate seasonally and in connection with migration. Normal physiological and stress factors may affect susceptibility to virus infections.

6. Very little information is available on the immediate circumstances of the bird/mosquito juxtaposition. Day? Night? At nest? While feeding? While brooding? Nestling? Adult? An exception to the prevailing ignorance here is the remarkable study by Russians in the Central Urals which revealed, among other interesting things, that nestlings of passerine and picine birds are bitten by mosquitoes mainly from the fifth to eighth day after hatching because homoiothermy is not attained before the fifth day and plumage growth protects the nestling after the eighth day. The reaction of various bird species to mosquito annoyance and biting is in need of exploration. Daily sequences of movement and rest on the parts of birds and mosquitoes, habitats frequented in daily or nightly rounds, distances to which the mosquito orientation stimulus of a bird or a group of birds reaches out, how far the mosquito ranges from its point of origin to obtain its first bird blood—these are the types of problems that need resolving. To place the habits of birds within the context of mosquito ecology will demand collaborative studies between ornithologists and entomologists. Laboratory studies should determine if viremia weakens birds, affects their ability to store fat, or suppresses their inclination to migrate.

CONCLUSIONS

Epidemics of disease produced by arboviruses have an important and complex emotional, political, and economic impact on affected human communities. This impact is much greater than consideration of actual mortality would suggest.

Efficient methods of predicting, preventing, or controlling these epidemics can be developed only when complete information is available on the life cycle of the viruses. Much of this information can best be provided by ornithologists. Extensive new research is needed, as well as a great deal more correlation of existing ornithological, virological, and entomological information.

Especially needed from ornithologists is information on bird population abundance, dynamics, and movements. More information is also required on the habits of individual birds, especially concerning their interrelationships with mosquitoes. A careful evaluation of the effects of human alteration of natural habitat on the ecology of infectious agents must be made.

The life histories of arboviruses can be elucidated only by focusing the efforts of many disciplines on all facets of their ecology. Wider interdisciplinary collaboration is needed not only in conducting the research but in planning it and evaluating results.

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