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PARASITES OF THE AMERICAN WHITE PELICAN

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ABSTRACT Metazoan symbionts, including parasites, infecting the American white pelican (AWP) *Pelecanus erythrorhynchos* comprise a list of 75 species, 7 of which are new host records. Several new geographic records are also presented, but generally these have a low value because of the migratory nature of the bird. Evidence suggests that some parasites, mostly flies and other arthropods but also nematodes and digeneans, produce detrimental behavioral or pathologic changes in the AWP. Some of the arthropods transmit microbial agents to the pelican. Two digeneans that have the AWP as a definitive host harm and even kill their catfish intermediate host, especially in aquaculture, and another causes abnormalities and mortality in amphibians. Some of the arthropods with low host-specificity can potentially transmit harmful microbial agents to humans and domestic animals. A few avian blood-flukes, intestinal flukes, and nematodes can potentially cause “swimmers itch,” gastroenteritis, and “anisakiasis,” respectively, in humans. Because of the life cycles of some helminths, presence of those worms can provide an indication as to the dietary items of a specific pelican individual, where the individual has been, and how long it has been present in an area. Feather mites, lice, and diplostomoid digeneans serve as good parasites to indicate phylogenetic relationships among different pelican species as well as relationships among the Pelecanidae and other families such as the Sulidae.

INTRODUCTION

The American white pelican (AWP) *Pelecanus erythrorhynchos* serves as a host for numerous parasites, several of them recently or not previously reported. It serves as an acceptable host for many parasites because it migrates over an extensive, defined, geographic range, all the while feeding on a large variety of prey items. Those prey species in turn serve as intermediate hosts for numerous parasites of the AWP, and many of those parasites as well as other symbionts without such complicated life cycles have co-evolved with the pelican. Consequently, knowledge about the symbionts, especially the truly parasitic fauna, provides knowledge about the dynamics of the bird host during certain seasons, in certain regions, or through time. Further, the AWP nests in groups, allowing for easy transference of certain parasites among flock cohorts.

Parasites of birds have been used before by several researchers as indicators of the bird's biology. For example, digeneans (Bartoli 1989) have indicated host diets, prey preference, habitat where prey was eaten, and host migration, even though digeneans and other parasites of fishes have been used more often than those of birds as well as used for additional categories of biological indications (e.g., Kabata 1963, Margolis 1963, MacKenzie 1983, Lester 1990, Overstreet 1993, 1997). These articles cite criteria one should meet, which vary considerably, depending on the type of question the biological indicator should answer. Analysis of the phylogeny of bird parasites as well as the co-evolution or host-switching with their hosts also have concerned lice (e.g., Page et al. 2004) and other parasites. That recent study and others involving

other host groups (e.g., Nadler and Hafner 1993) have strengthened our understanding of phylogeny by taking advantage of a variety of molecular approaches rather than performing strictly morphological analyses. Molecular techniques now provide additional useful tools to investigate a variety of parasitological problems.

This article intends to provide a preliminary foray into many of the AWP's symbionts. “Symbiont” is a general term used here to encompass organisms that cover a complete range of benefit and harm to a “host,” whether commonly referred to as a “parasite” or not (Overstreet 1978). They include organisms ranging from those with only a loose bond, such as a stable fly, to those “true” parasites like the digenean *Bolbophorus damnificus*, which is specific to pelicans and exhibits a complicated life cycle in which the intermediate hosts but not the pelican host are harmed. For purposes of this study, we usually refer to each of the symbionts, regardless of the degree of host-specificity, ability to harm a host, or other aspects of its association with the host, as a parasite. Some of these negatively influence the bird, pose a potential public health risk, or provide useful biological information about the pelican or its associates. The focus of this article is not meant to emphasize disease in the AWP resulting from parasites but rather to show how parasites may play an important role in understanding the biology of the bird.

MATERIALS AND METHODS

Data for this article have been gathered from prior collections made by the authors, specimens borrowed from museums, and the literature. Collections consist of parasites from a relatively small number of the AWP. Those

birds from the Mississippi-Louisiana region were shot or died during their migration in conjunction with the USDA/APHIS/ Wildlife Services, provided by the Wildlife Rehabilitation & Nature Preservation Society, or collected from aquaculture facilities. A single specimen was shot at the Summer Lake Wildlife Area, Oregon. We compare our data on the AWP with that collected or described from the brown pelican (*Pelecanus occidentalis*) in Mississippi or elsewhere. Parasites were removed from birds that were either freshly killed or birds that had been collected earlier and their alimentary tract frozen or fixed in alcohol or formalin. A few birds were examined thoroughly for parasites in general, and specific organs of others were examined for certain species of parasites. Parasites were prepared using a variety of methods common in our laboratory, including some described by Cable (1977), Crites and Overstreet (1991), and Overstreet and Curran (2005). In the most recent collections, we placed representative specimens in refrigerated 95% molecular grade ethanol or directly into a -70°C freezer for molecular studies.

RESULTS

Appendix 1 lists most of the parasites about which we have knowledge and provides the site in the bird, the geographic location of the infection, and principal references, when applicable. When we considered recorded parasite names to represent junior synonyms, we usually included the accepted names only. We also include in the table infections of parasites that we encountered for this study. The list, plus a single unidentified coccidian protozoan, contains 75 species, 9 of which are flies and 1 a flea with very little specificity toward the AWP but important to the bird's health. About three of the reports represent synonyms or misidentifications of binomial species already on the list. That leaves about 62 listed species, not counting the flies and flea, even though some unidentified species probably represent a complex of species. Of the 62, about 24 have not been reported from the brown pelican (e.g., Dyer et al. 2002). Many are confined to both pelican species (e.g., Dronen et al. 2003), but others infecting both pelicans also infect other birds. About 10, consisting of lice, mites, and helminths, infect the AWP only. Questionable identifications and an indication of whether infections also occur in the brown pelican are noted in Appendix 1. New geographical records are not individually marked. Appendix 1 also lists seven new records for the AWP based in some cases on careful examination of just a few specimens. These are marked with an asterisk. Even with the inclusion of this study, historically few specimens of the AWP from few localities have been thoroughly

examined for parasites. And from these few individuals, often the site of infection has not been recorded. As a result, we predict that many additional parasites infect the AWP. For example, the brown pelican hosts the eimerian coccidian protozoan *Eimeria pelecani*, but no protozoan other than a record of the coccidian *Sarcocystis* sp. in cysts in the pectoral muscle (Forrester and Spalding 2003) is known yet from the AWP.

DISCUSSION

Because some parasite identifications were based on single, few, or incomplete specimens and because some identifications or infections required annotation, we mention a few relevant points. For the cestodes, no scolex was found among our tapeworm material of *Paradilepis* cf. *caballeroi*, so the species could not be identified, but, based on the diagnosis by Forrester and Spalding (2002), we assume *P. caballeroi* is probably correct. There exists some debate about the status of species in *Paradilepis* and related genera. *Parvitaenia heardi* (= *Glossocercus caribaeensis* by Scholz et al. 2002a) was described from the great blue heron (*Ardea herodias*) in South Carolina by Schmidt and Courtney (1973), with the brown pelican listed as an accidental host. Rysavy and Macko (1971) reported *Parvitaenia eudocimi* (= *Cyclustera ibisae* by Scholz et al. 2002a) from the brown pelican in Cuba as well as from the white ibis (*Eudocimus albus*), for which it was named. They described *P. caballeroi* from the double-crested cormorant (*Phalacrocorax auritus*). Additional well-fixed material of dilepidids from the AWP should allow exact identifications and perhaps reveal the presence of several additional species. For example, Scholz et al. (2002b) found five different dilepidid species in "*Phalacrocorax olivaceus*" (= the neotropical cormorant, *P. brasilianus*) in Mexico.

For the digeneans, *Bolbophorus confusus* has been reported by several authors as one of a few diplostomoideans infecting the brown pelican and AWP. Overstreet et al. (2002) have shown that at least some of those records represent one and in some cases two related species of *Bolbophorus*. Whether *B. confusus*, which we consider to be a European species, exists in North America still has to be determined (Overstreet et al. 2002). One of the two species previously reported in part as *B. confusus* by at least some authors (e.g., reviews by Olson 1966, Overstreet et al. 2002, Appendix 1) was considered as *B. damnificus*, and it can be readily acquired by the AWP from feeding on infected channel catfish (*Ictalurus punctatus*) (Overstreet et al. 2002, Overstreet and Curran 2004). The other species is referred to as *Bolbophorus* sp. of

Overstreet et al. (2002). It has been demonstrated to differ from *B. damnificus* as shown by sequences of four different gene fragments by Overstreet et al. (2002) and corroborated by Levy et al. (2002) using one fragment. Levy et al. (2002) showed that it infected several fishes but not the catfish. Adults of both of the two species can occur in the same individual pelican host (Overstreet et al. 2002).

When we sequenced the ITS 1/2 for three preadult specimens from a single AWP in Oregon, the percentage value for DNA sequence similarity with *Bolbophorus* sp. of Overstreet et al. (2002) was 99% in contrast with 88% when compared with the same fragment of *B. damnificus*. The three specimens also had a similarity value of 99%, indicating the immature Oregon specimens were *Bolbophorus* sp. of Overstreet et al. (2002). Also, based on specimens of diplostomoideans we have seen from northern North America, we suspect that the report of *Diplostomum spathaceum* from the AWP in Manitoba by McLaughlin (1974) probably represents a species of *Bolbophorus*. He found only three helminths, and only *D. spathaceum* was a digenean. Since it appears superficially similar to the two species of *Bolbophorus* that are common in the AWP in Canada, we treat the report as a misidentification.

We found another diplostomoid reported as *Bursacetabulus pelecanus* in the AWP as well as in the brown pelican. Whether it is conspecific with *Bursatintinnabulus macrobursus*, which we also found in both local pelican species, is being treated by Charles Blend, Overstreet, and Curran (unpublished data).

Several host records for digeneans deserve comment. *Ribeiroia ondatrae* has been reported from various gulls and the muskrat. McNeil (1949) listed the AWP as a host in Washington, Forrester and Spalding (2002) reported it from Florida, and Dyer et al. (2002) reported it from the brown pelican in Puerto Rico. Originally, Price (1931) did not notice the esophageal diverticula and considered the species in a different genus, but Lumsden and Zischke (1963) confirmed their presence in the type material. The presence of this worm in pelicans is important because of the effect of the species on its amphibian intermediate hosts discussed below. Species of *Renicola* are difficult to differentiate, primarily because eggs obstruct the view of most of the organs. We appear to have two species. Specimens from Mississippi are similar to *Renicola thapari*, but our relatively young specimens appear different enough from the much larger and more fecund specimens described from the brown pelican in Panama (Caballero 1953) and reported later from that host in Florida and Louisiana by Courtney and Forrester (1974) to consider the identification tentative. A portion of a damaged speci-

men collected from Oregon seems to represent a different and possibly new species or one of the few renicolids that infect other white pelican species (e.g., Stunkard 1964). The identification of *Prosthogonimus ovatus* was based on a single specimen from the wash of the oviduct and a small portion of the cloaca, but it was not initially observed in the cloaca. It has a smaller body, suckers, and eggs than *Prosthogonimus folliculus* from the American bittern (*Botaurus lentiginosus*), and, based on reported North American species of the digenean, it seems most consistent with the description of *P. ovatus*, a species known from several birds, both small and large. Actually, we expect specimens that have been reported from different cosmopolitan hosts as *P. ovatus* to represent a complex of species. Our measurements of what we identified as *Austroilharzia variglandis* are slightly smaller than those reported by Stunkard and Hinchliffe (1952) and may represent an atypical infection. In any event, the eastern mudsnail (*Nassarius obsoletus*) (also known as the mud whelk, *Ilyanassa obsoleta*), intermediate snail host for *A. variglandis*, occurs along the northern Gulf of Mexico as well as along the eastern US seaboard, where infections in it have been investigated (Barber and Caira 1995). Gulls appear to be the primary avian host for the species. *Gigantobilharzia huttoni* (see Leigh 1957) and *Dendroilharzia pulverulenta* (see Forrester and Spalding 2003) infect the AWP in Florida and presumably elsewhere, and other blood flukes infect the brown pelican and other pelicans around the world (e.g., Yamaguti 1971).

Nematodes in the genus *Contraecacum* require a taxonomic revision. There have been six nominal species reported from the AWP, but, considering a synonym and misidentifications, we considered only four species acceptable; occasionally at least three species occur concurrently in an individual bird. The morphological features of the species do not fit all the descriptions corresponding to the names (e.g., Deardorff and Overstreet 1980). In any event, we have seen three species listed in Appendix 1 as concurrent in the AWP from Mississippi and Louisiana, with *C. multipapillatum* being the most common in those localities. As the populations of both the AWP and the brown pelican increase, the juvenile infections in the two local mullets (*Mugil cephalus* and *Mugil curema*, common second intermediate hosts) become more abundant, resulting in pelican infections, which in turn commonly reach over several hundred specimens in an individual bird. In Oregon, we have seen *C. microcephalum* as identified using the work of Barua et al. (1978), and only several specimens occurred in the bird. What we call *C. microcephalum* in North America may be a distinct species but closely related to the European form. *Contraecacum*

microcephalum has been reported throughout the world. Heavy infections by a species reported as *C. microcephalum* from Tanzania were held responsible for pelican mortality there (Nyange et al. 1983).

The external features of the clitellate glossiphoniid leech *Theromyzon* sp. from Oregon differed from those described for *T. rude*, but the species is clearly in the genus. Six young specimens were restricted to the breast, neck, and head area, and none was associated with the cloaca. Mark Siddall and Elizabeth Borda (American Museum of Natural History) are in the process of sequencing material to establish its identity. No leech has been reported previously from the AWP, but Rothschild and Clay (1957) mentioned that leeches occurred in the vent and gular pouch of pelicans.

Feather mites are treated below under phylogenetic relationships. We expect several more feather mites infest the AWP than have been reported. For example, nymphs of three species of hypoderatid mites in the subcutaneous tissues of the brown pelican were reported in Louisiana and Florida by Pence and Courtney (1973), and we have seen unidentified, presently unavailable for study, species in the subcutaneous fat around the trachea of the AWP. Adults of these mites inhabit the nests. Also, the trombiculid *Womersia strandtmanni* has been reported in the brown pelican by Vercammen-Grandjean and Kobeginova (1968). That chigger caused skin lesions in ducks (Clark and Stotts 1960). Based on knowledge of mite infestations in other pelican species, we suspect related or identical species occur on the AWP.

Bird health

Depending on what one wishes to consider a cause of disease, there could be several of the organisms listed in Appendix 1 that have a direct or indirect negative influence on the health of the AWP. The fleas, ticks, and flies all can pose a threat to the health of the bird, especially weakened young, captured, or disabled individuals. These have low host-specificity with the pelican; for example, there are eight species of flies listed and presumably many more exist. These arthropods have been observed on young individuals in "nesting areas" in large numbers, and often the birds in question died (Johnson 1976). Whether the young were unhealthy and attracted the flies or whether the flies caused the birds to become unhealthy is uncertain, but, in any event, the flies aided in the demise of many individuals. Johnson (1976) found the adult flies annoying young birds that hatched primarily late in the season in Chase Lake National Wildlife Refuge. The young birds were unable to avoid the flies by moving into open areas, and, once a few flies started feeding on a bird's flesh in the head or else-

where, many more became attracted to feed, and these laid eggs in the bird, resulting in even greater numbers.

The flies offer additional means of causing disease. Because they are not specific to the pelican, they often leave one individual or one host species and find another. Even though hippoboscids have little ability to fly and infest their hosts primarily through direct contact, most other flies like the blowflies and stable flies travel from host to host. Consequently, those that fly the farthest can more readily pick up a bacterial, viral, or some other infection from one wild or domestic host species and transmit it to another host such as the AWP or even a human.

The soft tick *Ornithodoros capensis* presents another problem. This common argasid has been held responsible for causing the parent birds to desert their nests, sometimes for two years. As indicated above, they are not specific to the AWP, but they infect several different aquatic birds. King et al. (1977a) found that three deserted brown pelican nests in Aransas National Wildlife Refuge, Texas, yielded 2,389 adult and nymphal specimens of the tick. This and perhaps another species (*Ornithodoros denmarki*) probably caused nest desertion by the brown pelican in Gulf of California nests. Scratching and preening behavior occurred from 32–68% of the morning and afternoon observation times in areas where desertion was greatest (King et al. 1977b). Death of the nestlings may result from transmission of a lethal Soldado-like arbovirus from the tick (Converse et al. 1975). Infestations also are known to reduce brood size in Texas (King et al. 1977a, 1977b). We think the actual importance of this tick to the AWP probably depends on air temperature. Infestations have been reported on the AWP in Texas (King et al. in Duffy 1983), where temperatures remain relatively high. We questioned various biologists such as Robert Johnson and Kory Richardson at Chase Lake National Wildlife Refuge, North Dakota, and Marty St Louis at the region in and near Summer Lake Wildlife Area, Oregon, and northern California, and they never recalled seeing any ticks on the birds or in their nests from these relatively cool nesting grounds.

Perhaps other agents also cause pelicans to desert their nests. Rothschild and Clay (1957) mentioned that entire colonies of pelicans in the southern seas have deserted their nests because of *Culex pipiens*, referred to as a "house-gnat" rather than a mosquito. The complex of mosquito species in the *C. pipiens*-group has been held responsible for extinction or shifting ranges of various bird populations because it transmitted both bird malaria and avian pox virus (e.g., Warner 1968). We do not include the non-specific mosquitos in Appendix 1.

Lice are much more specific to the pelican than are the flies; in fact some lice species apparently infect no other bird except the AWP. They feed on blood and can occur in the thousands on birds that cannot adequately preen themselves, such as weakened young, captured, and disabled individuals. When someone encounters a pelican with large numbers of lice, the person should assume that the individual bird is in poor health. On one specimen of a brown pelican from Mississippi in September 1993 with a distorted bill, we observed thousands of specimens of lice, primarily of *Pectinopygus occidentalis* but some of *Colpocephalum occidentalis*, on its head, back, and wing feathers. The same occurs with the counterparts *P. tordoffi* and *C. unciferum* on the AWP. In fact, we observed feather mites associated with the lice on the brown pelican. The "pouch louse," *Piagetiella peralis*, is a biting louse that cannot be controlled by normal preening because it occurs in the gular pouch. The healthy AWP usually keeps an infestation in check, but weakened individuals often exhibit hemorrhagic ulcerative stomatitis and inflammation of the mouth (Wobeser et al. 1974, personal observations). The effect may be serious, and infestations are readily transmitted to young during nesting when infested parents are feeding them. Not all individuals of *P. peralis* infest the inner surface of the gular pouch, where its large numbers can cover the entire surface along the lower mandible and on the roof of the mouth without producing severe lesions. On 250 examined young birds, Johnson (1976) found them in the pouch of all, and 53 of a subset of 90 had some at the base of the neck, bottom of the feet, and axil of the wings. He noted that such external infestations appeared to subside after the birds reached 2–3 weeks of age. Wobeser et al. (1974) reported a large number of immature lice over the entire body of a dying young juvenile. In the only examined adult from Oregon, we found, in addition to those in the pouch, numerous immature specimens tightly lodged along the shaft of the primary wing feathers and a few younger specimens among the breast feathers. None was associated with pathological alterations.

Whether helminth infections harm the AWP depends on factors such as the number of worms present, prior state of the bird's health, and bird age. Individuals of some members of the anisakid nematode genus *Contracaecum* often occur in the hundreds in the proventriculus and adjacent organs of the AWP. Oglesby (1960) estimated over 1,100 individuals from a single AWP that had died in Florida. These were tentatively identified as *C. micropapillatum*, a species that Deardorff and Overstreet (1980) found in low numbers concurrent with considerably larger numbers of *C. multipapillatum* and *Contracaecum rudolphii* in other specimens in Mississippi and Louisiana.

Adult and fourth stage individuals typically associate with an ulcer where they attach and perhaps feed on the host response tissue. The secretions and excretions by juvenile worms are probably more responsible for local inflammation and necrosis than those by adults (Liu and Edward 1971, Fagerholm et al. 1996). We have seen ulcers both with a well-delimited conspicuous fibrotic protective capsule, allowing the nematodes to feed on inflammatory cells without disturbing the adjacent stomach tissue, and without such encapsulation. When without the capsule, the lesion is typically associated with extensive inflammation. After the bird host feeds, the nematodes often detach from the ulcer and entwine among the prey material. When individuals of various species of *Contracaecum* were found present in large numbers, some observers (Owre 1962, Huizinga 1971, Fagerholm et al. 1996) suggested that they help macerate or digest the prey as an initial stage in the host's digestive process. *Contracaecum multipapillatum* and related species also have been suggested as being associated with mortality or poor health of the bird host. Morbidity of hosts of all ages can be suspected when at least some individuals of a relatively large worm burden penetrate through the mucosal layer, when infections have an associated secondary microbial infection, or when an individual is starved (e.g., Oglesby 1960, Owre 1962, Fagerholm et al. 1996). Dyer et al. (2002) also suggested that the same species may have contributed to the emaciation and death of brown pelicans in Puerto Rico. Grimes et al. (1989), who tested the effects of four anthelmintics on *Contracaecum* spp. and two digenean species in the brown pelican, mentioned unpublished data by Courtney, who demonstrated that nestling pelicans with 95% of the nematodes removed by treatment showed higher weight gains than untreated controls.

Digeneans can also harm the AWP. As with some of the other agents indicated above, the pathological effect often depends on the number of individuals and other factors such as a secondary bacterial infection. *Phagicola longus*, a small species, probably affects the AWP that nests along the coast because a marine snail and mullets act as intermediate hosts. For example, nestling brown pelicans 4–5 weeks old from Louisiana contained over 18,000 specimens of *P. longus* along the small intestine and ceca, many in the mucosa and lamina propria. *Mesostephanus appendiculatoides*, present in lower numbers (e.g., averaging 1,112 specimens per bird from the Floridian Gulf of Mexico coast) attached to the villar tips and occasionally penetrated the epithelium (Humphrey et al. 1978, Greve et al. 1987). It was acquired from coastal silversides as well as mullets. Both digeneans, also occurring in the AWP, distorted host tissues and produced an inflammatory response

but did not kill birds in captivity. As few as 15 specimens of the larger *Ribeiroia ondatrae* deep in the proventricular mucosa produced necrosis, possibly contributing to mortality of the brown pelican (Dyer et al. 2002). According to Rebecca Cole (personal communication, National Wildlife Health Center, Madison, Wisconsin), a heavy infection of *Pholeter anterouterus* along the intestine of an AWP in Florida killed the bird, possibly in conjunction with an acid fast bacterial infection.

The diet of fish allows the potential for harmful effects in addition to helminth infections. Because fish bio-accumulate various pesticides and other toxic agents, pelicans and other piscivorous birds can further accumulate such compounds (e.g., Forrester and Spalding 2003). Well-documented cases of the brown pelican with bioaccumulation of high levels of DDT and other pesticides in the late 1950's and early 1960's and then other pesticides in 1975 resulted in thin eggs and loss of fledged offspring. The reduced production of young decimated the brown pelican population in the northern Gulf of Mexico and other areas, and a return of successful breeding colonies took several years (Johnsgard 1993). A condition of far less concern involves older individuals feeding on physically dangerous items. Lesions commonly observed by us in the stomach suggest that punctures by spines such as those on pectoral and dorsal fins of catfish and other prey can develop secondary infections and perhaps produce death when the prey is not eaten head-first. Catfish spines killed two adult AWP (Forrester and Spalding 2003); one lacerated the jugular vein and the other perforated the esophagus and stomach. Johnson (1976) observed two young birds, one of which died, with penetrating fish vertebrae lodged in their throat. Related to this kind of damage was a case of potential death resulting when an AWP engulfed a wooden-handled ice-pick (Mattis and Deardorff 1988). The bird with the pick had difficulty standing, remained in a squatting position with a contracted neck, could not fly, and could no longer feed or be force-fed. Once the bird was x-rayed and the condition diagnosed, the pick was shown to have entered down the esophagus handle first and perforated that organ, so that the pick could be removed from its lodgment and the bird saved only by human intervention.

Health of intermediate hosts

Not only can the AWP be harmed by a few species of helminth parasites, but a few of the helminths that have little effect on the pelican can be transmitted by the pelican and have a drastic influence on the intermediate host population. Good examples include two diplostomatid digeneans that infect catfish and a cathaemasiid digenean that infects amphibians. These diplostomatids, *Bolbophorus*

damnificus and *Bursacetabulus pelecenus*, both can produce mass mortalities of the channel catfish, at least in aquaculture conditions (Overstreet et al. 2002, Overstreet and Curran 2004). The problem with *B. damnificus* is more confusing than originally presumed by fish farmers and managers because more than one species of *Bolbophorus* infects the AWP (Overstreet et al. 2002), with a single individual bird capable of harboring at least two of those species. Only one of these is known to infect the catfish. For the two indicated species that infect the catfish, their eggs are released with the pelican's feces into the aquaculture ponds. The miracidia (infective larvae) of both infect the appropriate snail host, and, after development of at least two asexual stages of the digeneans and ultimate production of large numbers of infective cercariae, individuals of the cercaria of each species are shed in large pulses available to infect the catfish. Those for *B. damnificus* enter the fish and finally lodge and encyst, typically in the muscle adjacent to the dermis in the caudal region, and those of *B. pelecenus* end up unencysted in the vitreous humor of the eye. Infection by *B. damnificus* also results in pathological alterations in the kidneys (Overstreet and Curran 2004). The snails and up to millions of associated cercariae occur along the shallow sides of fish-ponds where young catfish occur and receive massive infections, often resulting in death (e.g., Terhune et al. 2002). We have exposed catfish to the cercariae of *B. damnificus* in the laboratory and produced death of the fish after periods ranging from minutes to days, depending on the dose of cercariae (Overstreet et al. 2002, Overstreet and Curran 2004). How many cercariae of *B. pelecenus* are necessary to harm the catfish was not established, but infections of another diplostome, *Austrodiplostomum compactum*, which matures in various cormorant species, infected the vitreous humor as well as the brain and spinal cord and also killed the catfish. For it to kill the host necessitated a larger number of the penetrating cercaria than did *B. damnificus* in short-term laboratory infections. Thousands of very young worms could infect the nerve tissue of the fish (Overstreet and Curran 2004). Consequently, the AWP does not necessarily serve as the only avian source of digeneans that can cause catfish mortalities and it is not the only scourge of the fish farmers wanting to rid their ponds of pelicans. The AWP and different cormorants eat catfish from the ponds, whether the catfish are infected or not. The example involving harm to amphibians concerns the cathamaesiid digenean *Ribeiroia ondatrae*. The metacercaria of this species produced limb malformations in a wide range of amphibians (frogs, toads, newts, and salamander) in wild and experimental hosts (Johnson et al. 2002), with survivorship declining significantly with

increasing cercarial exposure (Johnson et al. 2001). Unlike the examples of diplostomids where the pelicans and catfish are the only known hosts, *R. ondatrae* infects a few different vertebrate definitive hosts in addition to pelicans as well as numerous amphibian second intermediate hosts and several snail species of the planorbid genus *Planorbella* as first intermediate hosts.

A similar problem involving harm to the intermediate host concerned recreational fishermen and those interested in the AWP from the late 1920s until many years after in Yellowstone Lake, Wyoming. The pelican colony on Molly Island had to be protected because the birds there transmitted the tapeworm *Diphyllobothrium cordiceps* to the local trouts. When Behle (1958) wrote on the AWP, he indicted that the Park Service officials then felt that the value of the birds offset the loss of available fish.

Health of humans and domestic animals

As indicated above, some of the flies can transmit microbial infections to pelicans and other hosts, including humans. For example, the blood-feeding stable fly *Stomoxys calcitrans*, as summarized by Roberts and Janovy (2000), can transmit the flagellates *Trypanosoma evansi* and members of the *Trypanosoma brucei*-complex, the agents of surra and sleeping sickness in large mammals, as well as epidemic relapsing fever, anthrax, brucellosis, swine erysipelas, equine swamp fever, African horse sickness, and fowl pox. This species also serves as the intermediate host for the nematode *Habronema microstoma* in horses. These infections are in addition to the biting that causes severe discomfort in humans and death in livestock. Some of the other flies can also transmit various agents. The possibility of the lice transmitting an agent has not been investigated, but that by pelican-ticks has been studied minimally (Forrester and Spalding 2003). One can say in general that transmission of numerous avian viruses, many of which are transmitted by arthropods, can have a serious negative influence on domesticated and wild birds and mammals as well as on humans (Perdue and Seal 2000).

The argasid soft tick *Ornithodoros amblyus*, which acquires short blood meals off the brown pelican and other seabirds in nesting islands off Peru, has been associated with the birds deserting their eggs and young. It possibly transmits infectious agents to the birds. At least two arboviruses, "Huacho" and Salinas," are transmitted by the tick. Although the effect on the birds was not established, Duffy (1983) reported that humans suffered swelling, itching, occasional gangrene, and even death following multiple tick bites.

Helminths also can be spread by the AWP to humans. For example, when the bird infected by the schistosome *Austrobilharzia variglandis* defecates in marine waters containing the eastern mudsnail (*Nassarius obsoletus*), the snail can get infected by the miracidia (larval stage hatched from the worm's egg) and this larva undergoes asexual reproduction, ultimately producing many thousands of cercariae. The cercaria is the invasive stage shed from the snail that infects the AWP or a variety of gulls and shore birds (e.g., Barber and Caira 1995). If it invades a human rather than the bird, it does not develop, but rather it establishes a host sensitivity response such that future invasions result in a hypersensitivity reaction in the skin of one who inhabits water containing the cercaria. As the host's defense responses react against the challenging doses of the cercaria, allergins are released from the cercaria that cause an inflammation. This reaction, called "swimmer's itch" or "clam digger's itch," is painful enough to keep people from entering beaches and other bodies of water that contain infected snails; and, consequently, public swimming areas often are closed, producing a local economic hardship. Patients are seldom severely harmed, but the hypersensitivity reaction keeps most from revisiting the location. Unlike the two-host schistosome life cycle, most helminths utilize a series of at least two intermediate hosts plus the definitive host. For example (e.g., Huizinga 1967), when a bird with the nematode *C. multipapillatum* defecates in near shore or freshwater habitats, some cyclopoid and presumably other copepod species feed on the released larval nematode, supporting development to a stage (third stage) or condition (exsheathed second stage) infective to a fish intermediate host. The larva, or more appropriately the "juvenile," can develop only in certain fish species. When other fish, or in some cases invertebrates, are eaten by animals other than the AWP or other avian definitive hosts, the worm migrates to the body cavity, becomes encapsulated, and remains infective to a pelican that feeds on the animal. Small fish intermediate hosts can be killed by the worm. Our original research based on non-human animals suggested that if humans ate this fish (e.g., primarily the striped mullet but also the red drum, *Sciaenops ocellata*, and other fishes), the worm would be digested (Deardorff and Overstreet 1980). However, later research involving RMO (Vidal-Martínez et al. 1994) showed that in some cases, presumably involving a warm period of acclimation, the worm could produce "anisakiasis." The term "anisakiasis" defines a disease in warm blooded mammals including humans caused by various ascaridoid species in the family Anisakidae and not just those in the genus *Anisakis*. Because of the recent increase in brown and white pelican infections following reduced

levels of DDT and related compounds, the potential risk is increasing. During that same period of depletion and recovery, the striped mullet in the northern Gulf of Mexico was overfished for the Japanese caviar industry and other needs. The reduced numbers of both avian and fish hosts subsequently reduced infections of *C. multipapillatum* in mullets, pelicans, and cormorants, but with increases in those hosts, heavy infections are recently beginning to return (unpublished observations).

A public health risk also occurs for those eating inadequately cooked American species of mullet infected with *Phagicola longus*, a digenean infecting a snail that feeds on eggs shed with host feces by the AWP or a few other birds; the fish becomes infected from the cercaria shed from the snail (Overstreet 1978, Deardorff and Overstreet 1991). Unlike the nematode that infects warm-blooded hosts as a juvenile, *P. longus* matures in the warm-blooded host, often causing grossly appearing gastroenteritis in herons and raccoon hosts (Overstreet 1978, Richard Heard and Overstreet, personal observations).

Indicators of biological activities

As described in abbreviated detail above, helminths undergo a complex life cycle involving two or more different hosts. The AWP is the final, or definitive, host for those listed in Appendix 1. The cycle in different helminth groups differs, and that for each species differs from all others in some ways, usually by the specific hosts involved. Knowledge of these life cycles and life history patterns can provide important biological information on feeding habits and migratory patterns of the host individuals. For example, *Phagicola longus*, *Mesostephanus appendiculatoides*, and *Contracaecum multipapillatum* all infect the striped mullet, *Mugil cephalus*, as the second intermediate host. When these parasites are observed in a pelican, one knows that the pelican has been feeding on mullet along the coast. In contrast, *B. damnificus* and *B. pelecanus* infect the channel catfish in fresh water, usually far from the coast, and the presence of one or the combination of both in the pelican indicates that it was feeding on the catfish. Of course there exists a variety of other helminths from both habitats, but most do not occur in large numbers. By looking at the relative numbers of these freshwater and coastal parasites as well as the presence or absence of each species, one can get a good indication of where the bird has been and how long ago the bird was feeding on what and in what habitat. Since there is a loss of individuals with time, there is a greater likelihood of a recent infection if there is a heavy infection of a species that can occur in large numbers (e.g., those species indicted above). Moreover, this indication of a recent infection

can be strengthened when some individuals of certain species possess few or no eggs, indicative of recent acquisitions. If specimens of parasites from coastal and inland habitats are both present, evaluation of all these features should provide the necessary feeding and migratory information. Humphrey et al. (1978) treated the differences in community structure of the above helminths in the brown pelican from the east and west coasts of Florida and from Louisiana. They pointed out the eventual decline in *P. longus* in adult brown pelicans could result from a possible immune response established during a tissue dwelling stage occurring in the fledgling pelicans. They also speculated on the community structure being influenced by a change from mullet as a dietary item when young birds no longer depended on food from their parents. Kinsella et al. (2004), who collected helminths from the AWP in Florida, noted that most of the helminths from their 29 birds had been acquired in the marine habitat, even though many of the birds were collected inland. The community of helminths in the AWP would probably provide a good model to demonstrate an interactive community (Holmes and Price 1986), especially since the parasites have such a diverse array of effects on the bird populations.

Even though few birds were examined critically by either Dronen et al. (2003) or us and little can be determined from incomplete data on prevalence or intensity, we can surmise that the endohelminths from the AWP from the Mississippi-Louisiana region had a greater richness than in the counterparts from the Galveston Bay, Texas, area. Ten endohelminths reported from six AWP from Texas compared with 19, or at least 20 considering synonyms, from Mississippi/Louisiana and with 33 from Florida, where the sample size was much larger (Forrester and Spalding 2002, Kinsella et al. 2004). In all cases, the worms were derived from a combination of freshwater and marine intermediate hosts. In Texas, the brown pelican, a bird that has a more restricted home range than the AWP, had 23 species, a number comparable with those we observed in the AWP but still considerably less than the number of endohelminths that occur in the brown pelican from Florida and presumably Mississippi.

We are also interested in knowing what parasites are residents in intermediate hosts in specific habitats. Specific intermediate hosts and cycles for many helminths have not been discovered. The presence of preadult specimens of *Bolbophorus* sp. of Overstreet et al. 2002 in an AWP on the Summer Lake Wildlife Area, Oregon, suggests that the bird acquired the infection in or near the Area.

Indicators of phylogenetic relationships

Different tools have been used to discern phylogenetic relationships within and among avian families, including pelicans. For example, Cracraft (1985) presented a closer morphological relationship, based on an extensive cladistical analysis, between Pelecanidae and Sulidae (gannets and boobies) than between pelecanids and the cormorants or anhingas, once thought to be more closely related to the pelecanids than any other birds. But Warheit et al. (1989), using just the number of ossicles per ring in the sclera of the eye's corneal hemisphere, separated the pelicans farther from the sulids than the other bird groups. Then, first using DNA-DNA hybridization (e.g., Sibley et al. 1988) and later using DNA sequences of mitochondrial 12S and 16S rRNA genes (1.7 kb) (Hedges and Sibley 1994), the biologists also separated those groups similarly to the arrangement of Warheit et al. (1989). Siegel-Causey (1997) concluded, as did Sibley and colleagues, that the originally designated Pelecaniformes was paraphyletic (having more than one unrelated ancestor), with none of the several studies supporting a monophyletic (single original ancestor) origin of the order. Only the relationship between the pelicans and shoebills appears consistent with all the molecular data. The author also considered the molecular studies in an elementary stage, with answers requiring a re-examination of traditional morphological characters. Nevertheless, preliminary parasitic data on infections with closely related species of feather mites, diplostome digeneans (species of *Bursacetabulus* and *Bursatintinnabulus*), and cyathocotylid digeneans (species of *Mesostephanus*) seem to support a close relationship of the pelicans with the sulids.

Appendix 1 indicates that many of the AWP parasites also occur in or on the brown pelican. There are a few groups of ectoparasites such as feather mites and lice and endoparasites such as diplostome digeneans and tetrabothriid cestodes that contain counterparts that differ between the two North American pelicans. These and related parasites allow us a better insight into the phylogenetic relationship between the two pelican species as well as among all pelicans and among the Pelecanidae and other bird groups.

Feather mites have been demonstrated to be good parasitic tools to indicate relationships within and among bird groups (e.g., Mironov 1999). For example, members of the genus *Scutomegnina* (Avenzoariidae) on the Pelecanidae and Sulidae show a closer relationship among each other than those from birds of either family show to the mites on cormorants and anhingas (Mironov 2000). Moreover, Mironov (personal communication, Zoological Institute, Russian Academy of Sciences, St. Petersburg, Russia) con-

siders specific mites from the AWP in general more similar to those on other white pelicans than to those on the local North American brown pelican. For example, *Scutomegninia gaudi*, originally described from *Pelecanus onocrotalus*, also occurs on the AWP, but *Scutomegninia remipes* occurs on all the "subspecies" of the brown pelican (Mironov 2000), and *Megaloptes major* occurs on the brown pelican (Mironov and Pérez 2000). We expect *Megaloptes triphyllurus* will be found on the AWP, since it occurs on other white pelican species. *Alloptellus pelecenus*, already known from *Pelecanus onocrotalus*, *P. crispus*, and *P. rufescens* (see Peterson and Atyeo 1972), probably also occurs on the AWP. We predict a species of *Plicatallotes* to be found on the brown pelican that is different from *P. peleceni*. Presumably, several more mite species will be discovered on the AWP. Because of the large number of named and presumably unnamed feather mites showing various degrees of host-specificity in members of Pelicaniformes, this group of parasites seems the perfect group with which to assess phylogenetic relationships among the birds.

Since feather lice—like feather mites—are different among brown, white, and other pelicans that have been studied, we predict this group will also provide a powerful insight into the phylogeny of members of the genus *Pelecanus*. For example, in North American hosts, *Colpocephalum unciferum*, *Pectinopygus tordoffi*, and *Piagetiella peralis* infest the AWP in contrast with *Colpocephalum occidentalis*, *Pectinopygus occidentalis*, and *Piagetiella busaepelcani*, which occur on the brown pelican. Initially (Kellogg 1896), *C. unciferum* was thought to infest both pelicans, but it was later shown to be different from the material on the brown pelican. Additional related species infest other pelican species and other related species. A cladistical analysis of the species should reflect phylogenetic relationships among all pelecaniforms, including the ancestral association among the different pelicans. Of the Pelecaniformes, pelicans and frigate birds are infested by members of *Colpocephalum*, but birds in several other orders are also infested (Emerson 1972). Members of *Pectinopygus* infest some birds in every pelecaniform family except Phaethontidae (tropicbirds), with several species on pelicans, boobies, and gannets as well as frigate birds, cormorants, and anhingas. Members of *Piagetiella* infest only pelicans and cormorants (Price 1970).

Members of the diplostomoid digenean genus *Bursacetabulus* are known from pelicans and a gannet only. *Bursacetabulus pelecenus* infects the brown pelican (Dronen et al. 1999) and the AWP, and *Bursacetabulus morus* infects the northern gannet (*Morus bassanus*).

Additionally, two other nominal species in the genus *Bursatintinnabulus* are reported from the same hosts (Tehrany et al. 1999), although we question the taxonomic status of those latter worms.

Six genera of cestodes in the family Tetrabothriidae have shown the genus *Tetrabothrius* to be pleisiomorphic (=ancestral) (Hoberg 1989, Hoberg et al. 1997). Members of the genus suggest an archaic association of the species among the Pelecaniformes, Procellariiformes, and Sphenisciformes as well as with marine mammals. Evaluating species infecting Phalacrocoracidae seems to illuminate the relationships among the cormorants (Hoberg 1987).

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APPENDIX 1

Partial list of metazoan parasites from the American white pelican.

Parasite	Site	Location ¹	ID ²	Y/N ³	Principal references
Cestoda (Tapeworms)					
<i>Cyclusteria ibisae</i>	intestine	FL, TX	ID?	Y	Forrester and Spalding 2003, Dronen et al. 2003, Kinsella et al. 2004
<i>Diphyllobothrium cordiceps</i>	intestine	WY, MT		N	Leidy 1872, Woodbury 1932, Scott 1955, Post 1971
<i>Hymenolepis</i> sp.	intestine	WY, WA	ID?		Woodbury 1937, McNeil 1949
<i>Paradilepis longivaginosus</i>	upper small intestine	WY		N	Mayhew 1925, Woodbury 1937
		MB			McLaughlin 1974
		SD			Hughhins 1956
		CA-NV			Matthias 1963
		FL			Forrester and Spalding 2003, Kinsella et al. 2004
		MS, LA			Present study
<i>Paradilepis caballeroi</i>	intestine	FL	ID?	N	Forrester and Spalding 2003
		MS, LA	ID?		Present study
<i>Spirometra mansonoides</i>	subcutaneous	FL		N	Forrester and Spalding 2003
spargana metacestode					
<i>Tetrabothrius</i> sp.	small intestine	TX		Y	Dronen et al. 2003
Digenea (Flukes)					
<i>Ascocotyle gemina</i>	intestine, ceca	FL		N	Forrester and Spalding 2003, Kinsella et al. 2004
<i>Ascocotyle leighi</i>	ceca, large intestine	FL		N	Forrester and Spalding 2003, Kinsella et al. 2004
* <i>Austrobilharzia variglandis</i>	intestine, mesentery vessels	MS		N	Present study
<i>Bolbophorus confusus</i>	intestine	USA,	ID?	Y	Hughhins 1956, Fox and Olson 1965,
(at least = <i>B. damnificus</i> in some references)		Canada			Dubois 1970, Dronen et al. 2003, Forrester and Spalding 2003
<i>Bolbophorus damnificus</i>	intestine	MS, LA		Y	Overstreet et al. 2002
<i>Bolbophorus</i> sp. of Overstreet et al. 2002	intestine	MS, LA		N	Overstreet et al. 2002
		OR			Present study
<i>Bursacetabulus pelecanus</i>	intestine	FL		Y	Forrester and Spalding 2003, Kinsella et al. 2004
		MS, LA			Blend, Curran and Overstreet, personal observations
		TX			Dronen et al. 2003
<i>Bursatintinnabulus macrobursus</i>	intestine	TX, MS, LA		Y	Blend, Curran and Overstreet, personal observations
		FL			Forrester and Spalding 2003

APPENDIX 1 (continued)

Parasite	Site	Location¹	ID²	Y/N³	Principal references
Digenea (Flukes) (continued)					
<i>Bursatintinnabulus bassanus</i>	ceca, intestine	TX	ID?	Y	Dronen et al. 2003
		FL	ID?		Kinsella et al. 2004
<i>Clinostomum attenuatum</i>	mouth cavity, trachea	FL			Kinsella et al. 2004
* <i>Clinostomum marginatum</i>	esophagus	LA		N	Present study
		FL, TX			Present study [▼]
<i>Clinostomum complanatum</i>	mouth cavity, trachea	FL	ID?	N	Forrester and Spalding 2003, Kinsella et al. 2004
<i>Dendritobilharzia pulverulenta</i>	blood vessels, heart	FL		N	Forrester and Spalding 2003, Kinsella et al. 2004
<i>Diplostomum spathaceum</i>	intestine	MB	ID?	N	McLaughlin 1974
<i>Echinochasmus dietzevi</i>	intestine, stomach	FL		Y	Forrester and Spalding 2003, Kinsella et al. 2004
		MS, LA			Present study
<i>Gigantobilarzia huttoni</i>	veins of intestinal wall	FL		Y	Leigh 1957
<i>Galactosomum</i> sp.	small intestine	FL			Kinsella et al. 2004
<i>Gigantobilarzia</i> sp.	blood vessels	FL			Forrester and Spalding 2003
<i>Ignavia renalis</i>	intestine	London Zoo		N	Yeh 1954
<i>Mesostephanus appendiculatoides</i>	intestine, cloaca,	FL		Y	Forrester and Spalding 2003
	ceca	MS, LA			Present study
<i>Mesostephanus microbursa</i>	small intestine	FL		Y	Forrester and Spalding 2003, Kinsella et al. 2004
<i>Mesostephanus</i> sp.	intestine, ceca	TX	ID?		Dronen et al. 2003
<i>Mesorchis denticulatus</i>	intestine, cloaca, ceca	MS, LA		Y	Kinsella et al. 2004, present study
<i>Microparyphium facetum</i>	cloaca	FL			Kinsella et al. 2004
<i>Phagicola longus</i>	small intestine	WA		Y	McNeil 1949
	cloaca, intestines	TX			Dronen et al. 2003
	small intestine, ceca	FL			Kinsella et al. 2004
		MS, LA			Present study
<i>Phagicola nana</i>	small intestine	FL		Y	Forrester and Spalding 2003, Kinsella et al. 2004
<i>Pholeter anterouterus</i>	nodules in wall of small intestine	FL		Y	Pearson and Courtney 1977, Kinsella et al. 2004
<i>Posthodiplostomum minimum</i>	small intestine	FL		N	Forrester and Spalding 2003, Kinsella et al. 2004
* <i>Prosthogonimus ovatus</i>	oviduct	OR	ID?	N	Present study
<i>Renicola thapari</i>	kidneys	FL		Y	Forrester and Spalding 2003, Kinsella et al. 2004
		MS			Present study
		MN, CA			Present study [▼]

APPENDIX 1 (continued)

Parasite	Site	Location¹	ID²	Y/N³	Principal references
Digenea (Flukes) (continued)					
<i>Renicola</i> sp.	kidneys	OR FL		N	Present study Forrester and Spalding 2003, Kinsella et al. 2004
<i>Ribeiroia ondatrae</i>	esophagus, proventriculus, intestine	WA FL MS, LA		N	McNeil 1949 Forrester and Spalding 2003, Kinsella et al. 2004 Present study
Acanthocephala (Spiny-headed Worms)					
<i>Polymorphus brevis</i> , immature (not presently available)	small intestine intestine	FL MS		N ?	Kinsella et al. 2004 Present study
Nematoda (Roundworms)					
<i>Capillaria mergi</i>	intestines, ceca	FL		Y?	Forrester and Spalding 2003, Kinsella et al. 2004
<i>Capillaria</i> sp.	intestines, ceca	FL TX		Y?	Forrester and Spalding 2003 Dronen et al. 2003
<i>Contracaecum multipapillatum</i>	proventriculus, esophagus	MS, LA FL		Y	Deardorff and Overstreet 1980, present study Kinsella et al. 2004
<i>Contracaecum rudolphii</i> (as <i>C. spiculigerum</i>)	proventriculus, esophagus	WY, SD MS, LA FL		Y	Woodbury 1937, Huggins 1956 Deardorff and Overstreet 1980 Forrester and Spalding 2003, Kinsella <i>et al.</i> 2004
<i>Contracaecum micropapillatum</i>	proventriculus	FL, MS, LA		Y	Oglesby 1960, Deardorff and Overstreet 1980, Kinsella et al. 2004, present study
<i>Contracaecum microcephalum</i>	proventriculus, esophagus	TX OR	ID?	Y	McDaniel and Patterson 1966, Present study
<i>Contracaecum bancrofti</i>	proventriculus	TX Mexico	ID? ID?		McDaniel and Patterson 1966 Yamaguti 1961
<i>Contracaecum</i> spp.	esophagus, intestine stomach	FL, TX		Y?	Forrester and Spalding 2003, Dronen et al. 2003
* <i>Cyathostoma phenisci</i>	trachea, lung	MS		Y	Present study

APPENDIX 1 (continued)

Parasite	Site	Location¹	ID²	Y/N³	Principal references
<i>Eustrongylides ignotus</i> , juvenile	proventriculus	FL		Y	Forrester and Spalding 2003, Kinsella et al. 2004
<i>Gnathostoma spinigerum</i>		Mexico			Camacho et al. 1998
<i>Microtetrameres pelecani</i>	proventriculus	FL		N	Forrester and Spalding 2003, Kinsella et al. 2004
<i>Paracuaria adunca</i>	proventriculus	FL			Kinsella et al. 2004
<i>Strongyloides</i> sp.	intestine	FL		N	Forrester and Spalding 2003
<i>Tetrameres</i> sp.	proventriculus	FL		Y?	Forrester and Spalding 2003
Hirudinea (Leeches)					
* <i>Theromyzon</i> sp.	feathers from breast to head	OR		N	Present study
Phthiraptera (Chewing Lice)					
<i>Colpocephalum unciferum</i>	feathers	SD, Canada, KS BC, MB, SK FL MS, LA, OR		N	Kellogg 1896, Huggins 1956, Price 1967 Price and Beer 1967 Forrester et al. 1995 Present study
<i>Pectinopygus tordoffi</i>	feathers	KS, UT, SD, W DC, MS, TX, CA, OR Mexico FL MS, LA, OR		N	Elbel and Emerson 1956 Carriker 1956 Forrester et al. 1995 Present study
<i>Piagetiella peralis</i>	gular pouches and body	FL WAMc SD SK MS, LA, OR		N	Leidy 1878, Forrester et al. 1995 Neil 1949 Huggins 1956 Wobeser et al. 1974 Present study
Siphonaptera (Fleas)					
<i>Ceratophyllus niger</i>	body and nest	BC		Y	Holland 1985
Diptera (Flies)					

APPENDIX 1 (continued)

Parasite	Site	Location¹	ID²	Y/N³	Principal references
Hippoboscidae (Louse Flies)					
<i>Icosta albipennis</i>	body	USA			Maa 1969 (listed as probably accidental, pelican species not identified)
<i>Olfersia sorida</i>	body	USA?		?	Maa 1969
Calliphoridae (Blowflies)					
<i>Bufo lucilia silvarum</i>	body	ND			Johnson 1976
<i>Lucilia illustris</i>	body	ND			Johnson 1976
<i>Phaenicia sericata</i>	body	ND			Johnson 1976
<i>Phormia regina</i>	body	ND			Johnson 1976
Muscidae (House Flies, Stable Flies)					
<i>Graphomya americana</i>	body	ND			Johnson 1976
<i>Musca autumnalis</i>	body	ND			Johnson 1976
<i>Stomoxys calcitrans</i>	body	ND			Johnson 1976
(Class) Arachnida					
Argasidae (Soft Ticks)					
<i>Ornithodoros capensis</i>	pouch, body, and nest	TX		Y	King et al. in Duffy 1983
Hypoderatidae (Nest Mites)					
<i>Phalacrodectes</i> sp.?	subcutaneous, trachea and neck	MS	ID?	Y	Present study (specimens not available)
Analgoidea (Feather Mites)					
<i>Metingrassia pelecani</i>	feathers	Canada			Mironov and Galloway, 2002
* <i>Plicatalloptes pelecani</i> (as <i>Alloptes</i> sp.?)	feathers	Canada, MS FL		N	Mironov, personal communication Forrester and Spalding 2003
* <i>Scutomegninia gaudi</i> (as <i>Scutomegninia</i> sp.?)	feathers	Canada, MS FL		N	Mironov, personal communication Forrester and Spalding 2003

¹ = Abbreviations refer to states in the USA and provinces in Canada.

²ID? = Questionable identification, see text for explanation.

³Y/N = Has (Y) or has not (N) also been reported from the brown pelican.

* = New host record.

▼ = Based on specimens loaned to us by the National Wildlife Health Research, Madison, Wisconsin.