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Parasite Pathoecology of Chacoan Great Houses: The Healthiest and Wormiest Ancestral Puebloans

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Archaeoparasitology and Pathoecology in the Southwest

Two fields of paleopathological investigation originated in the Southwest. Archaeoparasitology is the study ancient parasite infection (Reinhard 1990). It includes comparisons between time periods of single societies as well as the comparison of parasitism between different, contemporaneous cultures. For example, Fry (1980) compared Fremont and Anasazi parasitism. Fry (1984) compared Archaic hunter-gatherer parasitism and Ancestral Pueblo parasitism. All of these studies fall into the definition of Archaeoparasitology.

Pathoecology is the reconstruction of the relationships between behavior, environment, and disease organisms in the development of ill-health (Martinson et al. 2003; Reinhard and Buikstra 2003, Reinhard et al. 2003, Santoro et al. 2003). This field developed from the need for fine-grained analysis of prehistoric ecological and behavioral conditions to assess the factors that affected disease. Pathoecological interpretation depends on archaeological information regarding parasitism, community size, trade patterns, water sources, subsistence practices, environment, medicinal use, and many other topics. Although the term is new, pathoecology has really developed over several decades. I view El-Najjar et al.'s (1976) study of Ancestral Pueblo anemia as the first pathoecology study. I believe that Stodder and Martin's (1992) multifactorial perspective on Ancestral Pueblo disease is the most advanced example of pathoecology. Reinhard's 1996 study of the factors that affected parasitism at Antelope House and Salmon Ruin is another application of pathoecology.

Ancestral Pueblo communities have long been the focus of archaeoparasitology. Samuels (1965) developed the methods for helminth (parasitic worm) egg recovery with

coprolites from Mesa Verde. Subsequently, Stiger (1977) provided the first intersite comparison analysis on Mesa Verde. Fry and his colleagues conducted the first regional comparisons of parasitism, focusing on Canyon de Chelly and Glen Canyon (Fry 1977, Fry and Hall 1974, 1986). Fry (1977) also presented the first cross cultural analysis between Archaic, Ancestral Pueblo and Fremont sites. He also pioneered the comparison of parasitism between subsistence strategies. Building on this previous work, Reinhard (1985 a,b,c, 1990; Reinhard et al. 1986) analyzed the diversity of helminths that parasitized Ancestral Pueblo peoples. By 1985, archaeoparsitologists identified eight species of helminth that infected Ancestral Puebloans (Figure 1).

Aidan Cockburn's insight into the origins of disease influenced the development of pathoecology in the archaeoparasitology of Ancestral Pueblo sites. Cockburn (1967; 1971) argued that the evolution of infectious diseases followed human evolution and the development of human cultures. Inspired by Cockburn, Reinhard (1985a) compared the parasitic state of Colorado Plateau Archaic peoples to Ancestral Puebloan sites. He verified Cockburn's hypothesis that occasional infections in hunter-gatherers became major health hazards in agricultural populations. Reinhard (1986) presented the following pathoecological explanations of why parasitic disease arose in Ancestral Puebloans relative to earlier hunter-gatherers. Parasitism was limited in hunter-gatherers due to small band size, band mobility, diffuse regional populations, and presence of natural anthelminthics (worm poisons) in hunter-gatherer diets. Hunter-gatherer parasitism was promoted by the consumption of uncooked meat and insects. Parasitism was promoted in Ancestral Puebloan communities by contaminated water sources, concentrated populations, more sedentary life, apartment-style living, establishment of

large latrines, activities centered on water (agriculture), and activities that expanded wetlands including irrigation of all types.

By the nineties, Reinhard (1992) recognized a wide variation in parasitism between Ancestral Pueblo villages (Figure 2). At some settlements parasitism was controlled but others were overwhelmed by their pathogens. This topic was explored by a comparison of pinworm (*Enterobius vermicularis*) prevalence in coprolites (Reinhard 1988). Pinworm was chosen as an indicator of general infectious disease because it is transferred by person-to-person and by environmental contamination (Figure 3). Over millions of years of mutual evolution with hominids and modern humans, pinworm has evolved multiple routes of infection including anal-oral, hand-to-hand, and airborne routes. Pinworms are exceptionally remarkable among human parasites because the female worm wriggles out of the anus of her host at night to scatter her eggs. Once outside of the intestine, she disperses eggs by two different mechanisms. Two types of eggs are produced in two parts of the pinworm uterus: light and heavy. Heavy eggs are laid on the perianal folds with an irritant excretion. The resulting itching (pruritis) and nocturnal host scratching transfers the nearly infective eggs to the host fingers. Other eggs are distributed by aerosol when the female's desiccated body bursts. At this moment, thousands of light eggs are released into the air and bed. Ultimately, these light eggs contaminate the environment. They settle on food, in water, and throughout the habitation. How long these eggs remain infective depends on warmth and humidity. In general, even in arid environments, human habitations have an elevated humidity. Thus, several infection routes result from the females' nocturnal excursions. Retroinfection occurs when the eggs hatch on the perinanal region and the larvae wriggle back into the

host. Hand-to-hand transfer of the eggs occurs when humans interact upon waking. Autoinfection occurs when humans eat food contaminated with the eggs from their own hands. Airborne infection occurs when humans inhale the eggs or when the air dissemination of eggs results in the contamination of food and water. Of course, other pathogens follow the same hand-to-hand, hand-to-mouth, and aerosol routes as pinworm infection. Therefore, high rates of pinworm prevalence suggest high rates of infection by other pathogens that are passed through the same modes of infection (Figure 1).

Some Ancestral Pueblo communities were extremely parasitized. In fact, some sites have the highest levels of pinworm infection recorded for ancient or modern peoples. In a clinical setting, only 5% of feces from pinworm-infected people are positive for pinworm eggs. The percentages of coprolites positive for pinworm from several sites exceed this. For example, 29% of the coprolites from Antelope House, 19% of the coprolites from Inscription House, and 21% of the coprolites from Chaco Canyon sites are positive for pinworm eggs (Table 1). This indicates that pinworm parasitism was unavoidable and that in all probability people had heavy infections. In such populations, pinworm infection is not just a nuisance, but reflects serious health risks, when one considers that other pathogens are spread by the same means.

Reinhard (1992) showed that the prevalence of pinworm parasitism co-varied with porotic hyperostosis prevalence at Ancestral Pueblo sites where both coprolite and skeletons were studied (Figure 4). Porotic hyperostosis is a general skeletal pathology indicator long used to assess maternal-infant health. The fact these indicators of disease had a positive, statistically significant correlation underscores the use of pinworm as a general gauge of Ancestral Pueblo disease state (Reinhard 1992).

Pinworm is not very pathogenic, but is a good proxy gauge of the infectious disease environment (Reinhard 1996). The pathoecological explanation of Ancestral Pueblo and Fremont culture pinworm variation was explored by Hugot et al. (1999). They found that sites in rock shelters without walled villages (some Glen Canyon sites) had the lowest levels of parasitism. Such sites had pinworm prevalence comparable to hunter-gatherers. Village sites outside of rockshelters had intermediate levels of parasitism. Walled villages built within rockshelters had the highest prevalence of pinworm (Figure 3). However, Chacoan Great Houses are anomalous in that they include the wormiest and healthiest sites. Salmon Ruin is among the lowest prevalence values (7%). Pueblo Bonito and Pueblo Alto are among the highest (21%). Therefore, the pathoecology of Great Houses is defined by factors other than size. Great House Puebloans adapted their use of the structures in ways that could promote or limit parasitism.

The remainder of this paper is an exploration of factors that could have limited the parasitism at Salmon Ruin relative to other Great House communities.

Chacoan Greathouse as a Nidi for Infection

Pavlovsky (1966) combined ecological factors into a predictive tool for infection. He realized that for parasitic disease to occur, all factors related to the survival and reproduction of the parasite must be present. These can include vectors, reservoir hosts, humans, and favorable external environments. He defined a nidus as that portion of a natural geographic landscape which contains a community consisting of a pathogen, vectors, reservoir hosts and recipient hosts, and possessing an environment in which the pathogen can circulate. He further found that pathogens possessed nidality. Nidality

is the characteristic of an infectious agent to occur in distinct nidi, such as being associated with particular geographic, climatic or ecological conditions. Thus, a nidus is a focus of infection. For humans, a nidus can be as confined as a single room containing a bed and with access to a rodent carrying plague infected fleas. However, a nidus can be as large as the community and its agricultural area for the transmission of hookworms.

Various types of parasites circulate in nidi. *Temporary parasites* live in the external environment, coming to the host only to feed. Temporary parasites include mosquitoes, chiggers, ticks, and leeches. In these species, every individual must have good dispersal capability and the ability to find hosts when needed. Also, they must possess attributes enabling them to survive in the external environment. Features of the host have less effect on survival and reproduction of these parasites.

Nidicolous parasites live in the immediate environment of the host. In the human environment, they live in beds, walls, granaries, caves, rockshelters, and under floors. Fleas, mites, bedbugs, triatomiid bugs, and the diseases transmitted by these bugs are examples of nidicolous parasites. They depend upon the host not only for food but for creation of their habitat.

Permanent parasites live on or in the host except when dispersing between hosts. These include most protozoa, roundworms, flukes, and tapeworms. They are completely dependent upon their host for both food and all other environmental requirements.

Factors Outside of Great Houses

Water Source, Giardiasis, and Amoebic Dysentery

Water sources in desert environments are foci for human activity. Therefore, a water source can become a nidus. As long as water sources are plentiful and flowing, and

as long as populations are not too concentrated around them, water sources are not necessarily a pathecological factor in the spread of parasitism. However, when water sources become few and stagnant, and when populations aggregate around them, then water contamination becomes a nidus and therefore a significant pathoecological problem.

Giardia lamblia has been found in Ancestral Pueblo coprolites (Gonçalves et al. 2002). This parasite is not too pathigenic in most adults. In fact, most infected people show no symptoms. However, when *G. lamblia* become established in stagnant water sources, it becomes a problem. It is most perilous to pregnant women and their babies. Disease in mothers and children is due to poor maternal nutrition caused by malabsorption resulting in intra-uterine growth retardation. *G. lamblia* causes malabsorption when the intestinal villi become blunted and the function of intestinal mucous diminishes (Carden and MacLeod 1988). Clinical symptoms include cramps, watery diarrhea, nausea, vomiting, and sometimes fever. Among pregnant women who exhibit symptoms, *G. lamblia* causes malabsorption and dehydration at a period when there is a need for accentuated nutritional requirements. Such women fall into a negative nutritional balance (Carden and MacLeod 1988). Carden and MacLeod (1988) summarized the effects of *G. lamblia* on the fetus and newborn. With protracted maternal infections, normal fetal development is impeded. With asymptomatic maternal infections, low birth weight and infant anemia are common (De Morais and Suzuki 1997). Generally, infants become infected after three months of age. Islam et al. (1988) found that there is some immunity conveyed from mother to infant. However, this immunity is not effective in infected infants. The immunity to *G. lamblia* increases with

age (Shetty et al. 1992), and so the pathology caused by *G. lamblia* is significant in infants and toddlers (Hjelt et al. 1992). Sullivan et al. (1991) showed that giardiasis is highly prevalent in children with chronic diarrhea and malnutrition and that giardiasis does not respond to standard therapeutic measures. Children who have low iron and/or vitamin B12 levels have more severe giardiasis symptoms (Awasthi and Pande 1997; Olivares et al. 2002). The epidemiology of giardiasis is well known (Taus et al. 1998; Hjelt et al. 1992; Harter et al. 1982). Subadults in the age range of 9 months to11 years are most susceptible to infection, though infections can occur at 3 months of age. In developing nations, 91% of infants of infected mothers become infected by 6 months of age. Of infected infants, 86% had diarrhea. Infected people tend to live in dwellings with dirt floors, simple latrines, groundwater drinking sources, and close contact with dogs. These aspects of life were common at Ancestral Pueblo villages (Reinhard 1996). In addition, person-to-person transmission of *G. lamblia* is common (Black et al. 1977; Keystone et al 1978; Birkhead and Vogt 1989).

Entamoeba histolytica was also a parasite of the Ancestral Pueblo (Gonçalves et al. 2002). Relative to *G. lamblia*, *E. histolytica* causes more dramatic pathology. They ulcerate the large bowel or ileum. Amoebas can cause nodular ganuloma formation, colitis, diarrhea. The disease can become systemic and eventually become an ulcerative disease of large intestine, liver, lung, brain, or other organs. Amoebiasis becomes symptomatic or even fatal during pregnancy (Abioye 1973; Lee 1929; Lewis and Antia 1969; Rivera 1972). The deaths are due to a rapid onset of profuse diarrhea with dehydration and severe anemia. Premature delivery results from colitis, diarrhea, dehydration, ketosis, and/or shock (MacLeod and Garden 1988). Weigel et al. (1996)

found that high *E. histolytica* load in asymptomatic infections was associated with decreased maternal serum hemoglobin and hematocrit levels, and iron deficiency anemia. Among women who had severe problems (spontaneous abortion, still-birth, low-birth-weight babies), there was a four-fold increase in the prevalence of amoebiasis relative to normal births (Czeizel et al. 1966). In infected but asymptomatic mothers, Weigel et al. (1996) found increased indicators of diminished intrauterine growth. Despite immunity conveyed by antibodies passed through the placenta and in milk, infants can become infected. When this happens, infants exhibit fever with severe watery, sometimes bloody, diarrhea. Colitis, appendicitis, intestinal rupture, and peritonitis result in a high mortality among infected infants (MacLeod and Garden 1988).

The PIII occupation of Antelope House, Canyon de Chelly is the best documented case of an Ancestral Pueblo village that suffers declining health due to water source nidi. Morris (1986) describes the pathoecological conditions that led to water contamination. Towards the end of the occupation, drought affected the region. More distant water sources dried up, and the population of Antelope House and Canyon de Chelly burgeoned. The increased population and decreased water resulted in contamination. Gonçalves et al. (2003) found both *E. histolytica* and *G. lamblia* in Antelope House coprolites. El-Najjar (1986; El-Najjar et al. 1976) found increased skeletal evidence of systemic disease in the PIII occupation of Canyon de Chelly relative to other time periods. Thus, there is a relationship between environmental stress, increased parasitism, and skeletal indicators of morbidity in mother and infants.

For Chacoan Great Houses, only coprolites from Salmon Ruin have been tested for *G. lamblia*. These were negative. The absence of giardiasis at Salmon Ruin is logical

due to the fact that immediate water sources are flowing. These water sources are the San Juan River and the smaller run-off streams flowing off of the hills and into the river. In this area, there is little chance for contamination. No coprolites from Chacoan Great Houses have been tested for *E. histolytica*.

Irrigation, Hygiene, and Hookworm

Hookworm has been found in coprolites from Antelope House and Pueblo Bonito, but at no other Ancestral Pueblo. Hookworm is the greatest parasitic threat to the mother, fetus, and infant. Iron-deficiency anemia resulting from intestinal blood loss is the major consequence of hookworm infection (Variyam and Banwell 1982; Ali et al. 1990). The major treatment for this type of anemia is administration of iron supplements. Gilman (1982) summarizes hookworm-induced iron deficiency anemia. The development of anemia is dependent on the intensity of infection, the species of hookworm, and the ability of the host to resist infection and to maintain adequate stores of iron. Loss of blood is caused by direct ingestion of red blood cells and by tissue trauma produced by worm attachment and feeding. The species that causes the more serious pathology is *Ancylostoma duodenale*.

The species of hookworm identified in prehistoric New World remains is *Ancylostoma duodenale* (Allison et al 1974). This is a fascinating, human-specific parasite that has evolved several infection modes and adaptations. Perhaps the most remarkable aspect of *A. duodenale* is its hypobiotic ability. Hypobiosis occurs when a parasite suspends it development in host tissues in a way that does not elicit a strong immunologic response. *A. duodenale* can go into hypobiosis in winter and come out of hypobiosis in summer. This is a marvelous adaptation because the females then lay their

eggs in the season that is optimal for larval survival. The larvae hatch from their eggs within a few days, exit the feces, develop through three larval stages as free-living soil nematodes. Then, as third stage larvae, they locate human hosts and burrow through the hosts' skin. Also, *A. duodenale* can achieve transmammary and transplacental infection. Thus, fetuses and infants can be infected without ever coming in contact with contaminated soil.

Hookworm causes specific problems in pregnancy. One of the most common causes of death in labor in the developing world is cardiac failure from severe anemia, attributed to hookworm infection (Cintron Villaronga 1967). As many as 90% of pregnant women are infected in endemic areas (Ananthakrishnan 1997, Navitsky et al. 1998). Crompton and Whitehead (1993) presented calculations comparing effects of hookworms on a non-pregnant woman and a pregnant woman. The model predicts that hookworms more rapidly deplete stored iron with a rapid effect on red cell density per milliliter of blood in pregnant women. MacLeod (1988) verified this model from the clinical perspective. Each worm consumes 0.27 ml of blood per day and only 20 weeks after initial infection, hypochromic, macrocytic anemia can develop. The minor symptoms of infection are indistinguishable from complaints of pregnancy (epigastric pain, heartburn, etc.). However, with moderate infections there is low grade fever, fatigue dyspnea, heart palpitations, flow murmurs, and anemia. In heavy infections, constipation or diarrhea, jaundice, emaciation, cardiac failure or pre-eclampsia occur. If a woman survives labor, she cannot recover as easily from post-partum hemorrhage, which can contribute to maternal death.

Hookworms have a negative impact on fetuses and infants (MacLeod 1988). Abortion, still-birth, and premature labor are associated with severe hookworm infection. Women infected with hookworm give birth to low-birth-weight infants (a 2% hematocrit drop in the mother correlates to a 100-gram decline in birth weight). Because of transplacental migration, infants are infected at birth. Severe and sometimes fatal hemorrhage occurs in infants less than four months of age. Chaudhary and Jayaswal (1984) first described an anemic infant resulting from transplacental migration. In a survey of hundreds of trnasplacental-infected infants in China, Yu et al. (1995) defined the symptoms of transplacental infection which includes bloody stools, melena, anorexia, listlessness and oedema. *A. duodenale* was the species implicated in these types of infection. Transplacental migration is not rare. Nwosu (1988) documented that 10% of Nigerian 316 newborns (4-5 weeks old) were infected with *A. duodenale*. Transmamary infections from mother to infant also occur with similar heath results (MacLeod 1988). Studies of many groups from around the world link hookworm disease, especially from *A. duodenale*, to significantly worse iron deficiency and anemia in children (Albonico 1998; Stoltzfus et al. 1998).

Hookworm infection is dependent on moisture, shade, and warmth. The Colorado Plateau is normally too dry to promote infection and in historic times, hookworm was unknown. The discoveries of hookworm eggs in Anasazi sites, and another parasite with a similar infection mode, *Strongyloides stercoralis*, were surprising (Reinhard 1995c; Reinhard et al. 1986). Clearly, Ancestral Puebloans made microenvironment nidi where parasite larvae could hatch and mature in moist, warm, shaded soil. Puebloans also spent

time in these nidi where they spread eggs and became infected by larvae. It is very likely that irrigated fields were hookworm and *S. stercoralis* nidi.

Studies of hand and foot washing in Bengal shows that the larvae can be washed off easily within a few minutes of coming into contact with the skin. The infection occurred in defecation grounds and washing was prescribed by religious rules (Nawalinski et al. 1978). We do not know if Ancestral Puebloans had similar rules, but it is very likely that hookworms could penetrate the skin of puebloan farmers while the farmers worked in irrigated fields. It may be that Puebloan men were more often infected than women, if the division of labor resulted in men working more in irrigated fields.

Factors Inside of Great Houses

Apartments, Plazas, Kivas and Second Floor Living

Pat Horne (1985) attributed the remarkable pinworm prevalence among Ancestral Pueblos as a result of apartment style living. As noted above, Hugot et al. (1999) elaborated this theme by detailing the aspects of architecture and village location that aggravated pinworm infection. Although, pinworm prevalence was highest in standing-walled villages built within rockshelters, it is important to note that there is no thoroughly studied Ancestral Pueblo site that has been documented a pinworm-free. Also, related cultures were infected. The earliest Basket Maker II coprolites from Bighorn Cave, Grand Gulch, Utah have a prevalence of 25%. Later, the diffuse populations of Ancestral Pueblo and Fremont in the Glen Canyon area were infected. Even the Sinagua inhabitants of Elden Pueblo were infected (Hevly et al. 1979). The infections resulted from air humidified by human activity and contaminated with floating eggs within confined spaces.

Although pinworm tends to be asymptomatic, heavy infections at high prevlance can result in real pathology including secondary bacterial infections in kids. However, to my mind the real relevance of pinworm relates to other diseases that are also transferred by air. For the Ancestral Pueblos, tuberculosis was the other air-borne disease. Fink (1985) provides the most poignant epidemiological description of the tuberculosis threat to Pueblos. His description is applicable to both diseases. Fink (1985) presented details of Anasazi life, such as communal living, lack of knowledge of the germ theory, and cramped living conditions, that promoted infectious diseases.

The San Juan occupation of Salmon Ruin is enigmatic in context of pinworm prevalence at other sites. Only 7% of 112 coprolites studied from that site contain pinworm eggs. This is approximately one fifth of the infection prevalence recorded for other PIII Pueblos, including Pueblo Alto and Pueblo Bonito. An explanation for this relates to Paul Reed's (personal communication) that primarily the second floor rooms at Salmon were used for human activities. The air conditions in the lowest rooms, and those closest to the windowless rear wall were the best for pinworm infection. Rooms with the most stagnant and humid air best promote airborne infection with pathogens. By building hearths in second floor rooms, the air there would have been drier. Any rooms opening towards the large Salmon plaza would have been healthier due to access to the dry, relatively breezy air outside the Pueblo.

Probably kivas were the main focus of airborne disease transmission at Salmon. I believe that subterranean rooms were the main feature of Puebloan disease transmission. In kivas, the air would have been humid. The air flow around the ventilator would have been sufficient to maintain airflow of particles around the room, but not enough to lift

infectious particles out of the room through the entry in the ceiling. That explains, in part, why Basket Maker II people developed high a prevalence of infection. They lived in pithouses. After Basket Maker times, kivas were the subterranean nidi of pinworm dissemination. One way to test this hypothesis would be to analysis sediment samples from kiva floors and compare these to analysis of living room floors and milling room floors.

By far the healthiest place to work and live was the outdoor plaza and outdoor rooftops. Sunlight desiccates and radiates pathogens, thereby reducing the number of infectious airborne contaminants. Clean air constantly moves across the plaza, providing people with alternate, healthy air. The humidity decreases there, relative to pueblo rooms. All of these factors made the plazas the healthiest places to live with regard to airborne infectious particles.

Sanitation and Hymenolepidid Tapeworm Infection

With regard to tapeworms, there are two main types of host. The definitive host is the animal in which tapeworms accomplish sexual reproduction. Intermediate hosts are infected with non-sexual stages. Usually, tapeworm definitive infections in humans occur when humans eat an infected intermediate host.

The most common tapeworm that is found in Ancestral Puebloan coprolites is different. It is *Hymenolepis nana*. This tiny tapeworm has evolved the ability to use an intestinal villus as its intermediate host. The larvae emerges and becomes an adult in the intestinal lumen of the same host. Thus, this tapeworm uses the human host as its intermediate and definitive host.

Tapeworms have two methods of laying eggs. The tapeworms anterior end is called a scolex. This attaches to the intestinal wall. Proglottids develop from the scolex. The proglottids are the sexually reproducing tapeworm organs. As the proglottids progress downward along the length of the tapeworm, their ovaries and testes mature, fertilization occurs, and eggs mature. When a proglottid filled with mature eggs, it is said to be gravid. In some tapeworms such as those that infect humans who eat poorly cooked fish, the eggs are laid through gravid proglottid genital pores. In other species, such as those that infect humans who eat undercooked beef, entire gravid proglottids break off of the tapeworm. These proglottids are partly motile, and squirm their way out of the body.

Hymenolepis lays infective eggs through genital pores and its eggs are infective when they pass into the environment. Although they have been found in Canyon de Chelly coprolites, they have not been found in Chacoan Great Houses.

Conclusion

The Chacoan Great Houses provided many potential nidi for temporary, permanent, and nidiculous parasites. How Great House inhabitants used their villages created or eliminated nidi. For permanent parasites, there is was no way to completely eliminate the transmission. However, some aspects of Salmon Ruin life reduced the prevalence of pinworm relative to other Great Houses. I suggest that lower human population density accomplished this.

The absence of fecal borne parasitism indicates that nidi of fecal exposure were eliminated at Salmon Ruin. The use of specific rooms as latrines was an effective way of stopping the spread of parasites such as *Giardia lablia*.

Nidi external to Salmon Ruin where hookworm and *S. stercoralis* transmission could have taken place did not exist. This was probably due to a different type of irrigation and gardening relative to that of Pueblo Alto and Pueblo Bonito where hookworms did infect humans.

In the future, more extensive analysis of Ancestral Pueblo coprolites must be done with all research methods. Currently, some sites such as Antelope House have been the focus of molecular, immunological, and microscopic analysis. Other sites that were studied long ago such as Glen Canyon were studied only through microscopy. When all method have been applied to all sites, then we will construct a complete picture of Ancestral Pueblo parasite pathoecology.

In the future parasitological methods must be developed for the analysis of remains in addition to coprolites. Many nidiculous pathogens such as bed bugs and kissing bugs live in walls and roofing. Therefore, archaeological excavations should include samples of sediment around architectural remains for analysis of insect exoskeletons. Analysis of trash sediments for all types of parasites must be developed in order to gain parasitological data from sites in which coprolites do not preserve. Once these approaches are developed, then a true archaeology of parasitic disease will emerge.

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List of Figures