

Pre-Columbian Tuberculosis: An Epidemiological Approach

JANE E. BUIKSTRA, PH.D.

Associate Professor, Department of Anthropology, Northwestern University, Evanston, Illinois

DELLA C. COOK, PH.D.

Assistant Professor, Department of Anthropology, Indiana University, Bloomington, Indiana

In a previously reported study¹ we developed a differential diagnosis for focal, resorptive skeletal pathology found in twelve individuals recovered from the Schild Mississippian Cemetery, Greene County, Illinois. The pathology was characterized by vertebral body destruction within the lower spine, frequently associated with osteoporosis and focal destruction of the articular surfaces of the appendicular skeleton. In an extreme case, a 20-21-year-old female (SB 201) displayed kyphosis and ankylosis involving fourteen vertebrae; however, the modal pattern for the series consisted of cystic resorption within only one or two vertebral bodies, with little to no proliferative response or involvement of other elements. Localization within the thoracolumbar region was typical, as was destruction of articular surfaces in cases of extravertebral lesions.

A differential diagnosis was made for the pooled Schild adolescent/adult sample, using data for 16 contemporary diseases. Attributes of lesion form and distribution, and mortality statistics, were stressed. Based on this differential diagnosis, it was suggested that the most likely association for the Schild pattern

was with one of two forms of modern pathology: tuberculosis or blastomycosis. Given a notable elevation of mortality for affected young adults and adolescents, tuberculosis was considered to provide the preliminary model of "best fit," though this conclusion was necessarily tentative, pending further investigations.

The present study was designed to test the Schild model on an expanded data base, using other prehistoric series from the same geographic region. All relatively complete skeletons from the supplementary samples were examined for indications of resorptive disease similar to those which characterize Schild. Upon identification of affected populations, we then explored the epidemiological relationship of this pathology to other biological and cultural variables, especially those which reflect subsistence strategy, relative population density, aggregate size, and inter-regional interaction. Factors potentially limiting our interpretive power, for example, differential recovery rates, were also considered.

Materials and Methods

The present research represents an extension of the earlier Schild study to include all evidence of grossly observable focal resorptive pathology for the archaeological samples listed in Table 1. The initial series of 175 adolescent and adult Schild individuals (over 10 years of skeletal age) has been expanded to include juveniles from the Schild Mississippian

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Correspondence and reprint requests to Dr. Jane E. Buikstra, Department of Anthropology, Northwestern University, Evanston, Illinois 60201.

TABLE 1
Lower Illinois Region Series
CALIBRATED, AVERAGED

SERIES	N	DATES ¹	CULTURAL AFFILIATION
Yokem Mississippian	66 (adults)	1292 ± 81 ²	Mississippian ²
Schild Cemetery	335	1068 ± 81 ³	Mississippian ³
Leadders Mounds	187	1018 ± 30 ⁴	(late) Late Woodland ⁴
Schild Mounds	176	783 ± 39 ⁵	(late) Late Woodland ⁵
Joe Gay Mounds	114		(early) Late Woodland ⁶
Koster Mounds	199	651 ± 48 ⁷	(early) Late Woodland ⁷
Homer Adams	64		(early) Late Woodland ⁸
Gibson	139		Middle Woodland ⁹
Homer Adams	13		Middle Woodland ¹⁰
Joe Gay	31		Middle Woodland ¹¹
Lawrence Gay	33		Middle Woodland ¹²

¹ After Long and Rippeteau³¹ Damon, et al.³²

² Crane and Griffin³³ Perino³⁴

³ Crane and Griffin³⁵ Perino³⁶

⁴ Tainter¹⁹

⁵ Tainter¹⁹ Perino²⁴

⁶ Tainter¹⁹ Cook¹⁴ Perino (unpublished data)

⁷ Crane and Griffin³⁶ Tainter¹⁹

⁸ Perino (unpublished data) Cook¹⁴

⁹ Perino (unpublished data) Buikstra²⁰

¹⁰ Perino (unpublished data) Cook¹⁴

¹¹ Perino (unpublished data) Cook¹⁴

¹² Perino (unpublished data) Cook¹⁴

sample, all skeletons from nine Woodland series, and preliminary observations on adults from the Mississippian component of the Yokem site. Thus documented are more than 1,000 years of prehistory and a geographic range which consists of the lower 70 miles of the Illinois River valley and adjacent uplands extending to and including the east side of the Mississippi River valley (Fig 1). We have examined all skeletons with two or more observable elements, yielding a total sample of 1,357 individuals.

It should be noted that data from juvenile materials *were* considered in this study, as they were *not* in the previous investigation. The earlier screening excluded juveniles because it was felt that lesions which would be most valuable in initial differential diagnosis were to be found in mature individuals. In the present study, however, given that our interpretation appeared to center upon distinguishing between tuberculosis and blastomycosis, the juvenile data could play a crucial role.^{2,3,4} First of all, we must recognize that our expectations for lesion form and distribution in juveniles is not identical to that for adults. For instance, tuberculosis results in metaphyseal rather than epiphyseal resorption in children.^{2,3,4} For the purpose of differential diagnosis, it is most important to note that modern data would lead us to anticipate

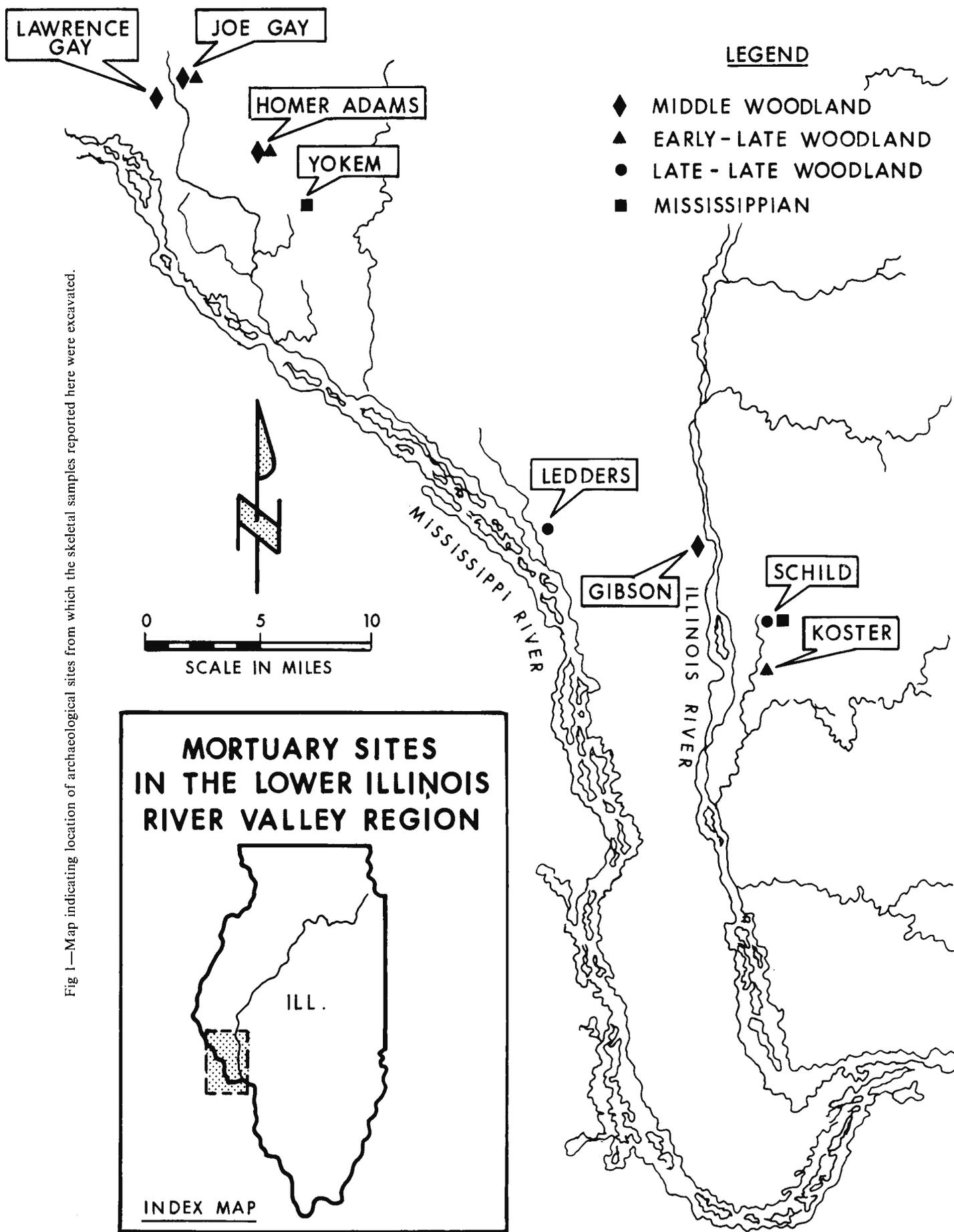
epidemiologically distinctive patterns for tuberculosis when contrasted to blastomycosis. Groups suffering from tuberculosis should show recognizable disease, for example, fusiform expansion of long bone diaphyses in infants and children.⁵

By contrast, although blastomycotic lesions have been reported for infants as young as 6 months of age,⁶ most sources agree that juveniles infrequently contract blastomycosis prior to age 10.⁷⁻⁹ Therefore, either the direct recognition of lesions attributable to tuberculosis, or indirect evidence of increased disease incidence in young juveniles could provide evidence important to us for diagnostic purposes. We will, therefore, describe resorptive lesions in juveniles which may be directly associated with the adult pattern, and also report certain other lesion types, specifically cribra orbitalia and meningeal reaction, which may serve as indirect or synergistic markers of the elevated disease load.

Results

The results of this investigation will be presented according to the chronological ordering of the study series, beginning with the earliest samples. The four most ancient series to be considered here comprise the Middle Woodland components of the Gibson,

Fig. 1—Map indicating location of archaeological sites from which the skeletal samples reported here were excavated.



Joe Gay, and Homer Adams sites, as well as the total Lawrence Gay sample.

MIDDLE WOODLAND

In the lower Illinois region, the Middle Woodland period extends from approximately 100 BC to AD 400.

None of the 216 Middle Woodland adults and juveniles showed any sign of the oval, granular-walled cysts which characterized the Schild Mississippian series. The only observable resorptive pathology was undoubtedly age-related, including Schmorl nodes and degenerative remodelling of the sacroiliac articular surfaces. It seems reasonable, therefore, to exclude the Schild pathology from the disease load of Middle Woodland populations in this region.

EARLY LATE WOODLAND

Chronologically, the next most recent period is termed early Late Woodland, dating from approximately AD 400 to AD 800. As in the Middle Woodland sample, there is no conspicuous display of resorptive skeletal pathology among the three early Late Woodland series reported here ($N = 377$).

One adult, sex unknown, from the Late Woodland component of the Homer Adams site shows a rather large (about 2 cm maximum diameter), smooth-walled cyst located within the medullary cavity of the left femur deep to the lesser trochanter, as well as the intertrochanteric line and crest. There is a drainage channel penetrating from the lesion to the external surface of the cortex at the distomedial aspect of the intertrochanteric line. There is no active remodelling in the region of the drainage channel. The extent of this lesion or the presence of associated noncontiguous lesions is difficult to determine, given that the fragmentary proximal femur diaphysis/metaphysis represents the major portion of this imperfectly preserved individual. No other skeletal remains from the Homer Adams series displays resorptive pathology.

None of the skeletons recovered from the early Late Woodland component of the Joe Gay site show cystic, resorptive foci; however, one individual—Mound 2; Burial 6—does present an unusual bit of remains which may reflect calcified pleural tissue (Fig 2). If this is indeed pleural calcification, it would be consistent with pulmonary foci of either blastomycosis or tuberculosis, although numerous other forms of pathology could produce similar remains. Given



Fig 2—Tissue artifact from Joe Gay site, Mound 2, Burial 6. This remains may reflect calcified pleural tissue.

the nonspecificity of this tissue artifact without confirmatory data from skeletal lesions, such evidence is at best suggestive.

The Koster early Late Woodland sample, the single Illinois Valley series, does display numerous lesions which should be termed resorptive; however, the distribution of these foci, as indicated in Table 2, does not center in the vertebral column. In addition, when lesions which are explicitly secondary to primarily proliferative response are deleted from consideration, along with Schmorl nodes, we are generally left with a few subarticular digital foci in adult females and one juvenile, and an apparent developmental defect in the spinous process of the first sacral unit which is best considered as a normal variation.

In the total Koster sample, there exists only a single instance of cystic resorption associated with coarsened granular reaction in the surrounding bone. This isolated example, occurring in the ischium of a 35-45-year-old male (Mound 4; Burial 1B, Fig 3), consists of a sinus draining to the ventral aspect of the descending ramus of the left ischium. A 14-15-month-old juvenile (mound unknown) displays two small resorptive foci confluent with the proximal epiphyseal surface of the metaphysis of the left humerus. The underlying trabecular bone does not appear coarsened, and there is a cuff of reactive tissue around the margin of the lesion. Given the absence of diagnostic vertebral lesions, and the numerous other possible explanations for the few foci which do appear, it seems that we cannot identify the Schild tuberculosis/blastomycosis-like pathology in the

TABLE 2
Resorptive Foci in the Koster Early Late Woodland Series

MOUND-BURIAL NUMBERS	AGE/SEX	LOCATION OF LESION
?-B	14-15 months/?	Left proximal humerus, metaphyseal surface
1-14	25-26 yrs/F	Subarticular region of metacarpals (extensive osteomyelitis throughout body)
1-21	14 yrs/F?	Non-ovoid resorption on superior and inferior aspects of L4, superior aspect of L5
3-7	21-24 yrs/F	Ventral aspect of manubrium sternum
4-1B	27-30 yrs/M	Left humerus diaphysis, left descending ramus of ischium
4-6	47+ yrs/F	Left carpal (navicular), metacarpal, subarticular lesions
4-9	27-30 yrs/M	Central portion of inferior surface of body of L5, superior aspect of S1—Schmorl nodes?
4-14	30-35 yrs/F	Right ulna, olecranon process in region of triceps insertion, left humerus medial to greater tubercle, right 4th metacarpal-subarticular
Kn5-6E	35-39 yrs/M	Distal right fibula metaphysis
5-14	50+ yrs/F	Multiple minor diaphyseal resorptive foci in association with primarily proliferative response.
5-28	41-42 yrs/F	Cubical and long bones of hands/feet (right side) show articular/subarticular lesions. Right aspect of frontal bone also displays two small foci.
6-8	14-16 yrs/?	Superior aspect of the spinous process of S1
7-19	9 yrs/?	Left 3rd metacarpal, subarticular

Koster series, or in any of the other early Late Woodland populations investigated here.

LATE LATE WOODLAND

The dates for later Late Woodland sites, such as the Ledders and Schild Mound groups, range be-

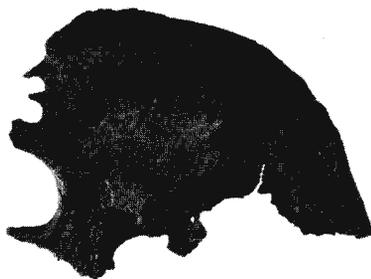


Fig 3—Descending ramus of left ischium, ventral aspect, of Koster Mound 4, Burial 1B, showing location of resorptive lesion.

tween AD 800 and AD 1050. The location for these two sites is indicated in Figure 1.

Ledders adults show no resorptive pathology, although three juveniles do have pathological changes which could be characteristic of tuberculosis, blastomycosis, or a number of other forms of infectious pathology. Mound 1; Burial 144, an infant, shows multiple long bone involvement characterized by fusiform, expansive remodelling of the diaphyses (Fig 4). The cortices are thinned, with the underlying trabecular structure resorbed. There is light fiberbone periostitis in the expanded region, but periostitis is not conspicuous in nonexpansive areas. Similar remodelling is seen in the distal right humerus of Burial 115 from Mound 1, which shows a sinus draining directly into the epiphyseal area. A third individual shows minor erosion in a proximal humerus epiphysis. In sum, though the Ledders juvenile pathology could be associated with tuberculosis, blastomycosis or a range of other mycotic and bacterial infections, the absence of skeletal lesions in adults precludes the positive identification of the Schild Mississippian pathology here.

The Schild Late Woodland mounds sample shows numerous examples of resorptive skeletal changes; however, the patterning for these loci (Table 3) emphasizes the appendicular skeleton and does not include the thoracolumbar vertebral bodies. Mound 3; Burial 2, a young adult male pictured in Figure 5, does present an instance of expansive resorption at the sacroiliac articulation which could be associated with tuberculous or mycotic pathology, but the existence of this case in isolation does not verify the existence of the Schild Mississippian pathology in the Schild Late Woodland sample. Given the absence of diagnostic vertebral pathology in a total sample of 349 terminal Late Woodland individuals, it appears that we must reject the notion that these groups suffered from the Schild tuberculous/mycotic-like affliction.

MISSISSIPPIAN

The uniformly negative results from the Woodland series which immediately predate the Mississippian populations that were buried at Schild induced us to conduct preliminary screening of a second local Mississippian sample. We wished to investigate the degree to which the Schild pathology appeared to be an isolated entity, or whether it might be more

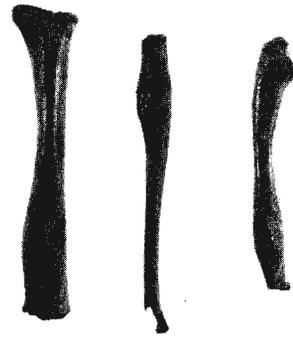


Fig 4—Long bones from Ledders Mound 1, Burial 144, showing fusiform expansion of diaphyses.

broadly associated with other groups which existed during the AD 1050 to AD 1300 period. The sample chosen for investigation was the Yokem series, excavated from a cluster of mounds located on the Mississippi River drainage (Fig 1). To date, only adults and adolescents have been observed.

A summary of the Yokem results appears in Table 4. All possible resorptive lesions are reported,

TABLE 3

Resorptive Foci in the Schild Late Woodland Sample. Individuals with Primary Proliferative (Osteomyelitic) Responses are not Included

MOUND-BURIAL NUMBER	AGE/SEX	LOCATION OF LESION
1-1	12-14 yrs/F?	Distal femur epiphyses
1-17	39-44 yrs/M	Ventral aspect of odontoid process, and anterior aspect of C2 body. Subarticular lesion on right 1st metatarsal. Given widespread periostitis, primary response does not appear resorptive.
2-4a	adult/F	Glenoid fossae of both scapulae, 2nd cervical vertebra, and right ischial tuberosity
2-18	35-39 yrs/F	Left humerus, dorsal aspect of anatomical neck
2-19	30-39 yrs/M?	Superior aspects of right and left 3rd cuneiforms
3-2	27-30 yrs/M	Auricular surface of left innominate
3-6	20-25 yrs/M	Medial epicondyle of left femur
3-18	adult/F?	Dorsum ilii of right innominate
3-20	35-39 yrs/M	Carpals, left tibia, distal femora
9-17	25-29 yrs/F	Epiphysis of right clavicle
9-28	39-44 yrs/M	Sacrum, on superior aspect of body of L1, apparently Schmorl node



Fig 5—Auricular surface of left innominate from Schild Mound 3, Burial 2. Note expansive resorption.

with those clearly defined by osseous reaction listed as “Group 1—Core.” A second group is recorded as “non-core” due to the fact that although resorptive pathology is suspected, either poor preservation or proliferative response (ankylosis) has rendered our determination imprecise. The following discussion will emphasize data derived from Group 1.

As evidenced by Table 4, the pattern for resorptive pathology in the Yokem series clearly deviates from the previously described Woodland pattern.

Furthermore, the deviation *is* in the direction specified by the Schild Mississippian model. Though the sample size is decidedly small ($N = 66$), an axial concentration of lesions is easily distinguished. Sacroiliac involvement, as well as thoracolumbar destruction (Fig 6) mirrors the Schild pattern. Lesions appear in the central portions of vertebral bodies, either having originated at the dorsal aspect or in the disc. Circumferential erosion is also common, with little proliferation of tissue, except in advanced or healing phases. The surface texture of the affected bone is generally coarsened and sclerotic, especially at the base of the circumscribed, oval lesions. We may therefore conclude that the Schild pattern is clearly identified in the Yokem sample.

The final osseous sample to be described here comprises the juveniles from the Schild Mississippian series. None of the Schild juveniles less than 10 years of skeletal age show signs of resorptive foci; however, one 6-8-year old (SA-76C) did display active periostitis within the iliac fossa, which might have been associated with a psoas abscess. Both this individual and a heavily diseased 10-12-year old, Knoll A; Burial 4, showed extensive porosity of the cubical and tubular bones of the feet, in association with periosteal elevation in the calcanea. Four other juveniles between the ages of 6 and 12 years showed similar porosity and periosteal elevation; no such patterns

TABLE 4
Resorptive Foci in the Yokem Mississippian Sample

MOUND-BURIAL NUMBER	AGE/SEX	LOCATION OF LESION
Group 1—Core		
1-6	40-50 yrs/Female	Sacroiliac articulation; bodies of 7th-12th thoracic vertebrae. 1st and 2nd lumbar
1-34	25-26 yrs/Male	3rd lumbar vertebra, body
2-5	41-42 yrs/Female	5th lumbar and 1st sacral unit, across disk involvement
2-15	17-18 yrs/Male	Midlumbar (poorly preserved), bodies affected
Group 2—Non-core		
1-8	15-17 yrs/Male?	Lower thoracic vertebra (poor preservation), body
1-9	50+/Female	2nd and 3rd lumbar vertebrae fused, no apparent resorptive lesions
1-36	50+/Male	Four thoracic (poor preservation) show resorptive foci which could be extreme case of degenerative (Schmorl nodes) change.
3-6a	50+/Male	12th thoracic through L2 are ankylosed, with no observable inflammatory response or resorptive foci. Resorptive foci on neck of left femur and distal right femur metaphysis.

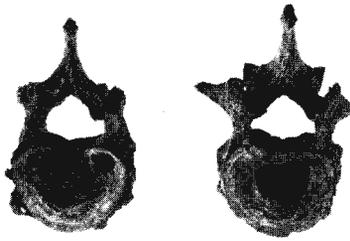


Fig 6—Lower thoracic and lumbar vertebrae from Yokem Mound 1, Burial 6. Note resorptive pathology.

were noted for younger juveniles. Though such data are admittedly tenuous, it is possible that this pedal porosity/periostitis could be evidence of an osteoarthropathy associated with pulmonary disease. It is important that this pattern was found in *none* of the Woodland juveniles. As fully one third of the Schild Mississippian children with suitably observable elements display the tarsal porosity/periostitis pattern, a pathology not common to the earlier Woodland groups may be present. Additional indirect evidence exists for the presence of a form of pathology in the Schild Mississippian juveniles which was not present in the other samples. As indicated in Figure 7, which reports frequency data for cribra orbitalia—commonly accepted as evidence of anemia—the Woodland pattern contrasts with that for the Schild sample. Woodland juveniles display the expected incidence for an anemia resulting from dietary causes, including iron deficiency, protein-calorie malnutrition, scurvy, and rickets. These and other specific nutrient deficiencies should not be observed prior to 6 months of age, given the presence in neonates of stores accumulated in utero. Such is the Woodland model, showing an incidence peak for cribra orbitalia at approximately 1-2 years, followed by a decrease in frequency to virtual absence by early childhood. The Schild Mississippian pattern, however, is different. There is a relatively high incidence of cribra in individuals dying between birth and six months, followed by the expected peak at 1-2 years. It appears that an underlying dietary deficiency anemia is present, but that another form of pathology has been superimposed. This would be compatible with the addition of

a new infectious pathology, such as tuberculosis, to the range of pathogens normally encountered by lower Illinois Valley inhabitants.

If tuberculosis is indeed the causative agent indirectly identified here, we might also expect other indirect indications of increased disease experience in juveniles, including elevated frequencies of meningitis, a prominent cause of childhood deaths from tuberculosis prior to effective chemotherapy.¹⁰⁻¹³ The investigation of bony meningeal reaction failed to identify a distinctive pattern for the Mississippian sample, although it has been shown in other studies¹⁴ that meningeal reaction is closely linked to the frequency of quasi-treponemal osteomyelitic lesions in the Woodland series. Given the high frequency of osteomyelitis in all series investigated here, it is not improbable that any minor effects of a disease of relatively low incidence would be obscured.

Recovery Rates

Before interpreting these results, it is necessary to consider an important source of bias: the frequency of observable elements per prehistoric skele-

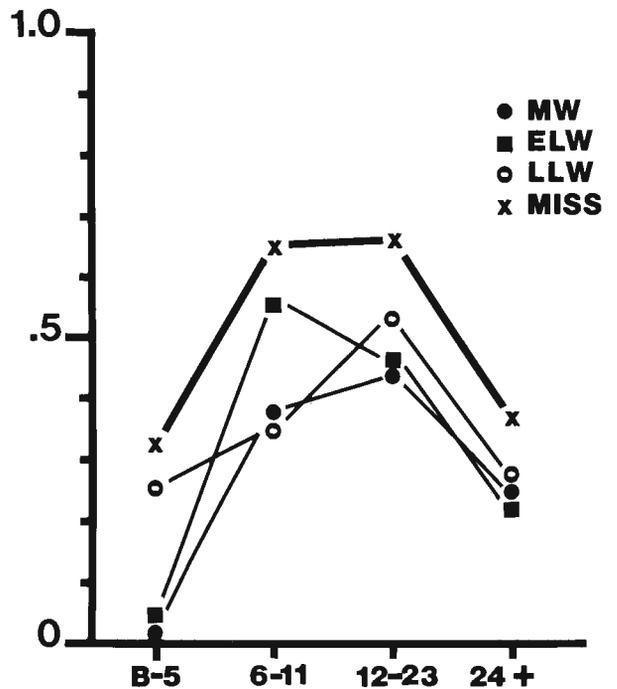


Fig 7—Frequency of cribra orbitalia in Woodland and Mississippian juveniles less than 6 years of age. Age in months appears on the horizontal axis. (Dot = Middle Woodland; Square = Early Late Woodland; Circle = Late Late Woodland; X = Mississippian.)

ton, relative to total expected frequency, if all bones for each individual were present and subjected to study. Table 5 presents recovery rates generated for three Woodland and two Mississippian skeletal samples. Each element is reported in terms of the observed number of bones as a percentage of the expected, given the number of skeletons studied and the distribution of bones in the human body. By comparing the recovery rates for the femora with those for the vertebrae, one quickly notes a differential recovery pattern between the Woodland and the Mississippian samples. (The vertebrae are the bones most crucial to our present analysis; the femora are bones most frequently preserved and recovered from these mortuary sites.) The relatively minor underrepresentation of vertebrae in the Mississippian series contrasts with the Woodland samples; apparently the recovery rates for the femur do not vary significantly between the sites, though vertebrae are clearly underenumerated in the Woodland series. Although numerous factors can affect this underrepresentation, including the poor preservation of vulnerable vertebrae and the vagaries of archaeological recovery, the main effect here probably derives from the high frequency of processed, or bundled, burials at the Late Woodland sites, when compared with the Mississippian loci.

Whatever the cause, it is easy to see that this differential representation of body parts can easily affect one's ability to observe area-specific pathology of expected low frequency. Given the small size of the Yokem Mississippian sample, we are still impressed by the observed shift in disease pattern from that displayed by prior Woodland populations; however, this impression should be qualified by a consideration of Woodland sampling biases.

Interpretation

Having tentatively accepted the idea that Mississippian groups did suffer from an osseous disease not found in earlier populations, we may then explore our data for support of the alternative tuberculosis and blastomycosis models. As our expectations for the form and distribution of resorptive lesions in individual skeletons do not vary significantly between the two diseases, our analysis must emphasize epidemiological data as well as indirect evidence concerning nonspecific osseous change.

In favor of the tuberculosis model are our epidemiological data from the Schild adults, who show marked young adult mortality and a lack of sex bias.

Local and generalized osteoporosis in both adults and juveniles would also be compatible with tuberculosis, as would the evidence of an increased disease incidence in very young infants. However, to be weighed heavily against such data is the complete absence of diagnostic resorptive vertebral lesions in Mississippian individuals of less than 10-12 years of age. Though we hesitate to base our conclusions upon negative evidence from admittedly fragile and incompletely preserved materials, the absence of resorptive pathology in juveniles *is* important. Though intuitively the diagnosis of tuberculosis may seem more pleasing, the presence of blastomycosis remains possible.

It is appropriate to consider certain biocultural factors which may be important in explaining the existence of tuberculosis and blastomycosis. We will emphasize three variables critical to the development of a human density-dependent disease such as tuberculosis, or an environmental pathogen such as blastomycosis: subsistence and settlement patterning, population density, and interregional interaction. Table 6 summarizes the trends in these biocultural variables.

The earliest series under consideration here date to the Middle Woodland period (ca 100 BC to AD 400). During this period of prehistory, population density for the Illinois Valley has been estimated by Asch¹⁵ at less than one person per square mile. Habitation apparently centered upon the main river valleys and their major tributaries; exploitation of natural resources was characteristically diverse, with emphasis on deer hunting and collection of wild plants.¹⁶⁻¹⁸

The possibility of frequent interaction with contemporary Middle Woodland populations from other regions is suggested by the presence of finely crafted "Hopewell" items which were manufactured from raw materials not available in the local area.¹⁹ The aesthetically pleasing Hopewell artifacts are most commonly found in burial mounds and doubtless had some social significance for Middle Woodland groups.^{20,21} It should be noted, however, that not all archaeologists accept the idea that Middle Woodland communities were characterized by either extensive trade relationships or complex social structure.²²

Thus, our baseline Middle Woodland model for subsistence, demography, and interaction describes a spaced series of hunting and gathering communities which had base camps within the major river valleys, but whose seasonal movements doubtless included

TABLE 5
Recovery Rates (%)

ELEMENT	SITE				
	Schild Mississippian	Yokem Mississippian	Schild Late Woodland	Ledder	Koster
Cranium	83	94	84	88	77
Mandible	80	91	76	73	72
Scapula	82	91	73	66	86
Clavicle	78	88	70	62	68
Humerus	90	92	92	73	87
Radius	84	86	79	66	79
Ulna	86	86	82	74	81
Innominate	85	92	84	67	86
Femur	90	96	91	88	89
Patella	52	67	53	47	50
Tibia	87	94	89	78	87
Fibula	85	85	80	58	76
Sternum	67	69	46	37	46
Ribs	64	76	34	35	44
Carpals	32	51	25	35	31
Metacarpals	61	72	47	43	47
Hand Phalanges	33	46	24	34	26
Tarsals	62	72	51	50	50
Metatarsals	60	73	53	45	48
Foot Phalanges	13	18	13	17	13
Cervical Vertebrae	73	81	51	49	49
Thoracic Vertebrae	78	88	53	51	56
Lumbar Vertebrae	80	90	59	53	63
Sarcum	80	94	66	55	81

adjacent uplands and other valley regions. Local group interaction may have been structured as part of an annual or even a multi-year cycle; however, there is evidence against extensive population movement or migrations.

Thus described, Middle Woodland does not provide an ideal environment for the development of a human density-dependent disease such as tuberculosis. Although there is indication of an interactive network which could have supported disease transmission, one essential element—sedentary human population aggregates of large size—is absent. Mycotic disease is also unlikely because Middle Woodland subsistence did not require intensive cultivation of the soil. In this biocultural milieu, the negative results of our present osteological research do not seem surprising.

Chronologically, the next most recent period is early Late Woodland, which is commonly characterized by an absence of Hopewell items as well as by certain other stylistic distinctions. Archaeologists see this as a time of increased population density,^{17,23} for

although sites may not increase significantly in size, they do increase in number. Subsistence strategy does not seem to have changed markedly during most of this period, but apparently there was active inter-group conflict, evidenced by the frequency of projectile points in human burials, as noted by G. Perino^{24,25} (unpublished data). Thus, though popu-

Table 6
Chronology of Biocultural Variables

	Sub- sistence Strategy*	Popula- tion Density	Supra- local Interaction
Mississippian	AG/h-g	stable?	+++
Late Late Woodland	h-g/ag	increase	-
Early Late Woodland	h-g	increase	-
Middle Woodland	h-g	<1/sq mi	+

* AG/ag = maize horticulture or agriculture; H-G/h-g = hunting and gathering of locally available resources. Capital letters indicate importance in excess of that denoted by lower case script.

lation density increased, opportunities for disease transmission were fewer. Aggregated, sedentary human populations are not present, nor is there an indication of intensive cultivation. Given these biocultural data, our inability to document either tuberculosis or blastomycosis is not unexpected.

The later Late Woodland period, dated here between AD 800 and AD 1050, includes groups which were engaging in maize agriculture for the first time in this region.^{17,18} There is also archaeological evidence of a continued increase in population density,²³ and sites are now located in uplands, as well as in the main river valleys.¹⁷ Although hunting and gathering of local resources doubtless remained important in local subsistence strategies, the cultivation of maize represents a major alteration in dietary composition and also affects settlement patterning. There is no evidence during this period of extensive trade or population movement.²⁶ Given the biocultural characteristics of the later Late Woodland period, the lack of evidence for a tuberculosis-like pathology is not surprising. Population density apparently has increased; however, the essential combination of a pathogen, a mechanism for transmission, and a relatively sedentary and sufficiently large human host population is not present. Our expectations for tuberculosis in this environment are low, and are compatible with the observed absence of resorptive spinal pathology in any terminal Late Woodland series.

If, alternatively, the pathology identified here is indeed blastomycosis, our osteological data are disquieting. Though intensity of agricultural activity cannot be measured precisely at this time, maize is conspicuously present in Late Woodland habitation sites.^{17,18} To the extent that an increased dependence upon maize reflects an intensified contact with soil-borne pathogens, we should expect to encounter mycotic infection in late Late Woodland skeletons. Such is not the case. Clearly, more archaeological information must be brought to bear on this question. However, at this point it appears that our Woodland osteological and cultural data are most compatible with an expectation that our Mississippian pathology is tuberculosis.

By approximately AD 1050, maize agriculture was well established within the Mississippian tradition. Population density had probably not increased significantly within the lower Illinois region; however, just to the south at Cahokia there is a major aggregation of individuals estimated by archaeologists to number between 25,500 and nearly

43,000.²⁷ Other major local concentrations of smaller size also exist to the north of the lower Illinois region, including an aggregate estimated at between 600 and 1,170 individuals at Larson site which is located at the confluence of the Illinois and Spoon rivers.²⁸ Artifacts recovered from Mississippian sites show many elements common to other, nonlocal groups of the Mississippian tradition. In this way, interregional interaction and communication are well documented.

Within this context, our expectations for the appearance of a human density-dependent disease should probably depend not so much on the size of the local group but on the aggregate size in major centers such as Cahokia. Low estimates of population density have frequently been used²⁹ as indirect evidence that tuberculosis and other epidemic pathology could not have existed in the prehistoric New World. We argue that the population aggregated at Cahokia could have provided a reservoir suitable for the development and maintenance of an epidemic disease, which would have pulsed out into the farmstead communities in adjacent regions. The tuberculosis epidemic reported by Carey³⁰ for the Eskimo Point native population provides excellent documentation of the impact of a crowd infection on a settlement of small size.

Alternatively, intensification of agricultural activity during the Mississippian period may have significantly increased exposure to soil-borne fungi. Based on epidemiological patterns in modern groups, we would expect that those individuals actually performing cultivation tasks would be most readily exposed to the fungus. If our Mississippian groups were suffering from blastomycosis, then agricultural activities apparently were engaged in by both men and women, beginning intensively in adolescence. Other osteological data, for instance, degenerative joint disease patterns, could be used to test this hypothesis.

Conclusions

In this study we have combined both biological and cultural data in the investigation of resorptive pathology in Woodland and Mississippian skeletal series from west-central Illinois. Information concerning the types of lesions and their distribution confirms the presence of a previously unknown disease in Mississippian populations. Adults and adolescents from Yokem and Schild Mississippian components clearly display cystic vertebral pathology, which in association with other peripheral osseous lesions distinguishes them from earlier groups. This

idea is supported indirectly by evidence from both infants and children of the Schild sample.

In our prior study of the Schild adult series, it may be recalled that on epidemiological grounds we were led to favor tuberculosis as the probable agent of the observed prehistoric distress. Now, as a result of our more extensive investigations, epidemiological data have forced us to rethink our earlier preliminary model. As mentioned previously in this paper, we expect that tuberculosis should be evident in juveniles, where low but recognizable frequencies of resorptive lesions should be observable. Leaving non-specific evidence aside, there are no diagnostic lesions in any Schild individual prior to 10-12 years of age; this would be our precise expectation if the pathology in question were blastomycosis rather than tuberculosis. Certainly, we must consider the indirect evidence of infant anemia, tarsal porosity, and the possibility of a psoas abscess in a juvenile (which would also be compatible with a blastomycosis model), and we cannot ignore the elevated young adult mortality in Schild Mississippians. The effects of prehistoric and modern sampling biases as well as the possibility that this as yet unspecified ancient disease bears no modern counterpart causes us to further qualify our statements. It appears, therefore, given the results of the present study, that the question of prehistoric tuberculosis in eastern North America must remain open, pending the further study and refinement of models for differentiating tuberculosis from mycotic infections.

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