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Keywords
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Syphilis: Etiology, Epidemiology, and Origin Theory

Angela Aristone

ABSTRACT

The origin and spread of treponemal infection, particularly its manifestation as syphilis, has been the focus of much political, religious, and scholarly discussion since the time of the Egyptian Pharaohs. There are many current interpretations which explore the complexity of diagnosing treponemal infection in human populations of the past and present, while attempting to account for the various manifestations of the disease in both the Old and New World. The study of the history and epidemiology of treponemal disease has been significantly challenged by the ongoing problem of confident diagnoses within skeletal assemblages. This paper assesses current origin theories, and offers reasons why the Unitarian hypothesis (which holds that syphilis co-evolved with humans, and that different strains of syphilis existed in both the Old World and New World prior to contact) is the most convincing to date. The speculation is made that treponemes may have initially evolved in slime-contaminated waters, and later spread to human populations.

INTRODUCTION

Discovering the origin of a particular pathology lends crucial information not only about the pathology and its process of disease, but it also aids paleopathologists in tracing a pathogen back into the archaeological record. Such ventures shed light on issues such as the possible migration of the pathogen and its differential manifestations as a result of climatic change, nutritional change, and even hygienic advancements and their effects on the pathogens future.

Treponemal infection, particularly its manifestation as syphilis, has commanded an abundance of attention in the present, and throughout the past few hundred years. Nothing conclusive is known about the origin of syphilis, just as nothing conclusive is known about the origin of the AIDS epidemic. Fortunately, this lack of conclusive evidence has encouraged curious minds to continue to hypothesize regarding its provenance.

There are numerous hypotheses about the origins of syphilis, and an abundance of facts are now known about the pathological process of syphilis, but there is still much confusion and ambiguity surrounding it. The purpose of this paper is to discuss the etiology and epidemiology of syphilis and to understand all the possible expressions of syphilis, both in the living, and in dry bone specimens. The significance of this venture is not simply to gain a better understanding of the process, the expression, and the transmission of the infection. The aim is to discover clues that, together with numerous provenance theories, can be employed in order to reach a sound conclusion which accounts for the multitude of factors that have confounded work in this area.

ETIOLOGY, EPIDEMIOLOGY, AND TRANSMISSION OF SYPHILIS

Syphilis is caused by Treponema pallidum, one of a small group of treponemes, which are virulent to humans, and which are members of the order Spirochaetales (Holmes 1990:205). The discovery of Treponema pallidum in syphilitic material was made by Schaudinn and Hoffman in 1905 (Wintrobe 1974:876). Treponemes have since been classified “a genus of spirochetes, some of them being pathogenic and parasitic for man and other animals, and including the etiologic agents of pinta (carateum), syphilis (pallidum), and yaws (pertenue)” (McCullough 1982:692). The term spirochaeta is defined by McCullough (1982:633) as “a genus of bacteria found in fresh or sea water slime, especially when hydrogen sulfide is present.” Interestingly, the presence of hydrogen sulfide (which is an ill smelling poisonous gas) completes the breeding potential of spirochaeta.

The advantage provided by the presence of hydrogen sulfide, and how it functions to intensify the breeding of the treponemes is unfortunately not mentioned in the literature. Further study regarding this microbiological reaction would be beneficial to an understanding of the transmission of treponemes to humans.

Treponemal infection causes four clinically different diseases which are distributed throughout the tropical and temperate areas of the world. The issue of whether they are caused by four differing species of treponemes is still somewhat confusing. Wasserheit (1991:19) has concluded that humans are the only natural host for Treponema pallidum, but Wintrobe (1974:876) reveals that the pathogen can not only infect humans, but also higher apes and rabbits.

Presently, syphilis is primarily a venereal disease that spreads from person to person during sexual contact. Endemic syphilis, on the other hand, is considered a disease of poor rural populations, specifically those which tend to be over-populated. It is acquired innocently, by sharing a drinking cup or by infection through a flesh wound. Its epidemiology seems to governed by what Willcox (1964:265) calls “two vicious circles.” The first circle begins with poverty, which allows the disease to breed from overcrowding, lack of clothes and washing facilities, dirt, flies and so on. The second circle starts when children become infected. They tend to become infected from close association with their own family and from play with neighbouring families. A family may be introduced to the disease through other children, or it
can spread to a child from an adult who has contracted it by either non-sexual or sexual contact. Syphilis that is transmitted through sexual contact is known both as venereal syphilis and acquired syphilis. A female that has been infected by venereal syphilis can pass the organisms on to her fetus such that the infant is born with the disease. This type of syphilis is called congenital syphilis. Apart from mode of transmission, venereal syphilis differs from the congenital form of syphilis in that it normally commences after the age of puberty. None of the treponemes have ever been cultured in vitro, and “no convincing morphologic, antigenic, or metabolic differences between them have been discerned” (Wintrobe 1974:876). The treponemes are primarily distinguished according to the clinical syndrome they produce.

Treponemes enter the body through the skin or through mucous membrane near the surface of the skin. In the case of venereal syphilis, the treponeme enters in areas of sexual contact, especially the genitals. In the case of endemic syphilis, as in the case of yaws, infection is less specific, entering the body anywhere on the surface by intimate contact. Syphilis can be contracted in this manner as early as childhood, especially in places of the world where children play together naked. The venereal form of syphilis, however, is intermittent and is thought to have directly followed habitations and colonizations of urbanized white races (Ortner & Putschar 1985:180). Once the initial infection begins, there is an incubation period which last anywhere from ten to ninety days before any of the first symptoms of the disease appears.

The Natural Course of Untreated Venereal Syphilis

Primary

Once syphilitic contact has been made, distinct signs and incubation period symptoms begin in three characteristic stages: primary, secondary, and tertiary. The first sign is a chancre or an ulcer, which seems to be a local tissue reaction found at the site of contact. Even when the chancre goes untreated it can often spontaneously disappear within three to eight weeks leaving a slightly noticeable scar. Within six to eight weeks after the initial appearance of the chancre, the patient often develops the symptoms of the secondary stage of the disease. These symptoms can sometimes be delayed up to a year and in some cases even longer.

Secondary and Early Latent

During the secondary stage, there are subsequent tissue reactions to the infection which are analogous to many reactions caused by a number of bacterial invasions. The secondary stage lasts only for a short time before the patient manifests the symptoms of the early latent stage. Ironically they seem free of all signs and symptoms with the exception of a periodic skin rash. During this phase of syphilis, both during the secondary and the early latent stages, the patient is highly infectious. The early latent stage is the most dangerous phase of syphilis because those infected are able to pass on the infection to others while they themselves seem quite free of symptoms. Once the patients enter the late latent phase, they may not encounter any further irritation. This simply means that the disease has settled into its chronic phase. At this point, physical contact with others is again harmless since those infected are incapable of passing on the disease. Periodically they may experience discomfort, headaches, a sore throat, mild fever, and in 75% of the cases, a skin rash still makes the odd appearance (Cartwright 1972:55).

Tertiary

Between three and ten years after the primary infection has manifested itself, the signs of tertiary syphilis begin. The infection becomes powerful and concentrated, and is able to attack almost every system in the body. Typically, lesions and gumma can appear anywhere in the bones, the heart, the throat, and on the skin.

Specifically, the toxicity of the treponeme attacks the epiphyses of the long bones. This reaction customarily frequents the bones of the upper extremities. Epiphysitis or osteo-chondritis results, and can be noted radiologically as, “a jagged ‘saw toothed’ appearance in the temporary zone of calcification in the distal metaphysis” (Willcox 1964:234). Ossification lines widen and become irregular as a result of, “dense granulomatous infiltration which results in incomplete formation of bone” (Cartwright 1972:234). Necrosis and fatty degeneration develops and appears to the naked eye as a yellow line. Often what follows is a disintegration of the bone and incomplete fracturing, or epiphyseal separation. As the disease progresses, syphilitic periostosis begins, appearing as periosteal cloaking, in the X-ray (Willcox 1964:234).

The most common sites of tertiary syphilis are the skin, and the skeletal system. Also, as Wintrobe (1974:880) points out, signs show up in the mouth and upper respiratory tract, larynx, liver, and stomach. As a result of non-specific histological change, diagnosing late benign syphilis is best undertaken and confirmed by serological testing.

Within five years of the onset of the tertiary stage, the majority of those who go untreated die. Cartwright (1972) explains that those who die at this point, tend to show the first signs of general paralysis of the insane (GPI). A number of those who develop GPI never become outright insane, but the disease process affects the brain in such a way that it changes their patterns of behaviour (although most are capable relatively normal lives) (Cartwright 1972:56). When the brain is affected, as it is with GPI, the personality changes that are associated with GPI can lead to suffering that is known to expand into helpless manic behaviour. Further
associated symptoms of GPI include painful joints and neurasthenia.

Additional symptomatic changes are present in the blood vessels and nervous system. The blood vessels easily become weakened, and their walls can balloon, rupturing the aorta, or even one of the vessels in the brain, causing death (Cartwright 1972:56). When the nervous system is affected, it causes a condition called tabes dorsalis. Eventually the process of tabes dorsalis leads to paralysis and incontinence. The usual symptoms associated with the affliction to the nervous system are light pains, impotence, adult epilepsy, and in those under the age of 50, apoplectic stroke (Cartwright 1972:56).

In surveying the clinical literature previous to Fleming’s discovery of penicillin, the cases of syphilis that involved skeletal manifestations only range between ten and twenty percent (Baker & Armelagos 1988:704). It is possible, however, that asymptomatic bone lesions during early syphilis go undetected, and as a result, the presence of bone lesions may actually be underestimated. The skeletal involvement in venereal syphilis most often affects the cranial vault, the nasal area, and the tibiae. Cumulatively, these three locations comprise 70% of all tertiary syphilitic bone lesions (Baker & Armelagos 1988:704). Periostosis during the secondary stage of syphilis is common but these changes are considered transitory and seldom leave any permanent alteration on the bone (Ortner & Putschar 1985:182).

When dealing with exhumed skeletal material, it is, of course, imperative to remember that the roots of plants, fungi, insects, and a host of other factors, can often produce lesions on the bone. Rothschild and Martin (1993:46) note that there have been cases where cranial lesions caused by beetles have been misdiagnosed as syphilis. Furthermore, such lesions allow for the leaching of elements from the bone itself, and also open the way for the introduction of other elements from the burial environment.

Tertiary Syphilis: Skeletal Indicators

At this point, the main interest will be those lesions that affect the skeleton during the tertiary stage of syphilis. Syphilitic bone lesions of the tertiary stage develop between two and ten years after the initial infection. It seems that all tertiary bone lesions are distinguished by an excessive osteosclerotic response to the infection of the skeleton. Changes in the bone are commonly a result of one of two things: either chronic nongranulomatous inflammation, or granulomatous (gummatous) processes (Ortner & Putschar 1985:182). Skeletal gummas are most frequently associated with the long bones of the legs, but they can affect any bone in the body.

The Long Bones

As mentioned previously, the tibia is the long bone that is affected most. It is, in fact, affected ten times more often than any of the long bones (Ortner & Putschar 1985:197). The lesions of the long bones are separated into the categories of nongummatous and gummatous osteoperiostitis. The nongummatous lesions are considered suggestive, but not necessarily diagnostic, of treponemal infection.

Localized nongummatous periostitis has been known to leave plaque-like exostosis which is elevated on the cortex of the bone. Ortner and Putschar (1985:197) note the possibility of “circumscribed build-up of [the] surface parallel lamellar bone of varying thickness and density on bones immediately beneath the subcutis” (the anterior surface of the tibia). Moreover, periosteal thickening is present in combination with cortical thickening. The endosteal surface and the periosteal build-up can often fuse with the old cortex. Generally speaking, nongummatous osteoperiostitis leaves an entire bone thick and heavy. The surface of the bone often looks rough and hypervascular (Ortner & Putschar 1985:197). Eventually, the nongummatous periostitis can cause sclerotic trabeculae to obliterate the medullary canal, while the old cortex may be loosened as a result of Haversian resorption such that, “on cross-section, the bone appears uniformly coarsely cancellous with loss of the distinction of cortex and medulla” (Ortner & Putschar 1985:197).

Gummatous osteoperiostitis is the type of lesion which is even more characteristic of syphilis. This lesion results in tumor-like enlargements of the bone that it affects. In the case of dry bone, the hypervascular periosteal bony build-up surrounds a ‘scooped-out’ defect, extending into the cortex” (Ortner & Putschar 1985:197). In this case, the ‘scooped-out’ lesions are diminutive and clustered, and resemble the caries sica that is characteristic of the cranium, only in this case, the lesion is larger. In extreme cases of the more sparsely distributed manifestation of gummatous osteoperiostitis, large weakened areas can be observed on the mantle of hypervascular periosteal bone, and deep cortex can be exposed such that it is easy to view (Ortner & Putschar 1985:197).

The Joints

Although the joints are not a direct area of syphilitic expression, they can be affected secondarily. Syphilitic arthritis is often resultant and visible in the shoulder, knee and elbow joints. This arthritic joint affection is commonly referred to as Clutton’s joint. Unfortunately, for the archaeologist, this in not visible in dry bone, and thus is not helpful in discerning a syphilitic presence. The features that are present in the joints tend not to be diagnostic of syphilis but, instead, of any of a number of arthritic afflictions. The one truly syphilitic feature that is diagnostic in association with the joints is the presence of gummatous bone lesions.

The Skull

Tertiary syphilis is frequently expressed in the skull. It is particularly evident in the cranial vault, and
in the perinasal area. The cranium can display concentrations of tertiary gummatous and osteoperiostitic lesions on the frontal bone. These lesions can, and often do, spread to the parietal and facial bones, and on to the lambdoidal suture. Gummatous lesions on the cranium are commonly called caries sica.

The lesions that appear on the cranium are not just superficial, as they can often lead to the destruction of the outer table, yet somehow avoid the inner table completely. In the case of untreated syphilis, individual foci will heal, but new foci will form in the area. The healed caries sica leave a depressed, sclerotic, radially grooved stellate scar (Ortner & Putschar 1985:190). As this process continues to form and heal, after time pitting will result. The pitted area will be surrounded by an ivory-hard reactive area, segments of which will have a hypervascular surface. In advanced caries sica, the afflicted area will persist, thickening and becoming markedly more sclerotic over time.

In syphilitic skulls found in Europe, sequestra can be found, often exhibiting a darker discoloration of the necrotic bone. This is caused by exposure of the affected bone through scalp ulcers. The sequestra are diagnostic, due to their worm-like appearance. Their presence is evidence of the disease process, and the one feature that is directly indicative of syphilis.

The facial bones can be affected during the tertiary stage, particularly the nasal bones, the bony nasal septum, the hard palate, the turbinates, and the medial walls of the maxillary antrum. These thin bones are usually obliterated completely in a way that is analogous to the effects of leprosy. The destruction of the ethmoid bones results in syphilitic saddle nose. In a dry skull, however, the mid-facial region looks empty. This differs morphologically from leprosy in that in syphilis the frontal bone is affected in association with nasal destruction. In the nasal septum and hard palate, "the sclerotic response is marked and the inferior nasal spine may be spared" (Ortner & Putschar 1985:192).

**Common Misdiagnoses**

Cartwright (1972:55) regards syphilis as 'the great mimic'. The name is fitting since it can often been mistaken on superficial examination for a number of other diseases. Symptoms such as osteonecrosis, producing the expanded appearance along with circumferential periosteal elevation, is not specific to syphilis. It has also been noted in sickle cell anemia, tuberculosis, yaws, and small pox (Rothschild & Martin 1993:163). Furthermore, neuropathic arthritis in the feet and in the ankles is naturally quite similar to severe destructive arthritis (Rothschild & Martin 1993:156). Ortner and Putschar (1985:180) explain that analogies between the bone lesions of venereal syphilis, endemic syphilis, and yaws, are so similar that diagnostic differences of individual lesions cannot be elicited with any certainty. Given their similarities, yaws and syphilis are mainly differentiated by the worm-eaten appearance of the skull that is characteristic of syphilis.

**Non-Venereal Treponematosis**

Non-venereal treponematosis are endemic in areas of the world that lie between the Tropic of Capricorn and the Tropic of Cancer. Yaws, pinta, and bejel are caused by treponemes that are both morphologically, and antigenically identical to Treponema pallidum (the cause of syphilis), but are clinically and epidemiologically distinguishable from syphilis (Wintrobe 1974:886). Pinta, caused by Treponema pertenue, is an affliction affecting the skin alone, and was once restricted to tropical America. Endemic syphilis was once frequent among the indigenous populations of the North African subtopics, the Near East, and the temperate areas of Asia. Endemic syphilis has not been known to flourish in the Americas. Yaws, on the other hand, is more plausibly misdiagnosed as syphilis, as it too affects both skin and bone.

**Yaws**

Yaws, caused by treponema pertenue, is prevalent in equatorial regions. Infection with yaws may possibly give immunity to syphilis (Holmes 1990:4). There have been descriptions of cranial periostitis assumed to have been caused by yaws that date back to 834 A.D. from the Mariana Islands in the western Pacific (Holmes 1990:4). Yaws is not a condition that has a congenital expression. It is ordinarily acquired during childhood and the most active lesions are noted in children and teens. For this reason, yaws is similar to, and sometimes confused with, congenital syphilis. The bone lesions that are visible in yaws only occur in about one percent of yaws patients (Ortner & Putschar 1985:180). The most frequent locations of lesions are in the long bones of both the upper and lower extremities, the hands, feet, skull, ribs, pelvis, and clavicle. The tibia has a tendency to bend (boomerang leg), leaving the lower leg looking very similar to sabre tibia. This is also a trait found in congenital syphilis that becomes apparent before the age of fifteen. Although much less frequent in yaws, there is evidence of destruction of the nasal bones, the nasal septum, and the hard palate. The expression of yaws on the frontal bone of the cranium shows shallow pitting, as opposed to the expression of caries sica that is seen in syphilis. Yaws is analogous to tertiary syphilis in that gummatous periostitis and osteomyelitis are both apparent.

**Bejel**

The causative organism of bejel is indistinguishable from Treponema pallidum. Bejel is a chronic, non-venereal treponemal infection of childhood and is characterized by early mucous membrane or mucocutaneous lesions. The latent period of bejel is indeterminate and can include complications of gummas on bone and skin. Bejel exists throughout the arid
regions of the Arabian peninsula, Middle Eastern countries, and North Africa. The first sign of bejel is a mucous patch which in some ways resembles condylomata of secondary syphilis. The treponemes are abundant in the moist early lesions and in aspirates from the regional lymph nodes. Lesions, referred to as late lesions, which resemble the lesions of late benign condylomata of secondary syphilis. The treponemes are abundant in the moist early lesions and in aspirates from the regional lymph nodes. Lesions, referred to as late lesions, which resemble the lesions of late benign condylomata of secondary syphilis. The treponemes are abundant in the moist early lesions and in aspirates from the regional lymph nodes. Lesions, referred to as late lesions, which resemble the lesions of late benign condylomata of secondary syphilis.

As such, it resembles that of venereal syphilis so closely that it complicates the diagnostic process and increases the possibility of misdiagnosis. The presence of the sabre tibia is frequent, often presenting itself as being longer than normal with a true forward curve. In this case, the fibula remains normal in both configuration and length. The difference between the sabre tibia in venereal syphilis and the sabre tibia in congenital syphilis is that the tibia length in the venereal form is not considered to be truly elongated and curved, which is to say that the posterior contour of the bone remains straight.

Skeletal changes in congenital syphilis seem to be more indicative than diagnostic, and as Ortner and Putschar (1985:201) point out, they should be critically evaluated within the context of the skeletal findings since lesions can be difficult to distinguish from tuberculosis and other infectious disease.

**HISTORICAL SOURCES**

With the absence of adequate skeletal evidence of syphilis, medical historians have been forced to turn to ancient and medieval writings in an attempt to establish the presence of syphilis in the Old World. Prehistoric evidence has played an important role in documenting the antiquity of some diseases including syphilis, tuberculosis, and several others. The evidence, in many cases, however, has been ambiguous.

**Egyptian**

By the end of the nineteenth century, a number of significant archaeological discoveries were made including medical artifacts, hieroglyphics, and mummies. These artifacts were successful aids in elucidating the role of sexually transmitted diseases in ancient Egypt. Between 1907 and 1911, G. Elliot Smith and F. Wood Jones extensively undertook the anthropological and pathological phases of the paleopathology of Egypt (Holmes 1990:3). The two men investigated skeletal remains taken from an area of Nubia which was flooded by the Aswan Dam. They worked with skeletal materials ranging from the pre-Dynastic through to the Byzantine era. The skeletal materials that were available seemed to show that syphilis was unknown throughout the given periods. Since their work, subsequent studies consisting of X-ray examinations of Egyptian mummies have further shown the absence of any definite cases of syphilis (Holmes 1990:3). This, however, does not necessarily indicate that syphilis was not present. The findings may have been more the result of their sample size. Information regarding sample size was not given in the literature.

**Greek**

By 460-370 B.C., Hippocrates had written the following description which may refer to venereal syphilis:

Congenital Syphilis

Congenital syphilis is transmitted transplacentally to the fetus. The result is spontaneous abortion during the first half of the pregnancy, still birth, or the birth of and infected infant which, in some cases, may not express syphilitic symptoms for many years. Characteristic skeletal change is almost always in the form of syphilitic osteochondritis as a result of haematogenous distribution of *Treponema pallidum* within the fetus in utero (Ortner & Putschar 1985:198). This uterine distribution of the treponeme leads to alterations in all areas of endochondral growth throughout the skeleton. It is most marked in the faster growing metaphyses: the distal femur and the proximal tibia. The lesions of congenital syphilis are calcified cartilage accumulations “in the adjacent area of lucency due to poor bone formation” (Ortner & Putschar 1985:198). It may be the result of syphilitic granular tissue forming in the area, or it may simply be a toxic effect on the endochondral ossification.

Congenital syphilis can be distinguished from many of the other treponemal infections by the presence of notched, peg-shaped Hutchinson’s incisors and mulberry molars (moon’s molars) (Walter 1971:188). This reaction is thought to be the result of infection to the tooth germ during fetal life.

Lesions to the skull appear as rounded and destructive foci but lack the characteristic features of the caries sica typical of venereal syphilis. It seems that in adolescents, congenital syphilitic bone lesions are analogous to those of adult venereal syphilis. Distinguishing between the later stages of congenital syphilis and venereal syphilis is thought to be impossible without the aid of clinical data since they are so morphologically similar.

When congenital syphilis is expressed in an adolescent, the involvement of the bone is neither as frequent nor as generalized as it is in the infant period.

By 460-370 B.C., Hippocrates had written the following description which may refer to venereal syphilis:
Many had apththae and sores in the mouth. Fluxes about the genitals were copious, sores, tumors external and internal; the swellings which appear in the groin; watery inflammation of the eyes, chronic and painful. Growths of the eyelids, external and internal, in many cases destroying the sight which are called figs. There were also other growths and other sores, particularly in the genitals. (Holmes 1990:4)

There is no doubt that this could refer to a number of sexually transmitted diseases, including gonorrhea, but it should not be discounted as a description of syphilis.

By 300 B.C., the earliest known documentations of Lepromatous leprosy were made by the Alexandrian medical school. They referred to it as “elephantiasis because of the thickening and corrugation of the skin” (Baker & Armelagos 1988:706). Baker and Armelagos (1988:706) believe that this was a result of an inexact translation, the term leprosy, as used in the bible, could therefore refer to syphilis. By about 30 A.D., Celsus had mentioned ulcers of the mouth, nose, and tonsils. Many scholars feel that his reference is to Hutchinson’s teeth (Baker 1990:4).

**Biblical**

A number of nineteenth century writers regard certain passages of the Bible as references to syphilis, most notably the plague of Egypt (Exod. 11:1), the disease which attacked the Philistines (I Sam. 5 and 6), and the plague of Baal-peor (Num. 25:8). In the early twentieth century, Hebrew scholars, such as Preuss, refuted these claims, pointing out that the number of deaths due to the plague at Baal-peor was reported to be 24,000, far too high a number to be attributable to syphilis (Holmes 1988:706). Baker and Armelagos (1988), however, have presented a series of biblical quotes that they feel could be references to syphilis. They consider the following quotation from Jeremiah 31:29, “the fathers have eaten sour grapes and the children’s teeth are set on edge,” to be a reference to Hutchinson’s teeth (Baker & Armelagos 1988:706). They also see syphilis in the condition put forth in Exodus 20:5, where “the iniquity of the fathers” is visited “upon the children to the third and fourth generation” (Baker & Armelagos 1988:706). Syphilis is inheritable only by the second generation but the passage is still considered relevant because “syphilis can be passed from one generation to another” (Baker & Armelagos 1988:706).

**Roman**

In the second century A.D., Galen differentiated between dry ulcers and the moist ulcerating tubercles analogous to mucous patches. This type of lesion was described by the ancient Greeks, and also by the Romans, as looking like mulberries or figs. Some have interpreted this as genital condylomata, which is diagnostic of syphilis (Baker & Armelagos 1988:706).

**Medieval**

Hudson (1963) points out that, during the medieval period, Saracen ointment, an early medicine containing mercury, was used by “lepers.” Hudson considers these lepers to be syphilitics who were misdiagnosed. His reasoning is that mercury has no effect on leprosy but it was “the mainstay in treating syphilis until the early 20th century” (Baker & Armelagos 1988:707). The generic term lepra was not used until the eighth century A.D.. Prior to this, this term was blended with the biblical concept of impurity and accordingly, acquired the stigma that is still attached to the word ‘leper’. Thus, the medieval diagnosis of leprosy, may have been an amalgamation of a number of afflictions including both leprosy and syphilis (Baker & Armelagos 1988:707). During the medieval period, many scholars referred to both “venereal leprosy” and “hereditary leprosy.” This is interesting, since leprosy is neither sexually transmitted nor hereditary. This confusion between leprosy and syphilis ended once genuine leprosy became less common, and syphilis began to be widely recognized as an affliction in its own right.

**TREPONEMAL DISEASE AND THE ORIGINS OF SYMULUS**

There are several theories that attempt to trace the origins of syphilis and account for its presence in both the Old and New World. Most of the theories put forward can be divided into three categories: the Unitarian Hypothesis, New World origin, and Old World origin. The fundamental problem with attempting to confidently delineate a theory as to how syphilis might have originated in New and Old World populations is that the body of knowledge on which we can draw to create an acceptable hypothesis is limited to pathological remains, ambiguous historical documents, and evolutionary theory. This problem is especially true for the New World origin theory, which is limited by the nature of skeletal remains. New World skeletal populations lack unambiguous identification of treponemal disease and accurate assessments of pre-Columbian archaeological age (Ortner and Putschar 1985:205). Thus far, there has not been a single theory which has shown to be sufficiently well-evidenced and convincing. A number of theories and a critical assessment of each will be presented, with the intention of arriving at a formulation for the origin and spread of syphilis which can be defended on the basis of the evidence I have presented thus far.

**The Unitarian Hypothesis**

The Unitarian hypothesis likely first presented in 1936 by Krumbhaar. His opinion was that syphilis existed in both the Old and the New World as far back
as prehistoric times. Hudson, who was to become the major proponent of the Unitarian hypothesis, later elaborated on this idea, as did Stewart and Spoehr. Hudson states that syphilis evolved with human populations and was present in both the Old and the New worlds at the time of Columbus’s discovery of America. Hudson further proposes that the four treponemal infections of yaws, pinta, endemic, and venereal syphilis, evolved simultaneously with humans. Each of the syndromes is seen as a biological gradient influenced by a variety of social and environmental factors which produce differential manifestations of treponematosis. Hudson emphasized the evolutionary relationship of yaws, pinta, endemic syphilis, and venereal syphilis, and regarded them all as varieties of one disease caused by one organism: *Treponema pallidum* (Holmes 1990:5). He felt the treponeme had evolved from saprophyte (a microorganism that is closely related to the treponemal organism) early in human evolutionary history, having been introduced via a break in the skin.

Hudson argues that treponematosis originated in the Paleolithic period as a childhood disease that was transmitted by skin-to-skin contact in sub-Saharan Africa where the climate is hot and humid. The implication is that the initial manifestation of treponematosis was yaws. The infection, according to Hudson, followed hunter-gatherers in their migrations throughout the world. As the groups moved into the drier areas that bordered the tropics, the treponemal activity migrated to moist areas of the body, and hence expressed itself as endemic syphilis. Endemic syphilis then moved to the New World from the Old World with the early migrants. The migration of New World populations into the tropical areas of the New World led to the manifestation of yaws once again. It was the unsanitary conditions of the crowded villages that facilitated an increased frequency of child-to-child transmission of the disease.

In the Old World, by the fifth millennium B.C., water, washing, bathing with soap, and separate sleeping quarters were made more accessible in the urbanized areas of Egypt and Mesopotamia. This new concern for hygiene created adequate barriers against the transmission of treponematosis by casual contact amongst children. The result of this was successful sexual maturation without the contraction of treponematosis. As a result, sexual contact became “the only personal contact of sufficient intimacy to permit the transmission of *Treponema*” (Baker & Armelagos 1988:704). Hudson feels that adults began to disseminate the disease through prostitution and promiscuity. Stewart and Spoehr have also speculated that treponemal disease existed in both the Old World and the New World, but they insist that different strains developed in these areas which were cultivated in isolation of each other. They feel that when European explorers made contact with the indigenous peoples of the Americas, they traded strains of treponemal organisms to which neither had developed an immunity. The result was a virulent syphilis epidemic in Europe upon the return of Columbus, and an increase in the expression of bony lesions in post-Columbian Native skeletons from the New World.

**Unitarian Hypothesis - Criticisms**

Jankauskas and Saul (1989) state that Hudson’s assumption of treponematosis originating in sub-Saharan Africa and its subsequent spread with human migrations depends on “the incorrect assumption that treponematosis is a single disease that is differentiated only by environmental factors” (Jankauskas & Saul, 1989:482). The feeble reasoning that Jankauskas and Saul (1989:482) use to dispute the claim of Hudson is that, “Caribbean immigrant children with yaws do not exhibit changes in clinical symptoms on arrival in the United Kingdom.” The assumption is that adaptations which happen on an evolutionary scale are expected to work “on arrival” in a new climatic area. This claim is simply too far reaching. For example, if someone with influenza were to get on a plane and travel to another climate would it make the influenza disappear ‘on arrival’?

Baker and Armelagos (1988:718) also argue against Hudson’s theory, saying that if treponematosis evolved with the Homo genus, and was among the diseases described in the ancients writings, then “skeletal evidence suggestive of treponematosis should be abundant in the materials recovered from Old World sites.” They go on to say that the case for pre-Columbian syphilis in the Old World rests on ambiguous descriptions and therefore must be rejected. Baker and Armelagos present a compelling argument. If this Old World case they refer to is resting on ambiguous descriptions then they are right, and it should not be submitted as evidence for syphilis. The problem with their refutation of the Unitarian theory, however, is their claim that evidence of treponematosis should be abundant in Old World sites. In biblical times lepers were banished, and sent to live in leper colonies until they died. If there was a concern for the possibility of contracting syphilis (the accounts of their separation from the rest of society imply this), then it seems fitting to assume that those who were afflicted would die and be buried within the confines of the colony. If this were the case, then no Old World sites would show signs of this condition unless one of these sites that Baker and Armelagos are referring to is the leper colony itself.

**New World Origin**

From 1490 onwards, something which appeared to contemporary writers to be a new disease, made a sweeping appearance in Europe. It is thought by many that after its introduction into Europe, the disease went on to spread into India, China, Japan, and eventually, to the rest of the world (Cartwright 1972:58). It seems that this pandemic began soon after Columbus’s return from Haiti in 1493. The sudden appearance of this disease and the high morbidity rates of 1494 led to the theory that a highly virulent strain of *Treponema pallidum* had been
occurred in Europe also coincided with the invasion of Italy by the army of Charles VIII of France in 1494. It is believed that the army contracted the disease during their attack on Naples. The army was not, in fact, French, but was composed of French, Spanish, Polish, German, Swiss, Hungarian, and English mercenaries. As an increasing number of soldiers fell sick, Charles was forced to withdraw and abandon his attempt at the conquest of Northern Italy. As the story goes, the army disbanded and returned to their native lands, thus spreading the disease throughout many parts of Europe (Cartwright 1972:58). Cartwright (1972) refers to this theory more as a legend than he does as a valid theory, but few authors have bothered to discuss the authenticity of this claim.

**New World Origin - Criticisms**

Ortner & Putschar (1985) note that Dennie (1962:204) advocates the New World Origin theory for the introduction and spread of syphilis in Europe. Dennie’s theory is that syphilis was particularly virulent after its initial introduction from the Americas through Columbus, and subsequently became milder. He argues that this transition from virulence to milder expressions of the treponeme reflects the normal progress of a newly introduced disease. Dennie, however, fails to define what he considers to be a new disease. His claim could also be taken to support the claims of Stewart and Spoehr and the Unitarian Hypothesis which also supports the introduction of a new disease into Europe, as discussed above. The two theories only differ in that Dennie fails to acknowledge the possible existence of a different strain of the treponeme already in place in Europe. The mixture of a new strain with the American form of the treponeme could have caused a reactionary disease in both the Americas and Europe in such a way, as to take on a virulent form that resembles neither.

Susan Saul (1989), an anthropologist and infectious-disease epidemiologist at the University of California, points out that the treponemal agents of yaws and syphilis are not interchangeable. She states that treponematoses is not a single disease and that recognition of this supports the Columbus theory (Jankauskas & Saul 1989:481). Her point here, however, is problematic. That syphilis is caused by *T. pallidum*, yaws by *T. pertenue*, and pinta by *T. carateum* is widely accepted. As mentioned earlier, Wintrobe (1974:886) points out the real issue, that the treponemes are distinguishable on clinical and epidemiological grounds, while morphologically and antigenically they are identical. When dealing with cases of dry bone specimens, analysis can rarely go beyond the morphological level in the majority of cases. It is on this level that the treponemes are identical and, as such, this is often the only thing which archaeologists have to go by. Thus, the fact that the family of treponemes are different does not support the Columbus theory any more than it discredits it.

Baker and Armelagos (1988:703) assert that their review of the literature strongly suggests a New World origin for syphilis. They say that “there is a vast array of skeletal evidence indicating the presence of a non-venereal form of treponemal infection in the Americas prior to Columbus’s arrival.” It seems that, if anything, this assertion proves Hudson’s Unitarian Hypothesis better than it does the New World origin theory. Hudson acknowledges the presence of treponemal infection in the New World as well. Its presence in the Old World, however, has been easier to recognize. Therefore, if it can be conclusively stated that the treponeme existed in the New World, then Baker and Armelagos should support the Unitarian theory.

Another concern is that many proponents of the Columbus theory discount references to syphilis in European historical documents from Hippocrates through to the Middle ages (Wintrobe 1974:876). Also, as Cartwright (1972:60) asserts, there is no evidence whatsoever of the disease in the imported Indians or among the forty-four seamen who were on board with Columbus; “the homeward voyage seems to have been remarkably healthy.” Cartwright goes on to say that it may be of some significance to note that the Columbus theory did not achieve any popularity until over a quarter of a century after Columbus’ return and the alleged first appearance of the disease.

Schendel (1968:103) has offered a unique and alternative interpretation to the origin debate. He asserts that syphilis originated in none of these places, and was brought to both continents from the Orient. He contends that syphilis was brought from Asia by prehistoric Indian tribes during their migrations across the Bering Sea.

**Old World Origin**

Cartwright (1972:60) notes a theory which holds that syphilis originated in Africa and was introduced into Spain and Portugal by imported slaves. Yaws, the African strain of treponematoses, is chiefly transmitted by non-venereal contact, and is seen only in hot climates where it begins as a skin eruption. If this same treponeme were introduced into cooler climates, it would settle into a venereal transmission of syphilis due to the fact that people are covered by clothing in cooler climates. As such, yaws and syphilis are probably different manifestations of the same disease.

Ortner and Putschar (1985:204) note Hyde (1891) who states that the ancient medical literature of China, India, Greece, and Italy provides “unmistakable proof
that early in the world’s history genital lesions were known to occur from sexual contact.”

Baker and Armelagos (1988:703) share Hackett and Holcomb’s view which asserts that venereal syphilis was present in Europe prior to the voyages of Columbus. Both parties feel that before this time, syphilis was simply not distinguished from leprosy. “The alleged epidemic resulted from the recognition of syphilis as a separate disease in the 1490’s.”

Cockburn adds an evolutionary framework to this theory and suggests that pre-Columbian syphilis existed in geographical isolation which led to speciation of Treponema. Cockburn feels that treponemal infections existed throughout the history of humankind, but was mild and chronic due to the small population sizes. Once populations expanded, however, more acute infections were selected for, which “spread by direct skin-to-skin contact among children” (Baker & Armelagos 1988:703). He further explains that, by 1492, European standards of living improved to the point where treponemal transmission was differentially affected. The strains that depended on skin contact were at a disadvantage and were replaced by a stronger strain that was sexually transmitted (Baker and Armelagos 1988:703). Thus, according to Cockburn, the discovery of America and appearance of venereal syphilis was more a result of social and economic factors than a case of cause and effect.

Old World Origin - Criticisms

Cockburn’s model for Old World origin uses the same rationalization as Hudson does to prove his Unitarian theory. Cockburn also fails to mention the American expression of the treponeme, focusing instead on the infection in general, which existed throughout the history of humankind. Perhaps this was an oversight, but if he feels it existed throughout time, and existed in all human populations (which is what he seems to state), then there is an obvious hole in his argument. If he were to complete his theory, accounting for the populations of both the Old and New Worlds, his theory would be a replica of Hudson’s and not an argument for Old World origins at all.

CONCLUSIONS

Hart (1983:126) points out that the only way that an accurate origin theory will ever be devised, is if accurate methods of dating and diagnosis are applied to skeletal material. Hart thinks that C14 dating could be usefully applied to treponematosis, although it is hard to conceive of this not having been already attempted. His main concern is based on his impression that few published analyses have been based on both radiocarbon dating techniques and accurate diagnosis. It is true that what we seem to be lacking is conclusive skeletal evidence that is both properly diagnosed, and decisively dated.

Archaeologists are also in need of a serological test for treponematosis that can effectively be applied to dry bone. For pre-Columbian material, basing a diagnosis on morphological traits is extremely difficult, since the material is old and fragile. Moreover, this can be complicated further in specimens that exhibit only a few diagnostic features, or, in cases where the individual has died previous to full blown tertiary syphilis and, as a result, exhibit mild skeletal markings. In cases such as these, serological testing on dry bone would alleviate the problems associated with morphologically ambiguous skeletal material.

The most common application of serological testing used by archaeologists has been to identify blood cell antigens (Isca & Kennedy 1989:253). In Czechoslovakia, however, a method has been developed that has proven to be capable of determining the presence of treponema. Recently, this method was used to identify yaws in bone samples in Australia, and also in identifying syphilis in 16th and 19th century cases from Europe (Baker & Armelagos 1988:719). Hopefully these tests will soon be refined and prove accurate enough to be successfully applied to the many ambiguous cases of treponematosis.

My concern throughout this paper has been to present as much relevant and accurate data as is known about Treponema pallidum and syphilis. The motive was to find clues that may have been overlooked in the theories that have been put forth, with the hope of devising a more accurate theory. It is my opinion that the most convincing theory is the Unitarian hypothesis, which accounts for a multitude of factors.

Hudson begins by stating that syphilis evolved simultaneously with human populations and is present in both the Old and the New World. The main point in the theory is that each of the syndromes that have been produced from the treponeme are mere biological gradients that have been influenced by social and environmental factors. He believes that the treponemal family evolved from saprophyte, which McCullough (1982:607) defines as “any organism living upon dead or decaying organic matter.” Its relation to the spirochaeta genus seems plausible based on its presence in waters which necessitate the presence of ‘slime’, possibly a reaction to dead organisms that thrive in water. If we note that the spirochaeta can exist in any of either fresh or salt water, we can see how this ‘slime’ could well have existed as long as water has, and hence, as long as water has been associated with human life. If this is the case (and I stress that this hypothesis is purely my personal speculation) then it seems extremely plausible that treponemes evolved with humankind just as the Unitarian hypothesis postulates. If the treponemes have basically been bred in water, then any contact with ‘slime’ contaminated waters, especially in terms of a break in the skin, would perpetuate the infestation of humans with the treponeme. This may account for Hudson’s speculation that treponemal infection began with casual contact. The theory also suggests treponemal infections evolved simultaneously with humans, implying that it was contracted by a specific human. If spirochaeta does breed in water, however, it would seem more probable that