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1990-02-06T07:11:34Z
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STRUCTURE AND EXPERIENCE IN THE MAKING OF APARTHEID

6 - 10 February 1990

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TITLE: Tracking Down the Treponema : Patterns of Syphilis in South Africa, 1880 - 1940

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TRACKING DOWN THE TREPONEMA: PATTERNS OF SYPHILIS IN SOUTH AFRICA, 1880 - 1940

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AIDS is the health horror-story of the 1980s. Projected morbidity and mortality rates herald social and economic mayhem in the future if the disease is not brought under control now. Other sexually transmitted diseases (STDs), while less dramatic and curable, have exacted a severe toll on people's health in the past and even still today.

Statistics on the current prevalence of STDs in urban, rural and migrant population in South Africa are patchy, although small-scale studies suggest widespread STD morbidity. A 1969-70 survey of 587 male and female patients attending a major urban black hospital found a prevalence of syphilis of 17% (1). A 1978 study of 186 urban black women attending a family planning clinic showed that 10% of 'well' women had gonorrhoea (2). STDs also afflict a substantial proportion of the migrant mineworker population. The incidence of STDs among mineworkers in 1983 ranged from 7% to 10% on Transvaal and Natal mines respectively (3).

High STD rates are ascribed to promiscuity and immorality by orthodox commentators who embrace a medical model of disease causation and assume that people are to blame for their own illnesses. In contrast, political economy approaches suggest that one must consider 'the relation of ecological, political and social aspects of disease to the economic transformations wrought by colonialism and capitalism in Africa' (4). Political economy approaches to health have illustrated how colonial conquest, coercive labour recruitment and disruption of subsistence production led to a decline in african health which the lack of medical services only exacerbated.

The vulnerablity of migrant worker populations to contracting STDs has been widely documented (5). A recent WHO report suggests that among the factors influencing the frequency of STDs are social problems such as unequal sex ratios, urbanisation and prostitution - all factors evident in migrant labour systems (6). Applying a political economy model to the problem of STDs, Lesley Doyal argues that

> 'the disruption of the economic and personal foundations of family life [by the introduction of a migrant labour system] led to the disintegration of long-established marital and sexual patterns. ...The growth of prostitution represented one form of adaption to the intolerable strains faced by men and women alike' (7).

Men sought out casual sexual partners while away from home, and deserted wives or single women resorted to prostitution as the only means of supporting themselves. STDs, previously unknown in many parts of Africa, became a serious health problem.

AWS 202

While there is undoubted truth to this approach, it is premised on questionable assumptions about precolonial health patterns in Africa. For example, Doyal argues that while pre-colonial Africa was not an idyllic, anti-septic world, it was adapted socially and physiologically to the natural environment (8). But very little is known about the interaction of demography, climate, ecology and disease in pre-colonial Africa. Gwyn Prins points out

> 'it would be unwise to believe that pre-colonial Africa was free of catastrophe, of perinatal mortality or epidemic disease; that somehow, before the Europeans came, there was "Merrie Africa". ...The arrival of Europeans was only one episode in African history. Its qualities imply nothing about the totality of what went before' (9).

This paper seeks to develop a more refined understanding of the spread of STDs in South Africa by evaluating medical evidence of the late nineteenth and early twentieth centuries on the prevalence and characteristics of syphilis. Close examination of government district surgeon reports in the Cape Colony in the 1880s, and the Transvaal in 1906 suggest a more complex epidemiological picture than that proposed by Doyal. While venereal syphilis probably was introduced by white settlers and transmitted from urban to rural areas by migrant workers, a pool of endemic non-venereal syphilis probably already existed in parts of South Africa and its manifestations were often mistaken for venereal syphilis. Only by the 1930s was venereal syphilis evident in urban areas and in previously unaffected rural areas. Today endemic syphilis is rarely seen in clinics. This paper puts forward evidence for the shift in the pattern of disease.

2. The Extent of Syphilis in South Africa

2.1 Settler Origins

There appears to be no written evidence of venereal disease among African populations in the early nineteenth century. David Livingstone who travelled in Southern Africa in the 1840s and 1850s noted that syphilis was common among tribes nearest the coast, but ambiguously stated that 'it dies out in the interior of Africa. ...It seems incapable of permanence in the centre of the country in persons of pure African blood' (10).

Venereal syphilis was introduced into the Cape by sailors, army troops and white settlers and the extent of syphilis was a source of concern in the Cape from the earliest days of white settlement. The deprivation of slave women and later, in the 1830s of freed slaves who were excluded from the white-dominated domestic and laundry sector, forced coloured women into prostitution. So many women recieved treatment for venereal disease at the pauper hospital that the authorities suggested the expense should be offset by their forced labour in prison (11).

The first reports of syphilis in the Transvaal seem to coincide with the opening of the diamond fields in the 1870s. A doctor, recalling his early medical experience in Potgietersrus, said that his african patients traced the disease to workers returning from Kimberley in the 1870s (12). Dr Long, primary medical officer of Basutholand, reported to the 1906 Transvaal Contagious Diseases amongst Natives Commission that 'prior to 1876 the disease was unknown in Basutoland. It was brought there by labourers returning from the Diamond Fields' (13). The disease was unknown to Xhosa and Fingos until the late 1880s. Dr Veale recalled his investigative tour of the Waterberg in 1892 when he found that 'the tribes who had most to do with the whites were badly infected' and africans attributed it to contact with Kimberley. The commission concluded that

> 'Its introduction appears to be of comparatively recent date, having usually been brought to the kraals by natives who had been to work at some mining centre, especially Kimberley, where ... the disease is very prevalent' (14).

African witnesses from various locations in Zoutpansberg testified to the commission in a similar vein. Lucas Molaba 'first saw it in 1884. It was brought by a man who came from Kimberley'. Chief Mohlaba maintained the first case in his community was 'a native who came from Kimberley' and the second 'a woman infected by a Basuto from Johannesburg' (15). African names for syphilis, stated the commission, 'seem to have been recently coined for a newly introduced disease, as they are not to be found in the original languages' (16).

A similar pattern is evident in Natal. Werner commented in 1905 that in Natal 'syphilis was certainly unknown till recently, and the Zulu word for the class of diseases to which it belongs (isimpantsholo) is of recent coinage' (17). The Zulu names for syphilis captured the connection between colonisation, proletarianisation and venereal disease: syphilis was called isifo sabelungu, disease of white men, or isifo sedolopi, disease of the town (18).

2.2 Syphilis in the Cape Colony in the 1880s

Legislative concern over the extent of syphilis in the Cape Colony dates to the Contagious Diseases Act of 1868. The Act was modelled on British legislation and intended to placate the War Office which claimed that British troops at the Cape were being decimated by venereal disease (VD): 13% had been hospitalised for treatment (19). This Act was later withdrawn. When the issue came to the fore in the 1880s, again the extent of the disease among British troops in colonial countries was of uppermost concern. A survey by the Royal Army Medical College of hospital admissions for syphilis in British army in 1887-96 had revealed that while in Britain army admissions for VD were 95,1/1000, in the Cape it was 134,4/1 000 and in India 189,7/1 000 (20). Part I of this second Contagious Diseases Act, passed in 1885, and enacted in the seaports of Cape Town, Port Elizabeth, East London and Kingwilliamstown, provided for the registration and

regulation of prostitutes. The Act also responded to local concern following district surgeon reports on the more serious prevalence of syphilis in the countryside. Part II of the Contagious Diseases Act gave district surgeons authority to place men or women under medical treatment for syphilis. It was only after the findings of select committee appointed to investigate the prevalence of syphilis and methods of control, that Part II was enacted in November 1888. By the time it was countermanded ten months later 1929 males and 1510 females had been treated.

District surgeon reports through the 1880s sounded a definite note of panic about the ravages of syphilis in rural areas. Aliwal North's district surgeon stressed the 'alarming manner in which this disease is spreading in the district' and warned that without some measure of control 'in a short time this country will be shunned by the world as a loathsome place and one to which all must give a wide berth' (21). Witnesses before the select committee of 1888 warned that the future of white and black races hung in the balance as syphilis 'has a very deteriorating effect upon the physique' (22) and 'unless checked it will take the energy out of the whole of your people' (23).

The threat of contagion from black to white provoked particular anxiety. Philipstown district surgeon commented: 'The blacks are the great vehicle for the conveyance and spread of this disease, and it is through them that so much misery is brought into many an innocent family' (24). Farmers dismissed servants or labourers who had contracted syphilis. The disease spread from district to district as infected people sought work with other white families or became thieves and vagrants (25). Among whites it was said to affect poor tenant farmers who spread the disease as they trekked in search of pasturage for their stock (26), but syphilis had also 'crept now into some of our most respectable families' (27). The disease aroused such terrible fears that Oudtshoorn residents felt 'that it has become dangerous to perform the common acts of everyday life, such as shaking hands, receiving money etc etc' (28), and in Cradock 'many people refused to go to communion because they knew people who were suffering from syphilis were there as communicants' (29).

Almost every district reported cases of syphilis. In areas where the disease was infrequent, such as Swellendam, cases were traced to servants brought over from the Karoo (30). But in several areas syphilis appeared to be epidemic. In Barkly West the district surgeon treated 1140 patients in 1883 following the establishment of a free dispensary and out-stations in the reserve. Patients came from Taungs, Kuruman, Sivonels, Douglas, and Hart's River for treatment (31). In Oudtshoorn the district surgeon treated 1074 patients in 1882 and 1003 in 1883 (32). In Richmond the district surgeon felt that it was no exaggeration that 'two-thirds of the coloured inhabitants are affected with this loathsome disease' (33). In Bedford the district surgeon had examined 2044 africans of whom 1122 were syphilitic (34). Carnavon's district surgeon concluded that syphilis was so extensive among africans that 'it can be said that they are to

be divided in three classes, of which the first has had the disease, the second one has got it, and the third one will get it' (35).

Evidence presented before the 1906 Contagious Diseases Commission told a similar story. Syphilis was said to be 'very prevalent' in the Cape Colony and Native Territories 'chiefly amongst the coloured persons and bastard races' and was 'spreading to the purely native races' especially among those returning from Kimberley and the Rand (36).

2.2 Syphilis in the Transvaal - the 1906 Commission

Concern about the extent of syphilis in the Transvaal resulted in the establishment of the Contagious Diseases amongst Natives Commission in 1906. Part of its brief was to examine the prevalence of STDs. It concluded that syphilis was 'very common' in the Pretoria, Western, Waterberg and Lydenberg districts, and 'very seriously prevalent' among the Basotho and Ndebele tribes in the west and south west parts of the Zoutpansberg district. Shangaan and Bavenda groups were 'comparatively free' of syphilis (37). The disease seemed to affect only certain tribes and certain parts of the Transvaal (38) and was more frequent in rural areas that in the towns (39).

Doctors' evidence about the extent of syphilis in their districts provided a grim picture. Dr Borle from the Elim Mission stated: 'The natives tell me that very often most of them in their kraals are infected. ...In some parts you have 1 or 2 per cent, and in other parts 80 per cent' (40). Dr PA Green, Pietersburg District Surgeon, speaking about the Zoutpansberg said: 'From what I have seen I should think that 8 out of 10 natives who consult me come in regard to syphilis' (41). Dr John Joseph Lowell, Potgieterust District Surgeon, estimated that 'nearly 80 per cent of the population suffer from syphilis, either hereditary, tertiary or secondary ...I should say that probably 9 out of 10 patients I see each day are syphilitic' (42). A German missionary, Mr Franz, felt that 'not less than 75 per cent of the western tribes (Basuthos and Matabeles) were syphilitics'. He treated 5000 cases between 1902 and 1906 (43).

2.3 Syphilis in the 1930s

By the 1930s syphilis appears to have spread widely in urban and rural areas and was no longer confined to the northern and western Transvaal and northern Cape. A Department of Public Health annual report suggested in 1925 that syphilis was decreasing in Bechuanaland and northern and western Transvaal and increasing in areas where it was formerly rare such as Pondoland and the eastern Transvaal (44). Natal seems to have been relatively unaffected by fears of epidemic syphilis at the turn of the century but by the 1920s concern over 'low morals' of young girls, premarital pregnancy, and the ravages of syphilis was evident in the activities of the church manyanos, the Bantu Purity League, and in the cooperation between male elders and

colonial authorities to control women's migration to urban areas (45). One study of seropositivity rates showed a 23,6% incidence in an urban group and 23,3% in a rural population in late 1930s (46).

Surveys in particular populations revealed high seropositivity rates. A sample of 500 waiting outpatients and their friends at a Pretoria clinic in 1921 showed that 47,8% gave a positive Wasserman reaction (47). Another survey conducted at an outpatient dispensary in Germiston location tested blood samples from every patient during a six month period in 1938. Of 712 samples, 316 (44,4%) were positive - a similar prevalence to the Pretoria survey (48). These figures may be biased: they are drawn from a population already seeking medical attention and may include STD patients.

Prevalence surveys among apparently healthy populations offer a better indicator of the spread of disease. A 1943 Medical Officer of Health report cites random testing in Kimberley resulting in a 32% positive incidence among abbatoir employees; and from 30% to 43% among other groups of labourers (49). Routine Wasserman tests among women attending ante-natal clinics, a good 'healthy' sample population, also indicated high seropositivity rates. At a Kimberley ante-natal clinic in 1943, 34% of coloured women and 46% african women registered positive reactions (50). A survey conducted among all women attending an ante-natal clinic in Germiston location over a six-month period in 1938 also showed widespread incidence.. Of 227 tested with the Wasserman reaction, 40,5% were positive (51). Among 2828 women tested at an ante-natal clinic in Springs between 1938 and 1944, 23,97% gave positive Wasserman reactions (52). A similar incidence occurs among 715 ante-natal cases at Edendale where 20,1% gave positive Wasserman reactions (53).

Syphilis appeared equally pandemic in rural areas. Several studies suggest prevalence rates were far lower in rural populations. A survey of 235 men attending a chiefs meeting at Flagstaff in 1930, revealed that 12.1% had a positive Wasserman reaction (54). Routine tests of 1184 maternity cases at the Sir Henry Elliot Hospital in Umtata during 1943 showed that 136 (11,4%) were positive on the Wasserman test (55). Routine testing of 57 antenatal cases at St Barnabas Mission revealed a slightly higher prevalence of 19% (56). Prevalence was even higher in Polela where, between 1941 and 1946, of 930 expectant mothers 35,3% were positive (57).

2. Evaluating the Evidence

2.1 Syphilis and Migrancy

Syphilis was clearly a major source of ill-health in South Africa from the 1880s and doctors of the time logically related the disease to the opening up of the diamond and goldfields. Certainly by the 1930s, syphilis could be directly tied to

migrant labour. A study tracing the sources of venereal infection in Polela, a rural village in Natal, showed that most married and single women were infected at home by their husbands or lovers who had recently returned from work in a town. Only two out of twenty male patients were infected by their wives (58). One might expect that a geographical breakdown of the incidence of disease might, as in the case of tuberculosis, be linked to the length and intensity of involvement in migrant labour in particular regions. However the medical evidence seems otherwise.

The Cape district surgeon reports and the 1906 Commission suggest that between 1880 and 1910 the burden of syphilis was highest in the northern and western Transvaal and the Northern Cape and low in Natal and Mozambique. Certainly, it could be argued that the high prevalence in the northern Transvaal is associated with the region's early involvement in migrant labour, but Tsonga workers had a similarly long history of migrancy yet low incidence of syphilis. From the 1840s Pedi men travelled long distances to the Cape farms and public works, while Tsonga men migrated to the Natal farms and sugar fields from the 1850s (59). When the diamond fields opened the following decade Pedi, Tsonga and South Sotho migrants predominated among the 50 000 to 80 000 who visited each year (60). From 1895 South Sotho and Tswana predominated on the diamond fields (61) while Mozambique and the northern Transvaal became the most significant sources of labour for the gold mines (62).

By the 1930s although syphilis was widespread in urban and rural areas throughout South Africa, prevalence still varied geographically. The Witwatersrand Native Labour Association conducted an examination of african mine recruits 1930 and found different prevalence rates in different ethnic groups (63):

Group Examined	Number Tested	% Positive Wasserman Reaction
Transvaal Basuto	200	29,5
British Basuto	200	25,5
Bechuana	100	22
Pondo	200	8,5
Mozambique	300	7
Xhosa	200	2

This does not correspond to the extent of involvement in mine migrant labour. The lowest prevalence rates among the Pondo, Xhosa and Tsonga recruits were in areas which contributed the highest proportion of the the mines labour supply by 1936. Almost 18% of the mine labour force was drawn from the Cape and 28% from Mozambique in 1936. (64). The Cape peasantry had managed to avoid mine recruiting until 1905 but between 1908 and 1910 the labour supply from the eastern Cape increased dramatically (65). By 1910 mineworkers constituted 71% of migrants from the Transkei and Ciskei and these areas contributed almost 30% to the mine labour force (66). The prevalence figures also do not correlate with the proportion of men in the 15-44 age

group involved in migrancy in particular areas in the late 1930s: while 28% of men were migrants in the high prevalence zone of Bechuanaland; 50% of men in Transkei and 70% in Ciskei were migrants, both low prevalence areas (67). Only among Lesotho workers does a high prevalence rate seem to match migrancy rates. Fifty per cent of men were migrants and they constituted 14,5% of the mine labour force (68).

This inconsistency between prevalence and migrancy rates is perhaps explained by careful examination of the aetiology of syphilis. Was the disease syphilis? To assess the South African epidemiological evidence some knowledge of the characteristics of syphilis is required.

2.2 A Comparison of Treponematoses: Venereal or Endemic Syphilis?

Treponematoses is the collective name given to four chronic infections caused by bacteria belonging to the genus Treponema. The agents of venereal syphilis and endemic (non-venereal) syphilis are both T. pallidum and of yaws and pinta are T. pertenue and T. carateum respectively. The treponemes have identical morphology and resemble thin, silver corkscrew threads. At present no laboratory tests exist which can distinguish between treponemes (69) and all treponemal infections reflect positive on the Wasserman test. Thus diagnosis depends on clinical observation.

Venereal syphilis is characterised by three stages. In the primary stage after an incubation period of three weeks a single sore or hard chancre usually appears at the point of innoculation on the genitals. It grows for a few days, then breaks down and forms an ulcer. Lymph nodes in the groin also become enlarged and hard. A period of latency follows after healing. No primary chancre occurs in endemic syphilis but an initial lesion is common in yaws.

The secondary stage of venereal syphilis is characterised by a rash which appears anywhere or all over the body including on the skin of the palms, soles, in the mouth, and around the genitalia and anus. Lesions are contagious, usually painless, do not itch and heal with or without treatment and with little scarring. The lesions are followed by latent periods over three to five years after which they are no longer infectious. Certain lesions concommitant with venereal syphilis also resemble endemic syphilis and yaws lesions. The most common lesions in endemic syphilis are mucous patches which occur in the mouth, and are, as in venereal syphilis, frequently accompanied by hoarseness. Also commmon are secondary-type lesions which prefer moist body surfaces such as axillary and genital areas.

The tertiary stage of syphilis may appear after twenty years of latency only in a third of cases. It is characterised by cardiovascular, neurological and visceral complications. It is unknown whether these occur in endemic syphilis but they never

occur with yaws. Common late manifestations of yaws and endemic syphilis are destructive gummata and ulcers of the nasopharynx, skin and bone which disable and deform an individual but seldom cause death. Late complications occur frequently with endemic syphilis but only in 10% of yaws cases.

The clinical manifestations of the disease described by doctors in the late nineteenth and early twentieth century are suggestive of endemic syphilis. Most doctors said they never saw cases of primary chancre and that all their cases were secondary syphilis. Dr Green mentioned that in the Pietersburg district he had 'never seen primary sores except on two cases' (70) and Dr Lowell too confirmed that while he saw hereditary, tertiary or secondary cases 'not in one case have I discovered a hard chancre' (71). District surgeon for Zeerust, Dr Bishop, also 'had never seen a hard chancre' (72). Doctors also commented that tertiary symptoms were absent. Explained one Cape district surgeon:

> 'I came across no symptoms of tertiary syphilis amongst the crowd of natives I treated some years ago for mucous tubercle ...all the young children, who had mucous tubercle at the same time, have grown up healthy, with apparently no undermining of the constitution. (73)

The predominant manifestation of the disease was secondary lesions. The typical characteristics, explained the Barkly West district surgeon, were 'moist sores in the throat and mouth, on the tongue and lips, under the armpits, and on the lower portions of the body' (74). An Oudtshoorn surgeon explained in greater detail:

> 'The greatest number of cases are of the secondary form, and the principal lesions are in the mouth and throat. In several tertiary cases we had extensive nodules on the tibia and bones of the skull, also ulcerations of the legs, while in others there was destruction of the nasal bones and soft palate. This year skin diseases are more common ...Pustular eruptions form the great bulk...During the whole year we did not observe a single primary sore' (75).

Several doctors argued that the disease displayed none of the classical signs of syphilis and should be classified as a new disease 'mucous tubercle' which more closely resembled yaws of central Africa and the West Indies (76).

The 1906 Commission report does not detail the particular secondary symptoms seen by medical practitioners. However in an article written in the mid-1930s a doctor recalled that while passing through the Potgietersrus area in 1888 he noticed syphilis cases displayed destruction of nasal bones, perforation of the palate, destruction of the skin, gummata of the whole cutaneous surface especially in the gluteal region, and severe bone and joint lesions. Tabes and general paralysis were absent (77). The descriptions of secondary lesions resemble those most common to endemic syphilis.

By the 1930s and 1940s there are reports of primary and tertiary cases. A doctor describing symptoms of syphilis in the Transkei in the 1940s noted that the primary stage was often seen, particularly amongst men and it was seldom extragenital. He had also seen tertiary symptoms such as cardiovascular disease and madness. Tertiary symptoms also appeared among mental asylum inmates where 7% of african inmates in 1936-40 suffered from cerebral syphilis (78). Autopsies on african hospital patients between 1924 and 1938 showed that 5,9% had died of undetected syphilitic heart disease (79). Another six-month study of post-mortems on natural deaths revealed that 12% were due to syphilitic aortitis (80).

Of course the absence of primary and tertiary symptoms may really just reflect the paucity of medical services for africans in urban and rural areas and preference for traditional healers. More convincing indicators which allow one to differentiate between venereal syphilis, endemic syphilis and yaws are geographic occurrence, modes of transmission, age-group most affected and the presence of congenital syphilis.

Hackett argues that the treponematoses are variants of an ancient strain which gradually diversified as variants were geographically isolated and adapted to climatic and human environmental and social changes (81). He suggests that by 1900 an equatorial, sub-Saharan zone of yaws was bordered in the semi-arid regions of the north and south by belts of endemic syphilis and that venereal syphilis predominated along the Mediterranean littoral and in southern Africa (82). Yaws and endemic syphilis occur in rural populations but while yaws is adapted to humid, warm countries, endemic syphilis occurs in warm, semi-arid and arid areas. Neither is endemic in urban population and neither will occur in same population. However endemic and venereal syphilis can occur in the same population. Venereal syphilis is an urban disease and can occur in any climate (83). The evidence cited in the Cape Colony district surgeon reports of the 1880s and the 1906 Commission suggests that the disease, defined then as venereal syphilis, occurred in particular areas where the climate was hot and semi-arid and was mainly a rural rather than an urban problem - characteristics typical of endemic syphilis.

The possibility that the disease was endemic rather than venereal syphilis gains greater credence if one compares the age-groups most afflicted by particular treponemal infections and different modes of transmission.

Venereal syphilis is usually transmitted directly from person to person through sexual intercourse though non-sexual and indirect means have been recorded (84). Endemic syphilis and yaws are not transmitted sexually but do spread through direct contact. Indirect contact through shared contaminated drinking utensils is the most common mode of transmission for endemic syphilis, but rare in yaws. Direct lesion-to-skin contact among children, and contact with fingers contaminated with saliva containing

treponemes is also an important mode of transmission for endemic syphilis (85) but most significant for yaws where overcrowding and poor community sanitation are also probably significant (86).

The peak incidence of venereal syphilis is among adults aged 18-30. The reservoir of infection for endemic syphilis and yaws are children aged two to fifteen and the peak incidence is between two to ten years (87). Endemic syphilis often occurs in family groups: it is acquired by children and spread to susceptible adults. Childhood infections of endemic syphilis and yaws provide cross-immunity against venereal syphilis in adulthood (88).

Cape district surgeons continually stressed their surprise at the number of children who had contracted syphilis and that it was not necessarily spread through sexual contact. Knysna's district surgeon, for example, commented:

> 'Those who see much of syphilis in this district cannot fail to be struck with the number of children and adults who quite innocently, I believe, have contracted the disease ...I may say quite a third of those brought under treatment are virgins, girls whose innocence is beyond a doubt and children, some of whom are only a few months old' (89).

Jansenville's district surgeon had noticed that 'wherever there are locations of natives syphilis spreads rapidly when one case exists, as the children all play together and so contract it one from each other' (90). In Colesberg the disease was 'most prevalent among young children and appears to be increasing in malignity, spreading through whole families' (91). Several doctors recounted cases of how infection of one child quickly spread to other children in the family and to the parents. Washerwomen and nursemaids were cited as the source of infection from black to whites. One doctor recounted how syphilis 'was communicated from nurse to child, the child in its turn communicating it to the mother through the nipple' (92). In all these cases the source of contagion was clearly never sexual intercourse but direct or indirect contact. Doctors traced cases to 'kissing, using the same vessels for washing or drinking purposes, drying with the same cloth after washing (93) and to 'using the same cup or spoon, foul linen, sheets, seat of privies' (94).

A similar pattern of widespread syphilis among children emerges from evidence to the 1906 Commission. Dr Veale, who toured the Waterberg in 1892, recalled: 'It was practically congenital and all the children in the location were syphilised; also all the young girls who went down to carry water' (95). District surgeons for Lydenberg and Middelburg agreed that 'congenital' syphilis was most common.

It seems that african inhabitants of the time recognised that endemic and venereal syphilis were transmitted differently. A district surgeon from Hebron commented in 1889:

'The natives themselves do not look upon the disease as venereal. They have a word meaning venereal disease, which they apply to gonorrhoea and chancres on the penis, but they do not apply it to mucous tubercle, but another word signifying 'the sores'. They look upon 'the sores' much as they do upon measles, that is once the disease is cured it is over and done with' (96).

Witnesses to the 1906 Commission also mentioned that africans in badly affected areas did not associate syphilis 'with the consequences of impure sexual connection' and in some cases tried to isolate infected persons to prevent the disease from spreading (97). This suggests familiarity with the disease (98).

Another clearly distinguishing characteristic of venereal syphilis is transmission to the foetus through the placenta. *Congenital manifestations are unknown in endemic syphilis and do* not occur in yaws. Congenital syphilis occurs especially if a mother contracts syphilis after the fourth month of pregnancy. Spontaneous abortion, stillbirth or premature birth are likely. A newborn baby may appear healthy or have only a sore mouth, and later develop secondary and tertiary symptoms simultaneously. A child may die from a lesion in a vital organ or, if it survives, develops characteristic deformities including protruding brows, abnormalities of upper jaw, saddle nose, saber shins and the Hutchinson triad (peculiarly shaped teeth, blindness and deafness) (99).

The absence of concern about high rates of spontaneous abortion, stillbirth and congenital syphilis in the 1880s and 1906 reports are only surprising if the disease was venereal syphilis. Doctors frequently commented that healthy children were borne to parents who were known to have had syphilis:

'The present generation of babies - there are swarms of them - [do not] suffer in appearance in the slightest from the fact that their parents are supposed to have had syphilis' (100).

But descriptions of disease in young children are sometimes ambiguous. For example, Lucas Molaba of Mpahlela's Location stated in evidence to the 1906 Commission:

'Syphilis has now covered the whole place. Our children die from it. You can scarcely find a woman with a baby in Mpaplela's who is not suffering from syphilis' (101).

And a description of syphilis *among children* by a *medical* missionary working at Jane Furse Memorial Hospital in Sekukhuniland in 1922 hints at two modes of transmission:

'At the present time most of the native children have either been infected before birth or else during the very early years of childhood and most of the terribly bad cases in adults are the result of the disease which first showed itself in early childhood'(102).

The possibility of this being mainly congenital syphilis is unlikely since this doctor still attributed contagion to non-sexual contact:

'Under native conditions of life, where blankets, clothing, pipes, cooking utensils, etc, are all very much common property in a large kraal, it is easy to see how the disease was bound to spread by leaps and bounds quite apart from any question of immorality (103).

Samples of school children in 1938-39 showed that the prevalence of syphilis varied from 10,7% of a random sample of 75 children in Witzieshoek to 46,4% of 97 children in Bochem (104) and in urban areas positive reaction rates ranged from 13,8% among 65 Pretoria school children to 42,5% of 80 children in Bloemfontein (105). Louis Leipoldt, following a study on venereal disease amongst Transvaal school children in 1920, expressed his 'grave doubts as to the truth of the almost universally accepted view that venereal disease, at least in the Transvaal, is so widespread and so prevalent' (106). He argued that high seropositivity rates were due to the Wasserman test being unable to distinguish between venereal syphilis, yaws and chronic malaria (107). His figures for the incidence of congenital syphilis are extremely low: in three coloured schools he found incidences of congenital syphilis of 0,8%, 1,8% and 3% and at an african school the incidence was just below 0,3% (108). This low incidence is consonant with endemic syphilis since women would have cross-immunity to venereal syphilis and endemic syphilis does not result in congenital transmission. Studies which cited a high incidence of syphilis in school children were possibly reflecting the prevalence of endemic rather than congenital syphilis.

In the 1930s in contrast there is undisputable evidence of congenital syphilis in urban and rural populations. A breakdown of the admission records from the East London Location Dispensary between 1932 and 1936 showed that of a total 137 syphilis cases, 58 (42,3%) were due to congenital syphilis (109) and that women with syphilis lost 76,7% of their children and those with gonorrhoea lost 51,6% (110). Records from King Edward VIII Hospital in 1941 showed that 22% of infantile deaths were due directly to syphilis (111). Figures were equally high in rural areas. Between 1941 and 1946 in Polela, 20,6% of newly born infants had congenital syphilis (112). A district surgeon from Nongoma in Natal stated:

'There are lots of people among the natives whose children are stillborn through syphilis ...the young women do not have so many children any more as they used to have in the past. If we were to go into the matter thoroughly we should probably find that syphilis is really the cause' (113).

2.3 Syphilis Patterns in Southern Africa

Further corroborating evidence for the existence of indigenous endemic syphilis can be drawn from reports of endemic syphilis in Bechuanaland, Southern Rhodesia and current South Africa which resemble the cases already discussed.

Around the turn of the century native commissioners began to draw attention to widespread prevalence of syphilis in certain districts in the southern regions of Southern Rhodesia. It was attributed to disruption following the 1893 Matabele war and 1896 rebellion and young men returning from Kimberley and the Transvaal gold mines (114). Serological sampling in survey conducted in Rhodesia in the 1950s revealed syphilis positivity rates ranging from 20% to 75% in southern rural areas (115). Willcox identified the disease prevalent in several districts in southern Rhodesia as endemic syphilis. Characteristics included common occurence in childhood, absence of a primary sore, secondary manifestations such as lesions in the mouth or on the lips, yaws-like lesions on the genitals or under the arms, syphilis-like enlarged inguinal glands, sore throat and osteocopic pains and tertiary sypmtoms such as gummata of the nose and soft palate, of the skin and skull, and periostitis (116) - characteristics similar to the disease described in South Africa in the 1880s and 1906. The disease was called 'njovera' locally and though the same word was apparently used for venereal syphilis contracted in the towns, people recognised it was a different disease (117).

Reports of widespread syphilis in Bechuanaland go back to the 1820s. The Batlhaping in Kuruman then recogised a disease called kwatsi, a word now used by the Bakwena to describe a large ulcer or treponematosis (118). The 1906 Transvaal Commission reported that estimates of the prevalence of syphilis ranged from 10-15% to 75-80% of the population (119). McArthur and Thornton claimed that 2367 cases of syphilis had been treated in Mafeking, Kuruman, Taungs and Vryburg in 1909 which was equivalent to 3,7% of the entire population of 68 000 in these districts. The Vryburg district was particularly bad, the incidence reaching 25%, but the general african prevalence was estimated at 5% (120).

Only in 1952 was the disease identified as endemic syphilis (121). Though inhabitants in the region used the same words to identify venereal syphilis and juvenile endemic syphilis viz. dichuchwa (Bakwena), thosola, rrasepipi (Bamangwato) and matsabane (Bangwakwetse) they ably differentiated between the two in practice (122).

In the late 1950s the World Health Organisation conducted a survey and mass treatment programme in the Bakwena Reserve of the Bechuanaland Protectorate. Seropositivity rates ranged from 25-64% in endemic syphilis areas. The clinical manifestations of the disease included rarity of a primary lesion, mucous patches in the mouth, aching legs, condylomata in the ano-genital and axillary areas, and a generalised papilloform rash. Late manifestations included single destructive ulcers on the body, and nasopharyngeal area and bone lesions. The disease was widespread among children but there were no cases of congenital syphilis.

The descriptions of njovera and dichuchwa at the turn of the century and the 1950s are reminiscent of doctor's reports in the Cape Colony and Transvaal outlined earlier and would confirm the contention that an indigenous endemic syphilis zone extended southwards to the Karoo. In the 1950s and 1960s four reports were published indicating the existence of endemic syphilis in the eastern Cape, northern Cape, numerous Karoo towns and an area outside Bloemfontein in the Orange Free State (123). Du Toit concluded that 'syphilis is still endemic in the whole of the Karoo and probably in the rural areas of other parts of the Republic as well, but has not been recognised as such' (124).

2.4 Periodising Disease Patterns

A comparison of the medical evidence of the 1880s, the 1906 Commission and the 1930s is suggestive of two different patterns of disease. The late nineteenth and early twentieth century was characterised by endemic syphilis already present and probably indigenous in a zone embracing southern Zimbabwe, northern and western Transvaal, south-eastern Botswana and northern and Cape interior. Venereal syphilis was restricted to large urban towns. By the 1930s doctors reported the presence of venereal syphilis in rural areas which in the earlier period had been unaffected. The migrant labour system not only exposed people to conditions where they were likely to contract STDs but also efficiently disseminated disease from urban to rural areas.

Gonorrhoea is another index of the relation between the spread of sexually transmitted diseases and migrant labour. One would expect a similarly high incidence of gonorrhoea in populations affected by venereal syphilis. Though the evidence offered to the 1906 Commission is contradictory, several witnesses testified that gonorrhoea occurred infrequently. Dr Mehliss, Medical Superintendent of the Rietfontein Lazaretto maintained that 'compared with syphilis, soft chancres and gonorrhoea are of comparatively infrequent occurrence' (125) and a Middelburg district surgeon commented that there was 'practically no venereal disease other than syphilis in his district' (126). In the earlier period when syphilis was thought to be rampant, other STDs occurred infrequently.

Thus rather than a simple picture of the transmission of sexually transmitted disease from white to black through the migrant labour system, the historical evidence is suggestive of prior indigenous epidemiological patterns which make explaining the transmission of disease more complicated. This begs the questions of firstly, why the epidemiological shift occurred and secondly, why doctors failed to recognise the different symptoms of endemic and venereal syphilis.

3. Treatment and 'Cure'

WHO conducted relatively successful campaigns to stamp out yaws and endemic syphilis in the 1950s. But curing patients of endemic syphilis or yaws removed their cross-immunity to venereal syphilis. And within a few years cases of venereal syphilis began to emerge in treated populations.

In South Africa medical services for africans have been sparse and poorly funded. Until the 1930s mission hospitals and outstation dispensaries were the main source of medical care and served vast populations. However the extent of syphilis was considered serious enough in the Transvaal to motivate some government action. The 1906 Commission recommended the establishment of a large lock hospital near Johannesburg (the Rietfontein Hospital), examination of africans at pass offices for signs of syphilis, free distribution of potassium iodide to all district surgeions and the establishment of district surgeon dispensaries (127). Between 1909 and 1911 the Transvaal administration mounted yearly district surgeon 'anti-syphilis tours' and several thousand people were treated.

The Public Health Act of 1919 provided for more extensive treatment facilities and legislated for the reporting of cases and enforcing treatment on recalcitrant patients by medical practioners and magistrates. VD clinics were established. During 1921-22 clinics were set up in East London, Pretoria, Pietermaritzburg, and Stellenbosch and the Jane Furse Memorial Hospital was opened for africans (128). Pretoria, Barberton, and Port St Johns opened clinics in 1926. The Rietfontein Hospital was rebuilt and expanded between 1923 and 1927 to accommodate 64 white and 384 black VD patients out of a total of 506 beds. The Bochem VD hospital and leper institution had space for 215 VD patients (129). In 1945 a new VD hospital was built in Lydenburg catering for the rural african population and district surgeon visits to rural clinics and dispensaries were increased from fortnightly to weekly sessions to allow for implementation of weekly injection treatment programmes.

The government also distributed free drugs for the treatment of syphilis. Mercurial ointment was distributed through justices of the peace, sub-native commissioners, missionaries and approved persons in african rural areas where district surgeon visits or medical treatment was impractical (130). The more effective salvarsan was distributed to local authority clinics. In 1923 the Department spent 3465 pounds on free drug issues which increased to 7199 pounds in 1928, 500 pounds over the budgeted amount. The department instructed clinics to use cheaper remedies, such as mercurial ointment, which medical experiment had shown was less effective and more toxic (131). Between 1940 and 1941 free drug expenditure rocketed from 11 000 to 20 000 pounds and the total cost of treating VD ran to 90 000 pounds per annum (132).

The effectivity of mercurial and arsenical treatments for endemic syphilis is unknown. Doctors complained that lengthy and painful treatment regimens, migrancy, and difficult access to clinics usually meant that patients never completed a minimum treatment schedule. But possibly the treatment was sufficient to weaken people's cross-immunity making them vulnerable to venereal syphilis.

3.2 Contemporary medical explanations of syphilis

Doctors in the 1880s and early twentieth century were firmly set on the idea that, appearances to the contrary, the disease they were dealing with was syphilis. Each time a doctor tried to suggest the disease was yaws, or fell some way between yaws and venereal syphilis, he felt the weight of professional disapprobrium.

This was partly due to the state of medical knowledge about sexually transmitted diseases. T. pallidum was only identified in 1905. Yaws was less well recognised and its destructive effects were not accepted until 1920s; even so there was often much confusion between the two. Endemic syphilis was only recognised in the 1930s (133). Thus the unusual manifestations of the disease were attributed to climatic conditions, 'seasoning' or gradual immunity to the disease. For example, one doctor explained the disease was really a milder version of European syphilis:

> 'The poorer classes are better fed here than in Europe ...and are consequently less liable to take the disease in its worst forms. The climate here is also in favour of a milder disease here' (134).

Another doctor claimed that primary and tertiary signs were absent as the coloured population was already 'syphilised' and had developed some natural immunity to the worst excesses of syphilis (135).

The 1906 commission also considered whether the disease was in fact yaws but members concluded that they were 'unable to associate ourselves with this view, though we do not doubt that indirect infection is a factor to be seriously reckoned with' (137) and it explained away the rarity of primary syphilis symptoms arguing this was due to 'the peculiar slightness of the symptoms and the natives' consequent disinclination to seek advice' (138). It maintained that

> 'the native is too ignorant and too indifferent to call attention to its presence ...consequently he does not come under medical observation until the later and more painful manifestations occur and compel him to seek advice' (139).

- in true medical fashion blaming contracting the disease and its unusual symptoms on the patient.

In the 1940s some doctors argued that different manifestations of syphilis among blacks and whites were due to blacks' lack of sustained contact with the disease rather than different genetic makeup. For example, Keen argued:

'Syphilis is a disease which has a definite evolution and I think this is very important in assessing our problem in South Africa where the disease is in a different state of evolution in our two races. ...I do not think that the difference in the clinical manifestations which we notice in the two races are due to fundamental racial differences but are more likely due to differences in the histories of the two races with regard to contact with the disease' (140).

Sax argued similarly:

'The rarity of Native tabes dorsalis and the exuberant secondary rashes, multiple bone lesions and extensive gummata of the Bantu may afford additional evidence of the brevity of their contact with syphilis' (141).

These ideas are based on the notion of the 'virgin soil epidemic thesis' which asserts that a disease is especially virulent when it finds a new population but eventually milder strains assert themselves to ensure the disease does not die out (142).

Racial and class perceptions also fed into the medical profession's insistence that the disease was venereal syphilis. Polygamy and initiation schools were interpreted as evidence for 'native immorality and promiscuity' conducive to syphilis (143). Other doctors accepted that the disease might be spread through non-sexual means such as sharing eating and drinking utensils as this reflected their belief that blacks and poor whites were personally dirty and inured to unhygienic living habits (144). The lack of tertiary symptoms, argued another doctor in a mine medical officers' journal in the 1930s, was attributable to the nature of work people performed:

'GPI is generally known to attack people who used their brains a great deal. Cerebral syphilis was less common amongst manual labourers' (145).

The failure to recognise a more complex epidemiological pattern ironically may be related to widespread acceptance from the 1920s that the migrant labour system encouraged transient social relationships most likely to transmit STDs. Ellen Hellman, liberal and urban anthropologist, condemned the social effects of migrant labour and the breakdown of family life:

'The principle cause of the high incidence of syphilis is the destruction of normal family life and the disturbance of the old tribal social customs consequent upon the system of migrant labour' (146).

Sidney Kark, a doctor who developed a curative health service in Natal rural areas in the late 1930s, described how migrancy, family separation, single-sex hostels, and the population imbalance in rural and urban areas encouraged promiscuity and the spread of syphilis. Temporary sojourn in towns, he maintained, was not conducive to the development of a moral social code and a stable family life. He argued that the prevalence of STDs was tied to South Africa's particular economic and social structure:

'We have on the one hand a set of conditions in urban areas ideal for the spread of syphilis, and on the other hand, a migrant labour force which successfully spreads this urban disease to the rural areas where social conditions are also suitable for its reception' (147).

Dr Gale, Secretary for Public Health, argued before the Fagan Commission in the late 1940s, that the migrant labour system helped transmit new diseases from urban to rural areas:

'The migrant labourer returning with untreated or inadequately treated venereal disease may infect his wife (and other women) in the rural area. From personal observation I am convinced this is the principle cause of the spread of syphilis and gonorrhea in the rural native areas. ...It is true these diseases (unknown among the original Bantu) were bound to spread among them; but the point is that the migratory system <u>favours</u> their uncontrolled spread' (148).

These politically attuned analyses accurately captured the relationship between social and economic factors and patterns of disease but perhaps blinded medical practioners to more complex disease patterns.

4. Conclusion

Public and medical concern about the widespread prevalence of syphilis has related the spread of the disease to the development of the migrant labour system. While this framework does highlight the connection between epidemiology and socio-economic transformation, it obscures pre-existing disease patterns which impinge on the way in which diseases become rooted in a population. Historical and contemporary evidence suggests that southern Africa falls into a zone of endemic syphilis and that syphilis patterns in South Africa cannot simply be ascribed to the introduction of the disease by white settlers and the transmission of the disease through the migrant labour system.

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