The Incidence of Tuberculous Infection among the Native Inhabitants of the Gold Coast and its bearing on the Question of Immunity.

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In the Report of the Principal Registrar of Deaths in the Gold Coast for 1933 it was stated that during the previous three years the proportion of deaths from pulmonary tuberculosis had far exceeded that of all other killing diseases with the single exception of non-tuberculous affections of the respiratory tract.

This disease accounted for 9.9 per cent. of the total deaths in 1931, 9.2 per cent. in 1932 and 10.5 per cent. in 1933. Tuberculosis of the respiratory tract and consequently the most infective form of the disease was far more common than any other type. In 1933 deaths from pulmonary tuberculosis numbered 658 as compared with 47 from other forms.

In the Report of the Medical Department, Gold Coast, for 1932-33, the Director of Medical Services wrote that the tuberculosis problem was one of the most important to be faced. He attributed the high incidence of this disease to insanitary housing, overcrowding, a total ignorance of the rudimentary laws of hygiene and lack of immunity. This lack of immunity associated with scanty tuberculisation of the native races is stressed by Young[1] in discussing the spread of tuberculosis among the African population of Nigeria.

As resistance to a disease may be natural or acquired, so lack of it may be due to inherent susceptibility or to absence of opportunity to acquire immunity.

According to Opie[2], there is no proof that the negro race has any inherent susceptibility to tuberculosis, and in another report[3], the same author writes: "There is indeed very little convincing evidence for or against the opinion that inherited susceptibility on the one hand, or inherited resistance on the other, modifies the progress of tuberculosis in human beings".

Carter[4], on the other hand, has tried to show that the black races have less innate resistance than the white. He cites the results of sanitorium treatment to show that tuberculosis undergoes improvement more frequently in white people than in negroes, and he attaches particular significance to the observation that Mulattoes are in this respect intermediate between whites and blacks. He points out that Mulattoes and blacks live under practically identical environmental conditions, and he concludes that the Mulatto is more resistant because the White race has transmitted part of its inherited immunity to him.

Carter has not considered the other possibility that the whites have by contact transmitted the disease to the Mulatto, and that by reason of this, acquired resistance may have been established. This possibility cannot be excluded.

Fishberg[5] states that tuberculosis is inherently not a racial problem and that one human race or ethnic group, when first encountering the tubercle bacillus, is as vulnerable to infection as another. It is only after they have been exposed to infection for generations that

they acquire a certain degree of resistance which causes milder clinical forms of the disease than are found in races which are virgin soil to the bacilli.

It would appear, therefore, that any want of immunity on the part of West African natives is due rather to lack of opportunity to develop resistance than to any hereditary or racial predisposition to the disease. This view is supported by Griffith[6], who says that races previously free from tuberculosis are much more susceptible to the disease than those who have long been exposed to infection. He attributes the greater resistance of infected communities either to a weeding out of the more susceptible human beings, the resistant individuals surviving, or to an acquired immunity produced by slight infections which have been overcome.

As acquired immunity depends on exposure to infection it would seem necessary to know the incidence of tuberculous infection before one can consider the degree of resistance to tuberculosis which a given population may have developed.

A comparison of the infection rate with the mortality or morbidity rates would then give a measure of the acquired resistance of the population in question. A high infection rate associated with low mortality or morbidity rates would suggest a considerable degree of resistance; the reverse condition would point to a low degree.

It was because the author was unable to find any

record of the incidence of tuberculous infection among the native inhabitants of the Gold Coast that the present investigation was begun.

Methods of Investigation.

An attempt was made to estimate the prevalence of tuberculosis from a study of mortality statistics, from records of clinical tuberculosis and from the results of tuberculin tests.

With regard to the first of these, mortality statistics are available for the various areas in the Gold Coast where death registration is carried out. These areas, 30 in number, represent a population of only 261,198 compared with a total native population of 3,357,950. In this connection it should be stated that there are only 3,146 non-Africans in the Gold Coast. No deaths from tuberculosis occurred in this small section and it has not been included in the investigation.

As the registration areas represent such a small proportion - approximately 7.7 per cent. of the total population - there is a possible chance of error if the figures obtained from them are taken to apply to the Colony as a whole. Moreover, they include the larger towns, seaports and mining districts where tuberculosis is more likely to be rife than in the rural districts and smaller towns. It is in the larger towns that the hospitals are mostly situated. The death registration figures are largely obtained from these hospitals where cases of disabling

illness, especially among the lower classes of natives, tend to congregate. The mortality rate obtained from these figures is therefore likely to be considerably higher than the rate one would get were figures available for the country as a whole.

Another source of error is that the deaths registered on medical certificates or after autopsy amount to only 55.0 per cent. of the total number registered.

From the figures available, the following table has been compiled to show the mortality rate from pulmonary tuberculosis for the past six years. It also shows the relation between the number of deaths from this disease and the total number of deaths during the same period.

Table I.

Year	Total number of deaths	Number of deaths from Pulmonary Tuberculosis	Percentage of deaths from Pulmonary Tuberculosis	Mortality Rate per 1000 population
1929	5415	497	9.17	2.25
1930	5972	566	9.47	2.50
1931	5972	596	9.98	2.28
1932	5905	545	9.22	2.01
1933	6264	658	10.50	2.50
1934	6550	642	9.80	2.45

As complete population figures for all registration areas were unobtainable prior to 1929, the mortality

rate could not be calculated before that year. For the reasons previously given, the rates in this table can only be taken as approximate.

Records of clinical tuberculosis are also liable to error as most of the cases seen at hospital have only come for treatment when the disease has reached an advanced stage and has produced disability. It is difficult to know what relation the number of these diagnosed cases bears to the number of cases which never come to hospital.

The following table, taken from the Reports of the Medical and Sanitary Department of the Gold Coast for 1929-30 and 1933-34, shows the total number of cases of tuberculosis treated at the various hospitals for each of the previous eleven years. It also shows the relation of this number to the total number of all diseases treated.

Table II.

Year	Cases of Tuberculosis	Percentage of all cases treated.
1923-24	411	.53
1924-25	414	.50
1925-26	571	.58
1926-27	698	.66
1927-28	910	.68
1928-29	1151	.65
1929-30	1175	.64
1930-31	1300	.48
1931-32	1340	.52
1932-33	1227	.51
1933-34	1193	.47

From this Table tuberculosis does not appear to be a rapidly spreading disease in a non-immune population.

The third method of investigation - the use of tuberculin tests - has the great advantage that it can be applied to any part of the population and to the healthy, or apparently healthy, individuals in it.

Cummins[7] says that there can be no doubt that sensitivity to tuberculin proves the existence of tuberculous infection, and Riviere[8] states that the cutaneous tuberculin test of von Pirquet, or the intracutaneous test of Mantoux, shows whether or not infection with the tubercle bacillus has occurred.

By means of one or other of these tests, therefore, the amount of tuberculous infection in the community as a whole can be determined.

According to Gloyne[9], von Pirquet's test has now been largely replaced by the intracutaneous test of Mantoux. Dow and Lloyd[10] consider that the latter test from the standpoint of accuracy is the best for determining tuberculous infection. The Mantoux test was, therefore, chosen in preference to that of von Pirquet in the present investigation.

Type of Infection and Disease.

The principal veterinary officer[11] has pointed out that tuberculosis among cattle in the Gold Coast is extremely rare, and the principal registrar of deaths[12], in

his report for 1934 wrote: "It is to be noted that little fresh milk is consumed and that tuberculous meat is a great rarity in this colony".

Owing to the prevalence of tsetse fly and trpanosomiasis, cattle can be kept only in a few restricted areas, and even in these areas a very small amount of milk is used for human consumption.

Practically all the milk used in the colony is the sterilized tinned variety. Infection from bovine sources is therefore non-existent and any tuberculous disease present must be the result of contact with human cases.

In this respect the type of tuberculous infection in the Gold Coast resembles that found in India where, according to Ukil[13], infection with the bovine type of bacillus practically never occurs.

From reports[14] of clinical tuberculosis, as met with in this Colony, the type of disease most commonly seen is fibro-caseous tuberculosis with a preponderance of the caseous process.

A similar type of disease was found by Ukil[13] to be common among the natives of India, and Everett[15] has shown that this type also formed about fifty per cent. of the cases seen among American negroes.

Fibroid phthisis is rarely seen in hospital practice in the Gold Coast, though to what extent it exists in people who never come for treatment it is not possible to say. The sputa of such cases as are seen at hospital contain as a rule great numbers of tubercle bacilli.

These are scattered broadcast in the process of expectoration. Since overcrowding is rife and hygienic habits bad, each of these cases must be a source of massive infection to others.

Material.

The individuals on whom the tests were carried out were healthy persons chosen from all classes of African society in three different parts of the colony.

The first district - Saltpond - is situated near the coast. Its chief town has a population of about 6000. There are numerous villages in the district - some along the coast, others a few miles inland. The population of each village amounted to a few hundred. Many of the people tested had lived in more than one town or village and there was more or less free intercourse between all the villages for purposes of native trade.

The second district - Accra - is also situated near the coast. Its chief town is a seaport with a population of 60,000. All the cases tested in this district came from this one town.

The third district - Tarkwa - is situated about 40 miles inland and, like the first district, consists of one moderate-sized town of about 5,000 inhabitants and numerous smaller towns and villages scattered through the rest of the area.

In this district there are a number of Gold Mines, but it was not possible to get tests carried out on the labourers in these mines. Only the ordinary inhabitants

of the area were tested. The mine labourers as a rule live in villages by themselves and do not mix to any extent with the natives of the district.

The work was begun in January 1932 and continued as opportunity offered till June 1935.

During that time 1964 individuals were tested. They were drawn from all classes of the community and were representative of the population of the colony as a whole.

Technique.

The tests were carried out with old tuberculin human - obtained from Messrs. Burroughs and Wellcome - used in dilutions of 1 - 5000, 1 - 1000, and 1 - 100. In order to avoid any risk of deterioration these were made up freshly each testing day, the diluent used being sterile normal saline to which 0.5 per cent. phenol was added.

A 1 c.c. tuberculin syringe, fitted with a No. 214 Burroughs and Wellcome needle, was used, the site chosen for the injection being the flexor surface of the left forearm. The skin was swabbed with ether and then stretched tight. The needle was inserted carefully into the skin and 0.1 c.c. of the solution was injected. Particular care was taken to see that the tuberculin went into, and not underneath, the skin.

Dosage.

It is stated by Gloyne[9] that the generally accepted standard test dose is 0.1 c.c. of 1 - 1000 dilution, but he recommends this to be reduced to 1 - 5000 for infants

and small children. The writer could not find any record of tuberculin tests having been carried out in West Africa by the Mantoux method. It was decided, therefore, to make the initial dose in all cases 0.1 c.c. of 1 - 5000 dilution and to give those who did not react similar doses of 1 - 1000 and 1 - 100 dilutions.

It was soon noticed among the earlier cases tested that a considerable number of people who received one injection failed to turn up again for examination and further tests. It was then decided to reserve the initial dilution of 1 - 5000 for those individuals who were likely to be seen regularly and to give those who were unlikely to be available for more than one test an initial dose of 1 - 1000 dilution. In the latter part of the investigation it was found possible to make the dose 1 - 5000 for all.

Forty-eight hours after the first dose the injected arms were examined for any sign of reaction.

If no reaction was seen, or if the result was doubtful, a second dose of 0.1 c.c. of 1 - 1000, or 1 - 100 dilution was given seven days after the first dose, and the presence or absence of reaction noted as before.

In cases where the second dose was 1 - 1000 dilution, and no reaction occurred, a third dose of 1 - 100 dilution was given seven days after the second injection.

It was not possible to get all non-reacting cases tested up to 1 - 100, but 80 per cent. of those negative to higher dilutions were tested a second or third time.

Control injections with a special control fluid, supplied by Messrs. Burroughs and Wellcome for the purpose, were given in 300 cases. Separate syringes were used for the control fluid and the tuberculin.

The results of the tests are shown in the following tables and graph:-

Table III.

Males				Females			Total		
Age Yrs.	No. Tstd.	No. Pos.	Per Cent. Pos.	No. Tstd.	No. Pos.	Per Cent. Pos.	No. Tstd.	No. Pos.	Per Cent. Pos.
0-5	89	20	22.47	75	13	17.33	164	33	20.12
6-10	195	51	26.15	101	34	33.66	296	85	28.71
11-15	214	107	50.00	77	33	42.85	291	140	48.11
16-20	144	98	68.05	44	28	63.63	188	126	67.02
21-30	340	270	79.41	182	123	67.58	522	393	75.28
31-40	149	128	85.90	136	104	76.47	285	232	81.40
41-50	59	52	88.13	72	51	70.83	131	103	78.62
Over 50	43	38	88.37	41	31	75.60	84	69	82.14

This table shows the results obtained in 1961 persons of whom 1375 received an initial dose of 0.1 c.c. of 1 - 5000 dilution of tuberculin and 586 an initial dose of 0.1 c.c. of 1 - 1000 dilution. Those who did not react were, as far as possible, tested out to 1 - 100 dilution.

Table IV.

	Males			Females			Total		
Age Yrs.	No. Tstd.	No. Pos.	Per Cent. Pos.	No. Tstd.	No. Pos.	Per Cent. Pos.	No. Tstd.	No. Pos.	Per Cent. Pos.
0-5	21	5	23.8	12	1	8.3	33	6	18.1
6-10	60	11	18.3	29	7	24.1	89	18	20.2
11-15	56	30	53.5	18	5	27.7	74	35	47.3
16-20	72	42	58.3	19	8	42.1	91	50	54.9
21-30	88	56	63.6	57	32	56.1	145	88	60.6
31-40	30	22	73.3	54	32	59.2	84	54	64.2
41-50	15	10	66.6	32	17	53.1	47	27	57.4
51 & over	3	3	100.0	20	16	80.0	23	19	82.6

This table shows the results obtained in 586 persons who received an initial dose of 0.1 c.c. of 1 - 1000 tuberculin. The figures are those obtained from this one standard dose, and do not include the increased number of positive reactions which were found when the non reactors were given a dose of 1 - 100 dilution.

Table V.

	Males			Females			Total		
Age Yrs.	No. Tstd.	No. Pos.	Per Cent. Pos.	No. Tstd.	No. Pos.	Per Cent. Pos.	No. Tstd.	No. Pos.	Per Cent. Pos.
0-5	45	8	17.77	35	6	17.14	80	14	17.50
6-10	37	8	21.62	26	9	34.61	63	17	26.98
11-15	49	23	46.93	24	11	45.83	73	34	46.57
16-20	44	23	52.27	20	10	50.00	64	33	51.56
21-30	233	158	67.81	114	61	53.50	347	219	63.11
31-40	108	79	73.14	84	48	57.14	192	127	66.14
41-50	42	31	73.80	38	23	60.52	80	54	67.50
51 & over	40	24	60.00	21	10	47.61	61	34	55.73

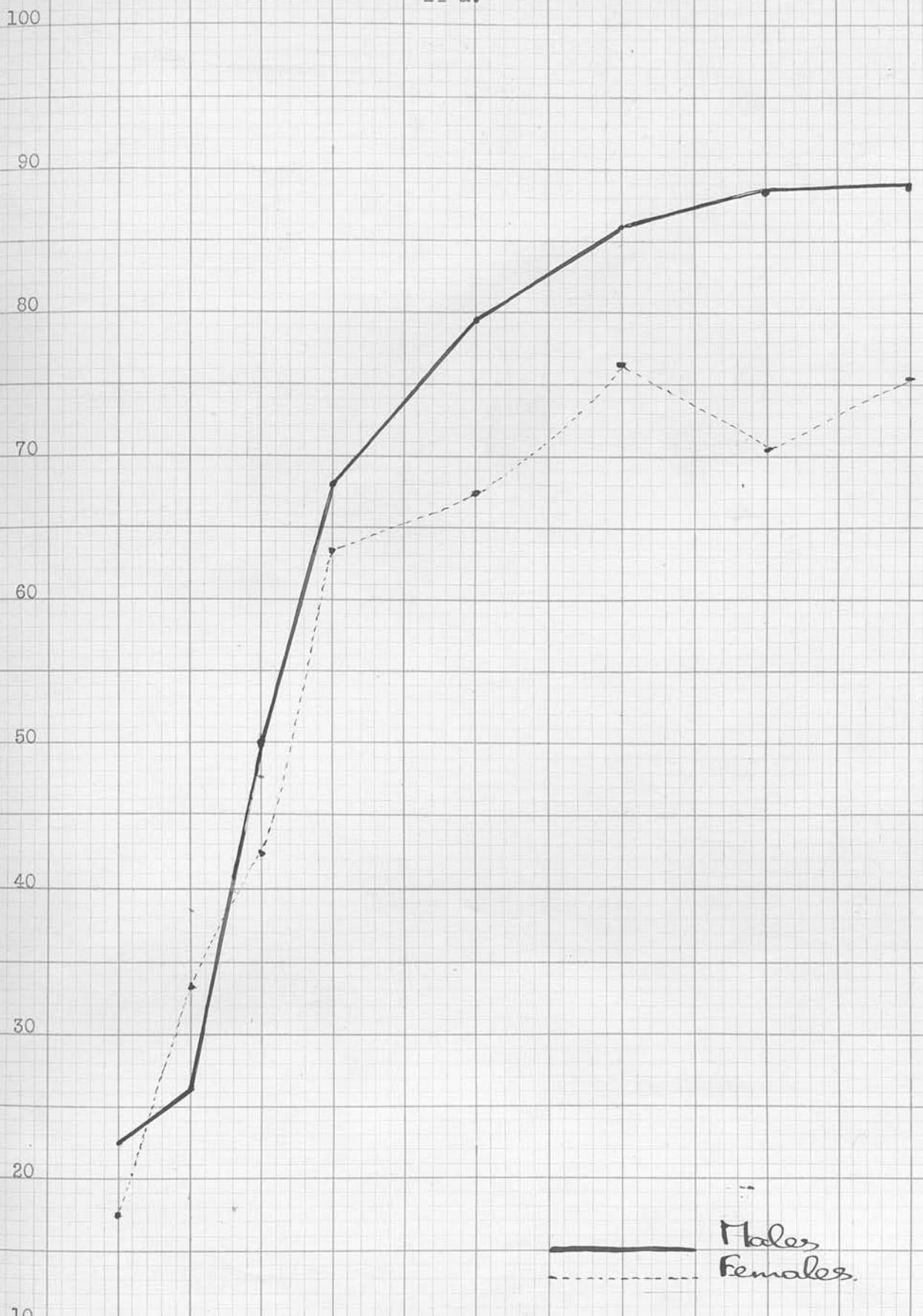
This table shows the results obtained in 960 consecutive cases who received an initial dose of 0.1 c.c. of 1 - 5000 tuberculin. The figures are those obtained from this one standard dose and do not include the increased number of positive reactions which were found when the non reactors were given doses of 1 - 1000 and 1 - 100 dilutions.

Table VI.

	Saltpond			Accra			Tarkwa		
Age Yrs.	No. Tstd.	No. Pos.	Per Cent. Pos.	No. Tstd.	No. Pos.	Per Cent. Pos.	No. Tstd.	No. Pos.	Per Cent. Pos.
0-5	84	16	19.0	7	1	14.20	73	16	21.9
6-10	233	64	27.4	10	3	30.0	53	18	33.9
11-15	218	100	45.8	13	7	53.8	60	33	55.0
16-20	124	86	69.3	16	10	62.5	48	30	62.5
21-30	175	121	69.1	52	41	78.8	295	231	78.3
31-40	93	72	77.4	17	14	82.3	175	146	83.4
41-50	51	36	70.5	6	5	83.3	74	62	83.7
51 & over	23	19	82.6	8	6	75.0	53	44	83.0

This table shows the incidence of infection in each of the three areas where the tests were carried out. Dosage was the same as stated for Table III.

On the two following pages results are shown in graphic form.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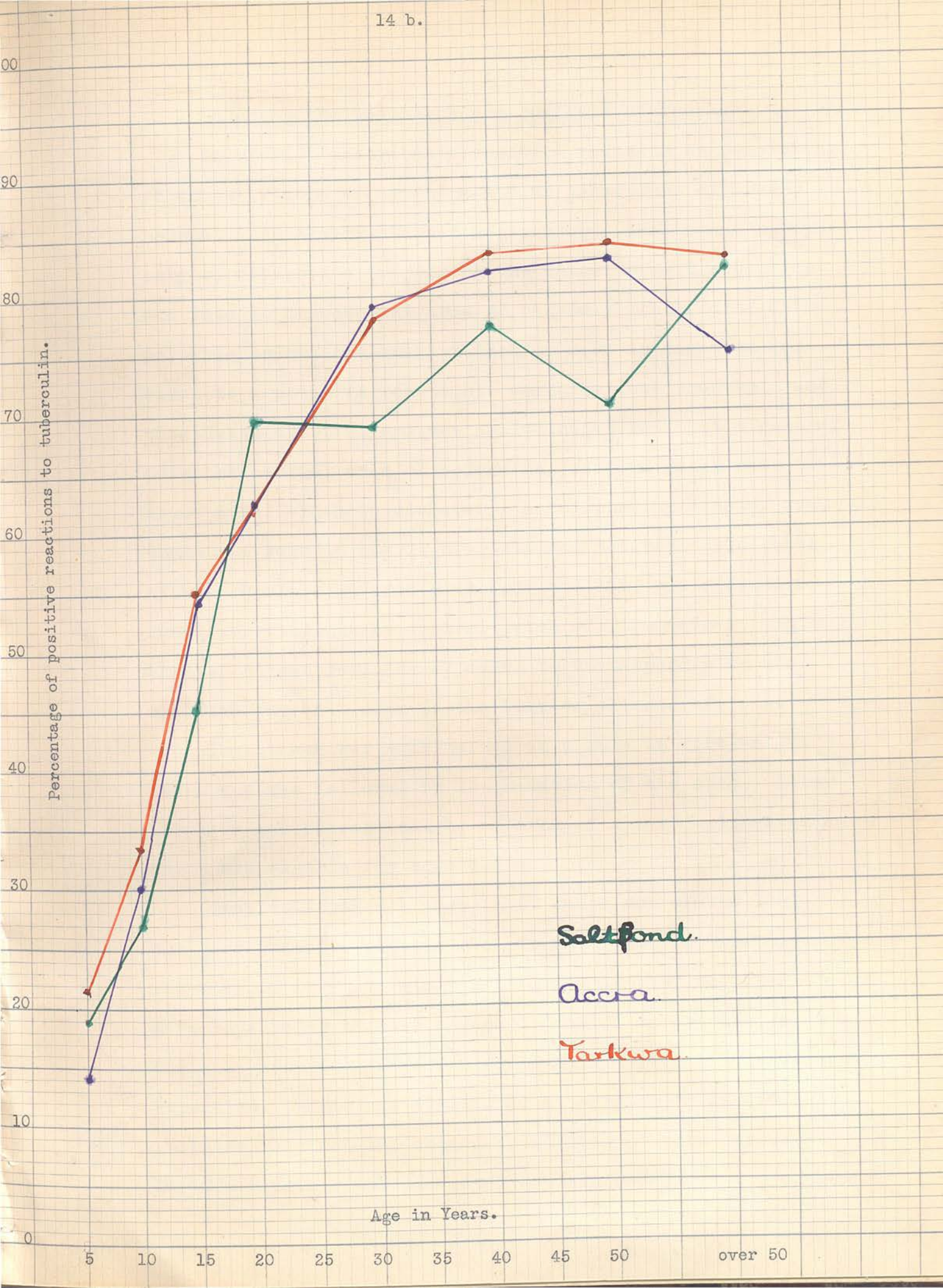
Percentage of positive reactions to tuberculin.
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Age in Years.
Saltpond.
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Tarkwa.

Notes on the Reactions and Analysis of Results.

Gaisford[16] describes a positive reaction as consisting of an outer zone of erythema and an inner zone of brawny oedema. In a dark-skinned African erythema is not always easy to detect and in judging the reaction most attention was paid to the central area of oedema. An area of firm palpable oedema one-quarter inch or more in diameter was taken as a positive reaction; anything less than this was considered doubtful and was called negative. As a general rule the central area of oedema in positive cases was about a half-inch in diameter. A surrounding zone of erythema was seen in the lighter skinned natives. In those with dark skins an area of slight swelling was frequently found surrounding the central patch of firm oedema. This area of slight swelling appeared to correspond with the erythematous area seen in the lighter skinned Africans.

The oedema was well marked as a rule in forty-eight hours and was followed by desquamation of the skin. Later the site was represented by an area of altered pigmentation which was visible for some weeks.

Vesiculation was seen in 97 cases out of the 1181 who gave positive reactions. In nine cases no reaction was visible in forty-eight hours. When they returned five days later to have a second dose of tuberculin, a definite area of oedema was found at the site of the first injection. This was similar in appearance to an ordinary positive reaction and seemed to correspond with the delayed positive reactions noted by Gaisford[16].

These were treated as positives in compiling the tables.

1961 persons were tested as described previously. 1375 received an initial dose of 0.1 c.c. of 1 - 5000 dilution of tuberculin. 682, or 49.6 per cent., gave a positive reaction. 693 did not react, and of these 607 were given a second dose of 0.1 c.c. of 1 - 1000 dilution. 110 gave a positive reaction which increased the percentage to 57.6. Of the remaining 497 who were negative, 377 received a third dose of 0.1 c.c. of 1 - 100 dilution. 58 more positives were obtained giving a total of 850, or 61.8 per cent. positive results.

586 individuals were given an initial dose of 0.1 c.c. of 1 - 1000 dilution of tuberculin. 297, or 50.68 per cent. gave a positive reaction. 289 did not react, and of these 190 received a second dose of 1 - 100 dilution. 34 gave a positive result which increased the percentage of positive cases to 56.48.

Control Cases.

Of the 300 persons who were given control injections all, except eight, gave negative results. In six of these eight, a doubtful reaction - that is an area of oedema less than one-quarter inch in diameter - was obtained. In the other two cases the reaction produced by the control fluid was exactly similar to a positive tuberculin reaction, the area of oedema in each case being nearly a half-inch in diameter.

These eight cases all gave positive reactions to tuberculin.

The control fluid was used in a dilution of 1 - 25 throughout.

Discussion.

Table III shows that there is a progressive increase in the incidence of tuberculous infection from infancy to adult life in both sexes.

Table VI shows that the incidence rates at each age period correspond fairly closely in each of the three districts in which the tests were carried out.

In children up to fifteen years of age the incidence is 34.35 per cent. In that part of the population over fifteen years of age the incidence is 76.28 per cent.

As it was not possible to get all negative cases in this series tested out to 1 - 100 dilution of tuberculin, it was decided to extract and tabulate the results obtained.

(a) In 960 consecutive cases when an initial dose of 1 - 5000 dilution of tuberculin was used.

(b) In 586 cases when an initial dose of 1 - 1000 dilution of tuberculin was given.

A certain number of those who did not react to these dilutions gave a positive reaction when stronger doses were used, but these were not taken into consideration in this part of the investigation. The results are shown in Tables IV and V. From these it will be seen that as in the larger group the incidence of infection increases steadily from childhood to maturity, but the percentage

of positives in each age group is lower than that found when the non reactors were tested out to 1 - 100 dilution.

Intensity of Reaction.

Moderately severe reactions with vesiculation in varying degree were found in 97 cases; 33 occurred in children under 15 years, and 64 in adults. 31 resulted when 1 - 5000 dilution of tuberculin was used, 55 when the strength was 1 - 1000, and 11 when it was 1 - 100.

In no case was the reaction so great as to prevent the individual continuing his or her work. This is in marked contrast to the sensitivity shown by the natives of South Africa. Allen[18], carrying out tuberculin tests among the latter, found that it was not practicable to use a dilution of 1 - 1000 on account of the severe reactions which it produced. No such extreme sensitivity was found among the natives of the Gold Coast, to whom doses of 1 - 1000 and 1 - 100 dilutions were frequently given without any unduly severe reactions resulting.

Ukil[13] found that in India severe reactions, especially in children, were often evidence of exposure to massive infection, usually within the family, and in certain cases indicated the borderland between infection and disease.

An attempt was made to find out if a similar relationship existed in the Gold Coast. In ten of the cases in which vesiculation occurred close contact with patients

suffering from advanced pulmonary tuberculosis was found but nothing definite could be discovered concerning the remaining 87 cases. As it was not possible to visit the various villages from which they came and carry out an inspection of their homes, one cannot say whether or not they were likely to have been exposed to recent massive infection.

Influence of Sex.

At each age period, except between the years 6 - 10, the incidence in males is higher than in females. When the figures of all the age groups are taken together the incidence in males works out at 61.96 per cent. compared with 57.28 per cent. in females. No cause could be found to account for the slightly higher incidence in males. From their habits and mode of life both sexes appear equally exposed to infection.

The higher incidence of infection in females than in males in the age group 6-10 years, shown in Tables III, IV and V, is possibly due to the fact that girls around this age tend to be kept more about the house than do boys. They are therefore more liable to get infection from any sick persons who may be in their homes. In this respect Ukil's[13] observation that in India the infection rate among girls of the marriageable age 11-20 years is considerably higher than among boys of corresponding age, is of interest. He attributes this to the custom of keeping girls of this age more or less confined to the house. In the

Gold Coast no such custom obtains of keeping girls of marriageable age in seclusion and the infection rate among these girls is lower than in boys. At the earlier age - below ten years - when Gold Coast girls do tend to be kept more at home, the infection rate is definitely higher in them than in the boys.

A similar state of affairs obtains in South Africa according to Allan[17], who found that among 547 boys between the ages of 5 and 15 years, 59.5 per cent. gave a positive reaction, whereas in 1005 girls at the same age period 68.5 per cent. were positive. He also attributes this to the fact that at this period girls are more confined to the huts than are boys. Below and above these age periods he found the proportion of positive reactors in the two sexes fairly equal.

Table VII, which follows, shows this increased incidence of infection in females at the ages stated.

Table VII.

	Gold Coast Per Cent. Positive		India Per Cent. Positive		South Africa Per Cent. Positive	
Age Group	Male	Female	Male	Female	Male	Female
6-10 years	26.15	33.66	29.50	30.30	59.5	68.5
11-15 "	50.00	42.85	29.70	35.00		
16-20 "	68.05	63.63	29.90	58.00		

Influence of Social Status.

In considering this the population was divided into four groups which, it was considered, adequately represented the different social grades. These groups and the infection rate in each are shown below.

School children, 6-15 years	40.0	per cent.	positive
Labourers	78.8	" "	"
Petty Traders and Artizans	76.8	" "	"
Educated Classes, e.g. Clerks, Teachers ...	78.2	" "	"

From this it will be seen that social position does not influence very greatly the incidence of infection. The uniformity of the infection rate is not to be wondered at when it is considered that an intense dislike of fresh air in their houses, promiscuous expectoration and general insanitary habits are common to all the different social groups.

Comparison of the infection rate found in the Gold Coast with that in other countries.

Investigations into the incidence of tuberculosis among coloured peoples have been carried out in various countries by workers using the von Pirquet test. Results of such surveys have been published by Ukil[13] and Gupta and Dutt[19] in India, by Schwetz, Cabu and Baumann[20] in the Belgian Congo, and by Allan[17] in South Africa. A survey among children in England has been recently carried out by Dow and Lloyd[10], who used the Mantoux test.

The results found by these workers have been tabulated on Table VIII and compared with those found in the Gold Coast.

Table VIII.

Age Years	Gold Coast	Belgian Congo	India	England	South Africa
0-5	20.12	10.5	11.4	23.0	39.6
6-10	28.71	32.8	30.1	39.0	58.9
11-15	48.11	38.2	33.3	58.3	71.6
16-20	67.02		38.1		81.0
21-30	75.28		51.3		93.0
31-40	81.40		56.0		
41-50	78.62		59.4		
51 & over	82.14		67.4		

The figures for the Belgian Congo and India are based on the results of von Pirquet's test. In England and the Gold Coast the Mantoux test was used, and in each case as many individuals as possible were tested out to 1 - 100 dilution. In South Africa Allan used the Mantoux test employing 1 - 5000 dilution only.

This table shows that the incidence of infection amongst children under 15 in the Gold Coast is distinctly lower than in England and South Africa, while, with the exception of the age group 6-10 years, it is definitely higher than in the Belgian Congo and India.

Relation of Infection to Immunity.

Willis[21], discussing immunity to tuberculosis, states that the animal which is infected with the tubercle bacillus is in some degree specifically protected against the disease for which that germ is responsible. He points out that the immunity which such an animal possesses is only relative or partial, and the size of the reinfecting dose is an important factor in determining the competence of the immunity.

Fishberg[5] writes that tuberculous infection during childhood, so long as it is not acute and fatal, endows the body with heightened resistance against renewed endogenous and exogenous infection with tubercle bacilli. The immunity thus produced is, he states, in most persons ample to protect them during the rest of life.

Heimbeck[22], working in a large municipal hospital in Oslo, tested with tuberculin every nurse entering the institution during the years 1924-1927. He used von Pirquet's method, and during the period mentioned he examined 397 probationers. 189 gave positive reactions and 208 gave negative results. The tests were begun in January 1924 and, up to the date of publication in 1927, 43 nurses had developed signs of tuberculosis. Of these 40 belonged to the group that did not react to tuberculin on entering the hospital, while only 3 cases came from the group of positive reactors. From these figures Heimbeck came to the conclusion that a negative von Pirquet test indicated susceptibility to tuberculosis

and a positive reaction the reverse. This conclusion was supported by his findings[23] in a series of medical students. Out of a group of 183 students 88 gave a positive reaction to tuberculin and 95 did not react. 44 of this latter group were immunized by vaccination with B.C.G. leaving 51 not immunized. Seven students eventually developed tuberculosis, one belonged to the group of 88 positive reactors. and six came from the group of 51 non reactors who had not been immunized. There were no cases of disease in the group of 44 who had been immunized.

Roodhouse Gloyne[9], in a recent review of tuberculin tests, came to the conclusion that, in a contact population, persons who did not react to tuberculin were more liable to develop tuberculosis than those with a positive reaction.

Pottenger[24] states that immunity in the course of a disease is a specific protection which the host develops as a result of its experience in fighting an infection. He uses the term allergy to express the inflammatory reaction which takes place when bacillary protein is brought in contact with the cells of an immunized host, which previously have been sensitised in the course of natural infection or as a result of the artificial injection of living or dead bacilli.

From the fact that allergy develops early in tuberculosis; that it is due to functional hypersensibility of the cells and hastens and magnifies the body's response

to further invading bacilli or bacillary protein; that the patient shows an increased power to resist and limit infection when it is present; that the reaction, as we produce it with tuberculin used therapeutically, is followed by improvement, he considers that it is a phase of the protective mechanism. He discusses the difference in reaction of preponderantly proliferative as compared with preponderantly exudative tuberculosis and points out that the former, though milder, shows a greater tendency to extension than to self limitation and that it heals with difficulty.

He shows that this proliferative type is favourably influenced by therapeutic doses of tuberculin and that this response to tuberculin is a response to the protein which produces the allergic reaction and at the same time stimulates the immunising mechanism. The preponderantly exudative lesions on the other hand, while they may be more acute, and are accompanied by more serious symptoms and show greater inflammatory reaction, yet they may, he states, heal more completely and in a shorter period of time. While the lesion is accompanied by a greater degree of allergy, he considers it to be accompanied by a greater specific defence, for extension is held in check more successfully than in the case of milder proliferative lesions, and he comes to the conclusion that allergy develops coincident with immunity and remains as a potential reaction as long as infection exists in the body of the host.

Topley and Wilson[25], discussing the question of immunity in tuberculosis, state that there seems to be little doubt that allergy is an expression of immunity. They add: "We must now consider why it is that in the presence of widespread tuberculous infection the great majority of adults living in civilised countries fail to develop clinical tuberculosis".

"From von Pirquet's figures it is clear that 90 per cent. or more of persons who have been brought up in large European cities and who have passed the age of adolescence react positively to tuberculin. That is to say, these persons are in the allergic state and are hence comparatively resistant to tuberculosis".

It has also been stated by Coutts[26] that it seemed undeniable that tuberculosis in childhood established some kind of immunity, and he believed that the recent relative increase of pulmonary tuberculosis in young adults is due to a larger proportion of persons reaching this age without having previously acquired and recovered from tuberculous infection.

Kenwood and Kerr[27] point out that in areas where the population has remained comparatively isolated from the rest of mankind cases of tuberculosis tend to be rare or absent. This absence or rarity is accompanied by a very low percentage of positive cutaneous reactions to tuberculin. The inhabitants of such areas show, however, when brought into contact with infection, an extreme degree of susceptibility to tuberculosis, the clinical mani-

festations of attack being characterised by an absence of fibrotic reaction, a tendency to rapid generalisation of the disease and a speedy fatal ending. They consider that the phenomena of the disease in civilised communities are best explained on a theory of active immunity acquired by contact with the tubercle bacillus which is so abundantly present in the general community that the majority of children under 14 years have already acquired definite tuberculous lesions. These lesions, whether arising from bovine or human sources, confer more or less immunity to the generalised and rapidly fatal forms which are not common till after puberty.

According to Opie[2], the tuberculin reaction has shown that among American negroes minor infections occur with increasing frequency from birth to adult life. He adds that there is abundant evidence that these infections confer immunity, though under unfavourable conditions this immunity may not be sufficient to prevent disease.

Borrel[28], working among Senegalese troops in France, found that they were extremely susceptible to tuberculosis and tended to develop a severe form of the disease. He found that this susceptibility was associated with a very low rate of infection, only 4 per cent. to 5 per cent. giving a positive reaction to tuberculin on their arrival in France.

He contrasts this with the tuberculosis incidence among the American negro troops in France. These were

not found to be any more susceptible to the disease than white troops, and Borrel attributes this to the fact that these American negroes had been exposed to infection since their childhood, and so were more resistant to the disease.

Sewall, de Savitsch, and Butler[29] have tried to represent the relation of allergy to immunity through a statistical study of tissue reactions due to superinfection following infection at varying intervals of time.

They infected guinea pigs with virulent tubercle bacilli and reinfected them again with similar bacilli at varying intervals of time.

They found that at a certain period after infection the violence of the local reaction to reinfection began steadily to diminish, and that the general stability of the reacting host - his power of adaptation and restoration - that is immunity, at the same time increased. By superinfection at 53 days after, they found that the animal's whole body probably shared the allergic state, but by withholding superinfection for longer periods they discovered that allergy diminished and immunity increased.

They state that, far from stigmatising allergy as indifferent or antagonistic to the development of immunity, they are impressed with the view that it represents crude vital energy specifically excited and capable of adaptation to the protection of the host whose tissues are trained by stimulation of the very antigen which first aroused them.

This all goes to show that, as Zinsser[30] puts it,

"the allergic state may, in certain circumstances, be regarded as an index of resistance, a thing which we believe to be true in tuberculosis".

Tytler[31] sums up the position as follows:-
"General opinion still seems to regard allergy as a most important factor in resistance to infection, though the view is usually expressed in a more tentative form than previously and is openly challenged in some quarters".

A different view of the relation between allergy and immunity is taken by Rich[32], who states that one may not use the tuberculin test and other similar allergic reactions as an index or measure of immunity. He has tried to dissociate allergy from immunity and to show that each can exist independently of the other. One of his methods of doing this was to render a large number of guinea pigs allergic and immune by vaccination with attenuated tubercle bacilli. Half were desensitised by gradually increasing doses of tuberculin, the other half being left allergic. Both groups were then infected with a virulent culture of tubercle bacilli, the desensitised animals continuing to receive their large daily dose of tuberculin in order to maintain the state of desensitisation throughout the experiment. He found that the desensitised animals were as highly resistant as were the allergic ones. From this he concluded that allergy had been abolished while immunity remained - immunity acting in complete absence of allergy. A criticism of this work appeared in a leading article in the British Medical

Journal[33] in which it was pointed out that it was unjustifiable to assume that animals whose antibodies are being continuously neutralised with massive doses of antigen are no longer allergic, and that it would be almost as justifiable to assume that an anesthetised man who was being continuously given whiffs of chloroform was dead because he showed no signs of resuming activity.

Rich himself admits that, while the body is acquiring an active immunity to bacteria during infection, the tissues ordinarily become hypersensitive to the protein of the infecting micro-organisms, and by the side of this hypersusceptibility of the tissues there is also developed a heightened resistance to the growth of the bacteria, and their spread through the tissues and into the blood stream is markedly inhibited.

In another experiment quoted by Rich to prove that allergy exists in the complete absence of immunity, he states that when animals were rendered allergic to a high degree by tuberclo-protein and then tested for immunity by the injection of virulent bacilli, no immunity to infection was present. According to Pottenger[34] the immunity mechanism is set in motion by an infection produced by living tubercle bacilli, to a lesser extent by dead bacilli, and possibly to a minimum extent by tuberclo-protein, but for an efficient protective response living bacilli are necessary. After the mechanism has been set in motion and immunity has once been established, then either living bacilli, dead bacilli, or tuberculo-protein

will stimulate it further and increase its efficiency.

If Pottenger's view is correct, this experiment is open to serious criticism on the ground that Rich did not supply the necessary stimulus to immunity production and it is therefore not surprising that immunity did not develop.

A view similar to that of Rich is taken by Cummins[35], who states that the native mine workers in South Africa have a very considerable degree of allergy, but have acquired little or no immunity to tuberculosis.

The report[36] on the investigation into the question of tuberculosis among the native workers in the Rand Gold mines is important in this respect and its findings must be considered.

On page 97 of this report it is stated that from 1st April 1928 to 30th September 1930, 93,979 native boys were tested as regards their reaction to tuberculin before starting work in the gold mines.

65 per cent. gave a positive reaction to 0.1 c.c. of 1 - 5000 tuberculin intradermally.

35 per cent. gave a negative reaction to this strength of tuberculin.

During their contract periods 566 of these boys developed recognisable tuberculosis. 452 cases occurred among 61,115 positive reactors and 114 among 32,864 who did not react to tuberculin. The incidence of disease was therefore twice as great among the positive group as among the

negative, the rates per 100,000 boys being 738 cases compared with 347 cases.

At first sight it might appear from this that the positive reactors have less resistance to the disease than those who do not react. A consideration of the report, however, suggests that there may be another explanation of the high incidence of disease in the tuberculin positive natives. On page 73 it is stated: "The transportation of the large numbers of natives underground takes a considerable time and a certain number may have to wait an hour or even two before they can enter the cage which is to carry them underground. On a cold winter's morning this waiting, which is often done in the open, or practically in the open, may make a serious inroad into the native's vitality".

It is further pointed out that the native runs a considerable risk of chill during the time spent - sometimes an hour or more - waiting to be hauled up out of the mine after knocking off work in a hot, damp place.

The sudden transmission from a tropical or subtropical climate to that of the high veld is given - page 64 - as another factor in lowering resistance. There is in addition the special risk on the Witwetersrand Goldfield of exposure to silica dust with its unfavourable effect on the incidence of tuberculosis.

The adverse conditions under which these mine boys work are summarised on page 276 of the report, as follows:-

"Lack of adequate acclimatization in a trying industry. Secondary respiratory infections in crowded hutments. The mixing of the relatively immune with non-immune persons. The poverty in vitamins A and D of an otherwise adequate dietary. The strain of deep mining and all that it implies in long hours underground, severe physiological stress and climatic contrasts between conditions underground and on the surface, and, lastly, exposure to silica dust".

Under these conditions it is not surprising that there should be a considerable incidence of tuberculosis among these native miners. The first to show signs of breaking down resistance and development of disease would be those who already harboured the infection but who had been able to keep it in subjection during their ordinary life at home, for, as has been pointed out by Philip[37], although the influence of the tubercle bacillus may be frequently beneficial, making for immunity against further attack from outside, the bacillus is also highly resistant and after remaining dormant for long periods is still capable of reactivity and reproduction, given favourable conditions.

The incidence of disease would, therefore, at first be greater among the positive reactors than among those not previously infected. With the occurrence of open cases of disease the infection would spread to those hitherto free from it.

If this infection were widespread one would expect extensive invasion of the non-immunes. However, with regard to those cases of active disease two important factors must be considered. According to the report the times of greatest danger in the development of tuberculosis are during the first year, and after the fifth year. The cases occurring in the first year are found mostly in boys who are infected before starting work in the mines and whose resistance breaks down under the strain of unaccustomed hard work and changed environmental conditions.

Elaborate precautions are taken to detect disease early, by means of regular weighings, careful clinical examinations, and by observation in hospital of all boys whose weight begins to fall.

Among the older mine workers, that is after the fifth year, the increased incidence of tuberculosis is caused largely by the silicotic element - page 352. According to Griffith[6], the type of disease which results from exposure to silica dust has a low degree of infectivity. These older workers who suffer from this type of disease are, in addition to other examinations, subjected to periodical X-ray examinations in an endeavour to detect tuberculosis at as early a stage as possible.

These two factors - the care taken to detect and remove from the mines all cases of tuberculous disease at an early stage, and the low infectivity of the type of disease which develops in the older workers - must

play a large part in limiting the spread of tuberculosis among the non-infected miners. It seems probable, therefore, that the higher incidence of disease among those previously infected is due not to lack of immunity but to breakdown of resistance under exceptional circumstances. The low rate among the non reactors to tuberculin appears to be due largely to the elaborate precautions taken to prevent infection.

That the positive tuberculin reactors have more resistance than non reactors is borne out by the fact, stated on page 107 of the report, that the most virulent septisaemic type of tuberculosis occurred only in 9 per cent. of the cases in the positive group, whereas 24 per cent. of the cases in the negative group were of this type. The comparatively benevolent local glandular tuberculosis was met with most often in the cases that arose in the positive group.

From what has been said previously it is clear that the conditions under which these native boys work on the mines are very different from those to which they and the rest of the African community are normally accustomed. It is of interest, therefore, now to consider how tuberculosis affects them in their home territories.

Recruitment of labour takes place largely from the Transkei district of British South Africa and from Portuguese East Africa. Allan[17], working in the former district, found that in the southern part of the area 77 per

cent. of the population reacted positively to tuberculin, while in the northern part only 44 per cent. gave positive reactions. Boys from the former area going to the mines had a low tuberculosis prevalence, whereas workers from the latter area where the infection rate was considerably lower showed a high tuberculosis prevalence. This, he points out, is in marked contrast with the findings on the mines which showed that the incidence of tuberculosis was lowest in the individuals least tuberculised as shown by the tuberculin test.

He made a further study for 2½ years of the spread of tuberculosis by repatriated mine boys and was impressed by the few instances where there seemed a definite history of infection in the families of these boys. He states that he does not wish to convey the impression that infection of such families does not occur, but that natives in their home surroundings and living their natural lives have a certain degree of resistance.

It is also pointed out in the report[36] - page 244 - that resistance may also be described as capacity to respond to improved conditions and as an example of this the Tuberculosis Gaol at Cradock is cited. Native prisoners who contract or are found to be suffering from tuberculosis are sent to a special gaol at Cradock. The diet is liberal and the work is mostly shoemaking, which is carried on in the open air. The total number of inmates from 1st January till November 1929 was 30. The records

showed that 12 of these had been discharged, 17 were still inmates and 1 had died. One man had been an inmate for over six years and several for three or four years. They looked well, most of them gaining weight, and all were running a fairly chronic course; in other words, all were showing a considerable degree of resistance.

It was found further that acute tuberculosis was more prevalent among natives from the Transkei than from Portuguese East Africa - report page 264. With regard to the former, it is stated that the men have lost the hard discipline of warriors and hunters and the women, with all the attractions of the traders' store close at hand, see the momentary advantage of tinned fruit and tinned milk over wild berries and herbs and the calabash of amasi (sour milk). The children are under nourished and in seasons of drought both adults and children approach the starvation line.

With regard to the natives in Portuguese East Africa, Professor Lyle Cummins is quoted on page 264 of the report as saying: "We were struck during our visit by the plenty and variety of the foodstuffs available. Mealie crops and Kafir-corn crops were reported to be excellent. Ground nuts, sweet potatoes and other vegetables were plentiful. The native women are excellent cooks and make full use of the foodstuffs available. The seemingly good conditions in which the native population lives appear to us much more favourable than those existing in the Transkei".

The Committee came to the conclusion that this apparent relative superiority of native life in Portuguese East Africa was very suggestive as being an advantage in respect of resistance to the acute generalized type of tuberculosis.

The analogies between the African native in his village and the European child in its relatively sheltered environment are pointed out by Cummins on page 255 of the report where he states:-

"Just as some of these healthy but infected European children break down into acute tuberculosis after intercurrent respiratory diseases or on quitting the leisure of childhood for the stress of wage earning, so does a certain proportion of the young African mine recruits break down with acute disease shortly after starting work in the mines.

In the European child under the favourable conditions of home life, the lesions remain 'larval'; the minimal and successive reinfections of childhood and adolescence leading on in the majority of individuals to a stage of compensated subinfection in which the resistance is so high that there is little risk of developing clinical tuberculosis. Thus it comes about that, especially in the well-to-do and well-fed classes, the danger period of adolescence is as a rule safely negotiated and the vast majority of the population remains healthy but tuberculin sensitive and relatively tuberculo-resistant.

In the African native while the initial process of infection is the same, there is no equivalent opportunity for 'acclimatization'. The change from tribal life to the strain of work on the mines is abrupt, nor does the African native yet appear to possess so efficient a mechanism for acquiring tuberculo resistance as does the European adolescent".

The Committee considered - report, page 261 - that the condition of these tuberculin sensitive mine boys represented, so to speak, a long drawn out phase of Borrel's "glandular stage", in which, owing to the favourable circumstances of life in the native territories, the process had not advanced any further towards generalization. In some of them indeed it might have gone a long way towards the healed and compensated tuberculosis that makes for resistance. In others the larval lesions might be close to breaking point. It was found that the more intense and the more recent the glandular infection the greater was the tuberculin sensitivity and the greater the risk of a breakdown into acute tuberculosis. The follow up of the tuberculin tested natives on the rand showed that there was amongst the boys giving a 'positive plus' reaction a decidedly greater liability to develop tuberculosis than was present in the 'positive', 'weakly positive' and 'negative' groups. The disease incidence figures for these groups were 10.3, 6.5, 4.5 and 2.9 per 1000 respectively - report, page 100. A similar condition has been found in Wales by Cummins[38], who has pointed

out that in that country the females show a more marked reactivity than the males, and this is associated with a more acute and severe type of tuberculosis than corresponding male groups.

The fact that:

1. The incidence of tuberculous disease was found to be greatest among mine workers from the area where incidence of tuberculous infection was lowest.
2. Those most recently infected, as judged by their greater sensitivity to tuberculin, were most prone to develop tuberculosis.
3. In its acute form the disease was most common amongst those who came from areas where the general conditions of living were worst.
4. There was no undue prevalence of tuberculosis among the families of mine boys who had been repatriated on account of this disease.
5. These natives in their normal home surroundings were found to show a considerable degree of resistance to the disease,

all suggest very strongly that the spread of tuberculosis among these South African natives is much more due to environmental and economic conditions than to hereditary susceptibility.

If the natives of South Africa are able to develop considerable resistance to tuberculosis while leading their normal home-life, there does not seem to be any reason why the native races of the Gold Coast should not do likewise. If they are exposed to infection under their customary living conditions they should be able to pass through what Cummins[39] calls the dangerous larval period into the stage of compensation and increased resistance without breakdown.

That this is probably taking place is suggested by the nature of their reaction to tuberculin. It has been shown that in South Africa, in India[13], and in Wales[38] individuals who were hypersensitive to tuberculin were more liable to acute and severe forms of tuberculosis than those who did not give such a marked reaction.

The hypersensitivity which Allen[18] reported among the natives of South Africa was not found in the Gold Coast races, although infection was widespread through them. Sewall, de Savitsch and Butler's[29] experiments suggest that with the lapse of time after infection allergy diminishes and immunity increases. It would seem, therefore, that the people of the Gold Coast have passed the stages of hypersensitivity and have advanced further on the road towards immunity than have the South African natives.

It has not been possible to carry out an elaborate investigation of tuberculosis in the Gold Coast, as has been done in South Africa, but from such information as

is available it has been shown that it does not appear to be a rapidly spreading disease in that colony. It has been found, further, that over 60 per cent. of the total population and over 78.9 per cent. of the adult population have been infected with tuberculosis. This is a notifiable disease in all its forms and, judging from the figures obtained in the registration areas, the morbidity rate works out at 4.56 per 1000, or 0.456 per cent. This rate is based on 1193 cases out of a population of 261,198, but too much reliance cannot be placed on it, firstly, because it is extremely unlikely that all persons who live in these areas and who develop the disease, are diagnosed and notified. Secondly, probably a considerable number of people who do not ordinarily reside there come into one or other of the registration areas for treatment when the disease has advanced so far that it is interfering with the power of earning their living. Even allowing for these possible errors it is evident that the vast majority of those who get infected develop sufficient resistance to prevent their infection from passing on into active disease.

It would appear, therefore, that the spread of tuberculosis among the natives of the Gold Coast is not due to any hereditary or racial susceptibility or lack of immunity, and that as time goes on such powers of resistance as have been developed should increase as they have done in other races.

Opie[2] has found in Jamaica, and Ukil[13] in India that the spread of tuberculosis in these countries is associated to a large extent with small and overcrowded dwellings, careless habits and indiscriminate spitting both inside and outside the houses.

Similar conditions of overcrowding, bad sanitary habits and food deficiencies are very common on the Gold Coast, and if in the efforts being made to combat the disease these factors can be satisfactorily dealt with there does not appear to be any reason to assume that lack of immunity or racial susceptibility will be a serious bar to progress.

Summary and Conclusions.

1. 1961 healthy natives of the Gold Coast between the ages of 1 and 70 years have been tested as regards their sensitivity to tuberculin, using the intra-cutaneous method of Mantoux.

2. 61.9 per cent. of males and 57.2 per cent. of females - taking all age groups together - gave positive reactions. They were therefore considered to have been infected at some period by the tubercle bacillus.

3. The incidence of infection increases steadily from infancy to adult life. 34.3 per cent. of children below 15 years, and 76.2 per cent. of adults have been infected and show no symptoms of disease.

4. The tests were carried out in three separate districts in the colony, about 100 miles apart. They showed that infection is spread fairly uniformly through all classes of the population and is not materially affected by occupation or social status.

5. The relation of infection to immunity has been considered. A survey of the literature suggests that in tuberculosis evidence of allergy may be taken as an index of developing resistance.

6. Tuberculosis among the native mine workers of South Africa has been considered, and it has been shown that under their normal conditions of life in their

Summary of Conclusions (contd.)

native territories they develop considerable resistance to this disease. This immunity may break down under conditions of exceptional stress.

7. The native population of the Gold Coast has acquired a considerable degree of immunity as judged by the extent of tuberculous infection among the healthy members of the community. This is sufficient to protect the vast majority of the people while they are carrying on their ordinary occupations.

8. The spread of tuberculosis in the Gold Coast is not due to any special susceptibility or lack of immunity on the part of the native inhabitants, but is to be attributed chiefly to other factors such as overcrowding, bad housing, food deficiencies and indifferent sanitary habits.

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