DIPHTHERIA AMONGST THE BANTU

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CONTENTS

Introduction

Diphtheria amongst the native tribes of Africa has previously been investigated by Borra (1939), Bourguignon, van den Branden & Geens (1939), Cauchi & Smith (1934), Elmes (1941), Emmerson (1941), Ferguson (1932), Fischer (1932), Grasset, Perret-Gentil, Friedman & Gross (1933), Higgins (1930), Kleine & Kroó (1930), Kleine (1940), Nélis, van den Branden & Bourguignon (1936), Ramon & Nélis (1934*a*), Ramon & Nélis (1934*b*), Saleun & Palinacci (1938). While they all agree that the proportion of susceptibles, as judged by the Schick test or estimations of the antitoxin content of the blood, is much lower than in Europeans of corresponding age groups, they differ as to the reason for the infrequency of clinical diphtheria. The majority are of opinion that native immunity is due to frequent subclinical infections by *C. diphtheriae*. The subclinical nature of the disease in natives is variously ascribed to climatic conditions, to the existence of strains of low toxigenic power, to racial immunity (perhaps of the nature of a physiological endogenic maturation without a specific antigen stimulus), to the formation of antibodies in response to heterogenic stimuli, or to the conditions of native social life leading to an early contact with *C. diphtheriae* and consequent production of an early specific immunity.

Most of the inferences have been drawn as the result of two findings: (1) the infrequency of clinical diphtheria, (2) the high proportion of Schick-negative individuals as compared with Europeans of similar age groups. A few investigations into the carrier rates of the populations in question have been done (Bourguignon *et al.* 1939; Emmerson, 1941; Nélis *et al.* 1936). Only Emmerson (1941) has reported on diphtheria among rural natives as opposed to those living under urban conditions. The opportunity for carrying out a complete investigation arose during the course of a general survey of diphtheria on the Witwatersrand. The results are reported in this communication.

Source of material

A rural population consisting of 499 Bantu children of the Tswana tribe was examined. Ages ranged from 1 to 20 years. They were drawn from the Kanye area of Bechuanaland Protectorate. Kanye is a typical large native village with a population of 8000. It lies 40 miles from the railway in semi-arid country and has little direct contact with Europeans. The total European population (administrators, traders, etc.) amounts to

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twenty people. Every month, however, there is a coming and going of some of the adult native males between the village and the gold mines of the Witwatersrand.

The urban population examined consisted mainly of 437 native school children aged 6–17 years living in Alexandra Township. The township lies 8 miles from Johannesburg. The population of 45,000 people is in frequent direct contact with Europeans. It lives under unsatisfactory, overcrowded, unhygienic conditions, and can be accepted as representative of the urban native population in any of the less satisfactory native townships of the Union.

Schick tests were carried out on 903 children (875 of whom returned for reading of the results), and the throats of all were examined for the presence of *C. diphtheriae*.

The clinical cases examined were encountered during the course of routine examination of swabs for *C. diphtheriae* at the South African Institute for Medical Research, Johannesburg.

Bacteriological technique

The throat swabs were immediately planted on Clauberg's tellurite indicator medium (Sutherland & Iredale, 1937). The inoculated plates were incubated for 24 hr. at 37° C. All positive or suspicious colonies were subcultured on Loeffler's medium and examined microscopically after 24 hr. incubation. Pugh's stain was used throughout. From all cultures showing microscopic *C. diphtheriae* McLeod's chocolate tellurite medium was inoculated (Anderson, Happold, McLeod & Thomson, 1931). After 48 hr. incubation the colony type was determined and several colonies picked off. These were examined biochemically in glucose (1%), saccharose (1%) and glycogen (0·5%) in Hiss's serum peptone water using bromcresol purple as an indicator. The cultures were also inoculated into 5% serum broth and examined for growth characteristics in that medium. The readings were made after 48 hr. at 37° C.

The virulence test was carried out on each strain in guinea-pigs according to the intradermal technique of Fraser, MacNabb & Roy (1937), 3–4 strains being tested on each animal and readings made at 72 and 96 hr. All strains of *C. diphtheriae* referred to in this paper were submitted to the above routine procedure, except that the swabs from clinical cases were planted on Loeffler's as well as on Clauberg's medium when received at the laboratory.

Carrier rates

The carrier rates according to age groups are shown in Table 1. These carrier rates contrast strongly with those reported for European school children in the Johannesburg area where no virulent organisms were found in the throats of 462 children (Murray, 1942). Doull & Fales (1923) have shown, however, that the carrier rate for virulent *C. diphtheriae* can vary considerably from one season of the year to another. Over a period of 6 months they report a decrease from 3·3 to 0·87%. For this reason, therefore, the European and Bantu children in the Johannesburg area were all examined in October–November and the question of seasonal variation does not arise in relation to the very different carrier rates between them.

Elsewhere in Africa native children have been examined for the existence of the carrier state. Cauchi & Smith (1934) report twenty cases of severe sore throat in Lagos all negative for *C. diphtheriae*; Bourguignon *et al.* (1939) found seventy morphologically

positive cultures in a series of 744, of which twenty-three strains were tested for virulence and five found to be positive. Nélis *et al.* (1936) were unable to find any carriers amongst a group of natives in the immediate neighbourhood of two European cases in the Belgian Congo. Emmerson (1941) found 0·2% carriers of virulent *C. diphtheriae* among 1005 urban natives in Port Elizabeth and none in 134 rural natives.

From these figures it would appear that though the number of carriers amongst Africans may vary considerably from place to place the carrier state does exist.

The 1 : 1·1 and 1 : 2·5 virulent/avirulent ratios found in this series differ considerably from that of 1 : 13·5 reported by Emmerson (1941) in South Africa; of 1 : 8 in the Philippines (Gomez & Navarro, 1923) and of 1 : 7 in India (Vardon, 1923). It is the reverse of that of 2 : 1 found by Forbes (1932) in London school children, but corresponds more closely to that of 1 : 3·4 reported in white children in the U.S.A. (Gill,

Table 1. C. diphtheriae *carrier rate of rural and urban Bantu children*

	Rural				Urban			
		Positive				Positive		
Age years	Total	Virulent	% virulent	Avirulent	Total	Virulent	% virulent	Avirulent
1/12–3	22	0	0	0	0	0	0	0
6– 8	76	0	0	5	49	1	2	0
9–11	131	5	2·3	3	139	1	0·7	8
12–14	187	9	4·8	9	214	6	2·8	11
15–20	83	2	2·4	1	35	0	0	1
Totals	499	16	3·2	18	437	8	1·8	20
Virulent/avirulent ratio			1 : 1·1				1 : 2·5	

(Excluding the age group 1/12–3 years, since there are no representatives of this group amongst the urban children, statistical analysis of the virulent carrier rates in Table 1 shows χ^2 to be equal to 2·07 and P to lie between 0·1 and 0·2. There is therefore no significant difference in the virulent carrier rates of urban and rural Bantu school children.)

1940) and 1 : 2·3 reported in the U.S.A. by Grossmann (1940). Dudley (1929), in discussing the relative absence of clinical diphtheria in the tropics, has suggested that the great preponderance of the avirulent strains may well be a good indication that in hot climates there is a close adaptation between man and *C. diphtheriae*. This does not appear to be the explanation in South Africa where, though clinical diphtheria is uncommon amongst natives, the virulent/avirulent ratio in this series is similar to that reported amongst white children in temperate climates.

THE SCHICK TEST

The results of the Schick tests carried out are shown in Table 2. Dunn (1929) found Schick-positive rates of 61·6, 44·4 and 33·9% in South African European children of the age groups 6–8 years, 9–11 years and 12–14 years respectively. As compared with these figures the Schick-positive rate for the Bantu children is very much lower.

This has been the finding of other workers and confirms their contention that native races in Africa are less susceptible to diphtheria than are the Europeans (Grasset *et al* 1933; Kleine, 1940; Cauchi & Smith, 1934).

CLINICAL DIPHTHERIA

During two years' work in Bechuanaland Protectorate at a native hospital serving a rural population of approximately 33,000, no cases of diphtheria were observed by me though particular search was made for the disease. This paucity of cases of clinical diphtheria is

the general finding in all the High Commission Territories of South Africa as Table 3 shows.

Clinical diphtheria amongst urban natives, on the other hand, is less uncommon than has been generally supposed. Over a period of 3 years (1939–41) the strains isolated from native cases in Johannesburg were fully investigated and, where possible, clinical details of the cases were obtained. During this period 109 cases were personally encountered. Owing to difficulties in obtaining clinical details about native cases they are available for only ninety. They are summarized in Table 4.

Table 2. *Schick tests in* 875 *Bantu children*

Age years	Rural			Urban		
	Total	Positive	% positive	Total	Positive	% positive
6– 8	68	8	11·8	128	22	17·2
9–11	103	12	11·7	114	13	11·4
12–14	174	10	5·8	184	24	13·0
15–17	79	4	5·1	25	3	12·0
Totals	424	34	8·0	451	62	13·7

(Statistical analysis of Table 2 shows an apparently significant difference in the Schick immunity of the urban and rural children ($\chi^2 = 7{\cdot}35$ and P lies below 0·01), but this is probably due to the inequality of the numbers aged 6–8 years in the two groups. The difference between the two groups, however, is in no way comparable to the difference existing between Bantu and European children of the same age groups.)

Table 3. *The incidence of clinical diphtheria in the Bantu population of Basutoland, Bechuanaland and Swaziland Protectorates during* 1935–9 (*Annual Medical and Sanitary Reports*, 1935–9)

Year	Cases of diphtheria		
	Basutoland (1936 population =559,273)	Bechuanaland (1936 population =260,064)	Swaziland (1936 population =153,270)
1935	15	10	2
1936	8	2	5
1937	16	3	1
1938	137	6	2
1939	14	—	—

Table 4. *Clinical types of ninety cases of diphtheria in natives*

Classification	No. of cases	Deaths
Rhinitis	16	2
Mild faucial	36	1
Moderately severe faucial	18	3
Severe faucial	8	3
Mainly laryngeal	12	4
Totals	90	13

Excluding one case of moderately severe faucial diphtheria of which the ultimate outcome is unknown, there was a case mortality rate of 14·4%.

The age distribution of the sixty-six clinical cases in which this detail could be obtained with reasonable accuracy showed twenty-one cases (31·8%) in the 0–2 years group, fourteen (21·2%) in the 2–5 years group, fourteen (21·2%) in the 5–10 years group, seven (10·6%) in the 10–15 years group, and ten (15·2%) aged 15 years or over.

BACTERIOLOGICAL TYPES

The bacteriological types were determined according to the criteria indicated in a previous communication (Murray, 1942). The distribution is shown in Table 5. Owing to the fact that many swabs reach the laboratory with no indication as to the race of the patient, full bacteriological details were obtained in only seventy-five of the cases. Eight strains were isolated from carriers during the same period. These have been included in columns 3 and 4 of Table 5, which also refer to strains isolated from sixty-two carriers during the examination of 936 native school children.

Table 5. *Bacteriological types of* C. diphtheriae *recovered from seventy-five cases and seventy carriers of diphtheria*

	75 clinical cases		70 carriers		Total				
	Virulent	Avirulent	Virulent	Avirulent	Virulent	% virulent	Avirulent	Total strains	% total strains
Mitis	41	25	26	37	67	51·9	62	129	89
Gravis	4	0	1	5	5	50·6	5	10	6·8
Atypical	3	2	1	0	4	66·7	2	6	4·1
Totals	48	27	28	42	76	52·4	69	145	—

As in the case of Europeans on the Witwatersrand this series shows a high proportion of *mitis* strains (Murray, 1942). It also confirms that in this area a virulence test is necessary irrespective of the type of organism recovered (Murray, 1942). This contrasts with the findings in some parts of Europe (Cooper, Happold, McLeod & Woodcock, 1936; Zinnemann & Zinnemann, 1939) and of Australia (Gregory, 1937) where avirulent *gravis* strains are scarcely ever encountered. But it is in accord with the findings of Frobisher (1940) in the U.S.A. who frequently found *gravis* strains to be avirulent.*

DISCUSSION

From these findings it is apparent that carriers of virulent *C. diphtheriae* are equally common among Bantu children whether under rural or urban conditions. In comparison with European school children the carrier rate equals that found during an epidemic of diphtheria and is in strong contrast with the absence of virulent diphtheria carriers among 462 white children in Johannesburg schools (Murray, 1942). The Schick-positive rate shows a significant difference between rural and urban Bantu children, but is low in both compared with European children of the same age. The distribution of the bacteriological types shows no significant difference between the two groups of native children and is similar to that found in European clinical cases on the Witwatersrand.

On turning to the clinical aspect, however, there is a disparity between the rural and urban groups. Clinical diphtheria among rural Bantu in Bechuanaland Protectorate is extremely uncommon. Its highest incidence in any of the High Commission Territories during 1936–9 occurred in 1938 in Basutoland when it reached 0·25 per 1000. For all three Territories the average annual diphtheria incidence is 0·048 per 1000. This may be compared with an attack rate of 1·84 per 1000 for England and Wales in 1930 (Forbes, 1932). Higgins (1929) reported an incidence of 0·59 per 1000 for non-Europeans (in-

* It should not be overlooked that Frobisher included in his category of *gravis* strains many which were frankly atypical and it is not clear how far his statement refers to such strains. It is only in connexion with 'typical' *gravis* strains that the claim of constant pathogenicity has been made. The *gravis* strains described in this paper have been classified on the basis of McLeod's description (Anderson *et al.* 1931).

cluding Coloured as well as Bantu) compared with 1·23 per 1000 for Europeans in Cape Town.

In Johannesburg during three years I encountered 109 cases. These were mainly cases which were hospitalized and were only those whose swabs came under my personal observation in the laboratory. Their number cannot, therefore, be regarded as indicative of the true incidence of the disease in the native population of Johannesburg, which totals 230,000. The majority of native cases are not swabbed, notified or hospitalized. It is probably true to say that the majority of them never see a doctor.

The high case mortality rate (14·4%) in an area where *mitis* strains predominate is probably not a reflexion of any unusual virulence of the organism though Europeans on the Witwatersrand also show a high case mortality rate (Murray, 1942). Most of the native cases are seen only at a late stage of the disease. Owing to the expense involved only those who are seriously ill consult a doctor. Another factor is the poor nutritional state in which these people exist, thus further loading the death-rate when diphtheria supervenes.

The demonstrated difference in incidence of clinical diphtheria in urban and rural Bantu who have similar carrier and Schick-positive rates helps towards a clarification of the problem as to why this disease should be relatively uncommon among native races in Africa. Of the various hypotheses which have been propounded the following may be considered.

(1) *Climatic conditions.* Dudley (1929) pointed out that the results of Taliaferro in Honduras 'suggested that hot weather in itself cannot have much effect in stimulating or accelerating the production of natural antitoxic immunity to diphtheria'. The results of this investigation also lead to the conclusion that climatic conditions, *per se*, have little to do with the degree of immunity to diphtheria. Only 13·7% of 451 urban Bantu children aged 6–17 years were Schick-positive while 69% of 197 Europeans aged 19–31 years and living under identical climatic conditions were Schick-positive (Murray, 1942). Also, rural and urban natives living under almost identical climatic conditions show a different incidence of clinical diphtheria. Dudley also suggested that 'lack of a yearly periodic disturbance in the balance between host and parasite results in a closer mutual adaptation between them. This close adaptation would mean a high Schick immunity in combination with a high ratio of carriers to cases of diphtheria.' These conditions of diphtheria exist among the natives of the Johannesburg area where, however, there is a very definite seasonal variation in the weather. The mean maximum and minimum temperatures in Johannesburg are 80 and 56° F. in December and 62 and 37° F. in June. The average humidity in August is 40%, while that in February is 76%. Therefore the low morbidity associated with a high carrier rate and high Schick immunity among the natives examined cannot be ascribed to any absence of seasonal weather variation leading to mutual adaptation of host and parasite.

(2) *Strains of low pathogenic power.* Saleun & Palinacci (1938) have suggested the existence of strains of *C. diphtheriae* of low toxigenicity in French Equatorial Africa as a possible explanation of the infrequency of clinical diphtheria. Doull (1930) makes a similar suggestion regarding the diphtheria bacillus in the tropics. In South Africa none of the 149 virulent strains encountered in this investigation gave a doubtful or weakly positive result in the virulence test, but the toxin-producing power was not tested in any of them.

(3) *Physiological endogenic maturation without a specific stimulus.* Heinbecker & Irvine-Jones (1928) investigated the lack of susceptibility of Polar Eskimos to diphtheria and concluded that 'their immunity does not depend on infection by the diphtheria bacillus', but Dudley (1929) points out that their results are capable of explanation by postulating the presence of *C. diphtheriae* in the community 12 years prior to the investigation. That this explanation is the true one is borne out by subsequent work amongst the Eskimos by Wells (1933), who found carriers of at least weakly virulent strains and concluded that their immunity to diphtheria is specific rather than the result of maturation phenomena.

It seems reasonable to assume that in the presence of numerous carriers of virulent *C. diphtheriae* immunity amongst the Bantu is also due to specific stimulus rather than to hereditary endogenic maturation.

Dudley (1929) reviews the subject of the genetic factor in Schick immunity and Hirschfeld's evidence in its favour and concludes 'that this heredity factor has an insignificant bearing on the degree of immunity possessed by a community as a whole, compared with the concentration, distribution and persistence of diphtheria bacilli in the environment'.

(4) *Heterogenic stimuli.* Kleine & Kroó (1930) considered the possibility that heterogenic stimuli might lead to immunity to diphtheria amongst the Bantu, but this seems an unnecessary assumption in view of the presence of the specific antigen. In a later paper Kleine (1940) came to the conclusion that native immunity arises from living in close contact in a heavily infected atmosphere.

Table 6. *Schick-positive rate in native races*

Author	Race	Age years	No. of subjects	% Schick-positive
Gomez, Navarro & Kapauan (1922)	Filipinos	6–10	79	12·7
Fletcher (1927)	Malays	5–10	118	9·3
Grasset *et al.* (1933)	Bantu	7–10	66	9·09
Borra (1939)	Ethiopians	5–10	176	14·8
Murray (1942)	Bantu	6–11	413	13·3

(5) *Environmental factors.* From many reports it is clear that not only is clinical diphtheria uncommon in the tropics but also that immunity in native races, as judged by the results of Schick testing, is acquired at an early age.

Table 6 illustrates this point. These figures may be compared with a Schick-positive rate of 84·1% amongst 1880 English school children aged 5–10 years (Fulton, Moore, Taylor, Wells & Wilson, 1942).

It follows that the majority of clinical infections will occur during the very early years of life. Fletcher (1927) reports that among Asiatics the greatest number of cases occurred before the fifth year of life. In the series reported here 53% of the cases in which the age could be ascertained occurred between 3/12 and 5 years of age and a similar finding was reported by Grasset *et al.* (1933).

It is clear that the curve of immunity to diphtheria in the Bantu has the same general shape as in a European population (Grasset *et al.* 1933). The carrier rate found in this investigation proves the presence of the specific antigenic stimulus assumed by Grasset and his colleagues. They explain its intensive early immunizing power as being due to the close contact existing under the conditions of native life, probably, as Dudley (1929) postulates, giving many native children active immunity without symptoms before they

even lose their maternal passive immunity. This would also explain the occasional occurrence of clinical cases during the first few years of life, clinical diphtheria developing in those children in whom antibody formation is least efficient. Such an explanation of the relative infrequency of clinical diphtheria assumes, however, that the incidence of diphtheria is inversely proportional to the density of a population if we regard increasing density of population as equivalent to increasing closeness of social contact. Forbes (1932), in an analysis of diphtheria and population density in London boroughs, found the opposite. The more densely populated areas had a greater diphtheria attack rate than those in which the population was less dense. He quotes similar findings by De Rudder in Berlin and Flugge in Breslau. In urban areas in South Africa the native lives under more crowded and congested conditions than could be found in any European slum, and even in rural areas there is overcrowding, for, as Dudley (1929) points out, 'native populations, even if sparsely distributed as regards square miles, often live in the closest of contact in crowded, cramped, unventilated huts'. On the basis of Forbes's findings, therefore, the morbidity of diphtheria should be greater amongst the crowded native population than amongst the better-housed Europeans—the reverse of what has in fact been found.

According to the figures of Grasset *et al.* (1933) 47·05 % of the native population aged 4/12–3 years is Schick-positive. In spite of nearly half the children in this age group being susceptible to diphtheria, the disease is relatively uncommon as compared with Europeans of the same age living under better urban conditions and is practically unknown under rural conditions. This suggests that although the environment is largely responsible for the high proportion of Schick-negatives in the Bantu, there is also a racial factor. It is probable, in view of the shape of the immunity curve (Grasset *et al.* 1933), that this racial immunity amounts to no more than a greater ability of the Bantu to form antitoxin quickly as compared with Europeans. It does not follow that this racial difference is necessarily a genetic one. It may be a function of the environment. Dudley (1929) states that 'the fraction of slow antitoxin producers becomes progressively smaller the more experience the whole group has had of diphtheria bacilli', and it has been shown that diphtheria carriers are common amongst the Bantu while their mode of life makes for frequent and intimate contact.

This view finds some support in the work of Black (1934), who observed that the morbidity is higher in negroes in the first year of life as compared with whites in the U.S.A. and lower in childhood. There is also a higher diphtheria mortality rate amongst negroes during this period in life which suggests a weeding out of the individuals with the poorest immunity response, while the remainder rapidly become immune largely because of their racial facility in forming antitoxin in response to the *C. diphtheriae* in their environment. Black (1934) found that the proportion of individuals reacting to immunizing injections of toxin-antitoxin by prompt formation of antitoxin was slightly higher in the negroes than in the white children. He thought that more extensive observations were necessary to show whether or not this is a characteristic racial difference. Frost (1928) found that the negro attack rate for clinical diphtheria is only one-third that of whites with the same carrier rates and Schick immunity and living under similar conditions in Baltimore, but he says the observations 'are not extensive'. He infers, however, that in the same community the two races react differently to the infection.

This inference, however, does not explain the different morbidity rates of urban and

rural natives. Grasset *et al.* (1933), on the basis of serological findings, were also of the opinion that 'mere contact with civilization does not exert a reinforcing influence on the development of diphtheria immunity. Rather the reverse.' This difference may be due to the poorer dietetic and hygienic conditions which, broadly speaking, prevail in native urban locations as compared with native villages. There is also the possibility of a proportion of the urban natives having a variable amount of white blood which (if racial immunity is accepted) would render them more liable to contract diphtheria than the pure-blooded natives. Every effort was made in this investigation to exclude all coloured persons and include only pure-blooded Bantu, but it cannot be absolutely certain that this has been successfully accomplished. In this connexion it is of interest to note that it was reported by the Medical Officer of Health, Wellington, New Zealand (1929), that no case had been seen in Wellington of a pure-blooded Maori suffering from bacteriologically proved diphtheria. The only Maori diphtheria patients admitted to Napier Hospital had European blood. In a later report by Turbott to the Director-General of Health, New Zealand, further confirmation of this point is given (*Lancet*, 1931). In 2000 children aged 5–15 years including full, $\frac{3}{4}$, $\frac{1}{2}$ and $\frac{1}{4}$ bred Maoris he found the Schick test positive in $24 \pm 1{\cdot}4\%$ of the $\frac{1}{2}$ and $\frac{1}{4}$ breeds, but only $10 \pm 0{\cdot}5\%$ positive in pure and $\frac{3}{4}$ bred Maoris. White children in the same district showed $63 \pm 5{\cdot}0\%$ Schick-positive. The Pirquet test showed no appreciable difference between the different Maori groups. The inference is drawn that the observations favour the hypothesis that herd immunity for diphtheria is higher in pure Maoris than in European cross-breeds as a result of special racial, or genetic, characters and not because the pure Maoris were subjected to more intense or frequent contact with the specific micro-organisms. 'The coloured races differ from the white in having a higher degree of racial immunizability which enables them to respond more rapidly to, and perhaps to smaller doses of, toxin than "Europeans".'

Higgins (1930), in Schick testing 160 non-European children (mainly Cape Coloured of mixed native and European blood) aged 6–15 years, found 29% positive. This is almost midway between the proportion found Schick-positive by Grasset *et al.* (1933) and by me in pure-blooded Bantu of the same age and that of 51% in Europeans as reported by Higgins, which gives further support to the possibility that racial ability to form antitoxin as well as environment determines the Schick immunity and non-susceptibility to clinical diphtheria of the Bantu.

An investigation into diphtheria amongst the coloured population of the Union might throw further light on this subject, and it is proposed to carry this out as opportunity offers.

SUMMARY AND CONCLUSIONS

1. A total of 499 rural and 437 urban Bantu school children were examined for their diphtheria carrier rate and Schick immunity.

2. Clinical diphtheria amongst the rural and urban Bantu was also investigated.

3. The virulent *C. diphtheriae* carrier rate was found to be 3·2% in the rural children and 1·8% in the urban with virulent/avirulent ratios of 1 : 1·1 and 1 : 2·5 respectively.

4. The Schick-positive rate in children aged 6–17 years was found to be 8·0% in rural children and 13·7% in urban.

5. Clinical diphtheria was rarely encountered under rural conditions, but was more common in urban natives.

6. The case mortality rate in eighty-nine clinical cases was 14·4%.

7. 53% of the clinical cases occurred in the 0–5 years age group.

8. 89% of the strains recovered belonged to the *mitis* type. No intermediate strains were encountered.

9. The reason for the infrequency of clinical diphtheria amongst the Bantu is discussed and various theories are reviewed.

10. It is concluded that the infrequency of clinical diphtheria is partly due to the environment, but that there is also a racial factor. It is suggested that the racial factor lies in an ability to produce antitoxin quickly. There is not sufficient evidence in this investigation to show whether the racial factor is genetic, but in view of Turbott's work amongst the Maoris it is suggested that the racial factor in immunity to diphtheria may be genetic amongst the Bantu also.

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