

The *Enterobacteriaceae* of South African Baboons

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SUMMARY

The results of routine rectal swab examinations, carried out on 776 baboons on the day of arrival at the colony and also on 394 animals which subsequently developed diarrhoea, are described. A dramatic increase was observed in the incidence of both pathogenic and non-pathogenic organisms during the diarrhoeic phase. The significance of bacterial species which are not pathogenic under normal circumstances as the possible cause of disease in animals subjected to stress is discussed.

S. Afr. med. J., 50, 994 (1976).

Brede¹ described the pathogenic *Enterobacteriaceae* found during routine examination of the intestinal flora of 851 baboons soon after capture of the animals. Geldenhuys *et al.*² have described the pathogenic organisms found in the gastro-intestinal tracts of baboons which died of uncontrollable diarrhoea.

In this article we present the results of routine rectal swab examinations carried out on 776 baboons (*Papio ursinus*) on the day of arrival in the colony, and also on 394 animals which subsequently developed diarrhoea.

MATERIALS AND METHODS

Rectal swabs taken from baboons were plated onto MacConkey agar (Oxoid CM 7) and SS agar (Oxoid CM 99) and inoculated into tetrathionate broth. After 18 hours' incubation at 37°C subcultures were made from the tetrathionate broth on MacConkey agar and SS agar. Colonies of *Enterobacteriaceae* were picked off these plates and inoculated into tryptone water (Difco), incubated for 3-4 hours at 37°C, and then identified by means of appropriate biochemical and serological tests.

RESULTS

The results of cultures of rectal swabs and stools taken from 776 baboons (*Papio ursinus*) on the day of arrival at the colony and from 394 animals suffering from diarrhoea are shown in Table I. *Escherichia coli* was the most prevalent organism, followed by *Streptococcus faecalis* and *Proteus mirabilis*. *E. coli* serotypes, of which types 0112, 0114 and 0128 were commonest, were isolated from 62 of the 776 animals (8.0%). *Salmonella* species

were isolated from 45 animals (5.8%). *Shigellae*, however, were found in only 5 animals (0.64%), 2 of which were infected with *S. flexneri* 4a, 2 with *S. sonnei* and 1 with *S. boydii*.

All animals were healthy on arrival at the colony. However, diarrhoea frequently developed in the baboons from about 5 days to 3 weeks after arrival. Enteropathogenic *E. coli* were isolated from 88 of the 394 animals (22.33%) suffering from diarrhoea. The main serotypes were 026, 0112, 0114 and 0128 which, apart from type 026, correspond with the serotypes recovered from animals on the day of arrival. *Salmonella* species were found in 64 animals (16.24%). Of these, *S. sunsvall* was found in 48 animals (12.18%). In the majority of these animals the diarrhoea lasted for only 1 day and very occasionally for 2 days or more. *Shigella* species were cultured from 68 animals (17.25%). *S. flexneri* 4a was the most prevalent type, followed infrequently by *S. flexneri* 2a. These recognised pathogens were therefore isolated in 55.9% of the diseased animals. In addition, *Klebsiella* species, which are now accepted as causal agents of enteritis in infants, were isolated from 209 (55%) of the sick animals.

The changes which occurred in the bacterial flora of the diarrhoeic animals are of interest. *P. mirabilis* was found in 26.67% of animals on arrival, but this incidence increased to 86.25% in animals suffering from diarrhoea. *Klebsiella*, which was found in 9.15% of animals on arrival, was present in 55.05% of diarrhoeic baboons. *Enterobacter* species, however, increased from 12.10% to only 14.10%, while *Shigella* species increased from 0.64% to 17.25%.

DISCUSSION

According to Honjo,³ during the quarantine of wild monkeys and apes first consideration must be given to the diagnosis of infection, followed by disinfection and isolation. Secondly, the quarantine should be carried out from the standpoint of zoonosis, and attention must be paid to prophylaxis and treatment. High mortality among monkeys in captivity revolves around a vicious cycle of malnutrition, stress and infectious disease.⁴ Good *et al.*⁵ stated that many of their animals, which appeared healthy on arrival, carried enteric pathogens (*Shigella*). Two to three weeks after arrival, the combined stress of transport plus acclimatisation to a new environment and diet provided the conditions for an outbreak of shigellosis.

In his study on 851 free-living chacma baboons (*Papio ursinus*) Brede¹ found an incidence of 13.75% of salmonellae, 19.38% of shigellae and 30.32% of enteropathogenic *E. coli*. These figures are considerably higher than the 5.79%, 0.64% and 8.00% found for salmonellae, shigellae and enteropathogenic *E. coli* in baboons, on arrival, in our present series. The difference in the incidence rates may be explained on the basis of different areas from which the animals were received.

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Date received: 15 December 1975.

TABLE I. ENTEROBACTERIACEAE ISOLATED FROM RECTAL SWABS TAKEN FROM BABOONS ON ARRIVAL AND FROM DIARRHOEIC BABOONS

Organism	On arrival		Diarrhoeic baboons	
	Number	%	Number	%
<i>Alcaligenes faecalis</i>	48	6,18	64	16,24
<i>Citrobacter</i>	—	—	2	0,51
<i>Enterobacter</i>	94	12,10	162	41,10
<i>Escherichia coli</i> (non-pathogenic)	621	80,02	371	94,16
<i>Escherichia coli</i> (enteropathogenic serotypes)	62	8,00	88	22,33
<i>Klebsiella</i>	71	8,15	209	55,05
<i>Proteus mirabilis</i>	207	26,67	341	86,55
<i>Proteus morgani</i>	58	7,47	86	21,83
<i>Proteus rettgeri</i>	38	4,89	39	9,90
<i>Proteus vulgaris</i>	44	5,67	66	16,75
<i>Providencia</i>	—	—	8	2,03
<i>Pseudomonas</i>	14	1,80	77	19,54
<i>Salmonella</i>	45	5,79	64	16,24
<i>Shigella</i>	5	0,64	68	17,25

The interval between capture and the date of arrival of the baboons at the colony varied from a few days to about 3 weeks. It is possible that many of the animals with positive bacterial cultures acquired infections during contact with their captors. Under stress of capture and transport, such infections — even if previously quiescent — frequently become active.⁶ Other problems are the possibility that the carrier will infect others unable to resist the pathogen, and that the carrier himself will become suddenly and acutely ill.⁷

The incidence of shigellae in our baboons increased from 0,64% on arrival to 15,48% in animals suffering from diarrhoea. Kaufmann⁸ in his zoonosis survey reported that diarrhoeic disease was the most commonly reported illness in both 1970 and 1971, and that *Shigella* was the most frequently specified cause of diarrhoea. This does not appear to be the case in the South African baboon, since the incidence of *Shigella* infection in this baboon is relatively low, even in animals with active diarrhoea.

Lapan and Yakovleva⁹ reported on the low susceptibility of baboons to this disease, although a high percentage of carriers was found among their newly imported baboons. Pinkerton *et al.*¹⁰ reported that dysentery in adult baboons (*Papio cynocephalus*) was rare in their colony. Similarly, the disease is usually limited to solitary cases among adult and conditioned animals in our colony. *Salmonella* isolations show that out of 45 animals carrying salmonellae, 35 carried *S. sunsvall*. The diarrhoea in these animals was of a mild degree and only lasted from 1 to 2 days. Brede¹ was of the opinion that the high incidence of *S. sunsvall* indicated endosymbiosis of this organism in the chacma baboon. In animals under stress in captivity, however, it triggered gastro-enteritis and bacteraemia, especially in baboons undergoing immunosuppressive therapy. In the present study the animals developed diarrhoea during the conditioning period and before any experimental procedures involving immunosuppression

were performed. This may explain the milder form of diarrhoea, which was apparently triggered by the stress of captivity.

Although enteropathogenic *E. coli* commonly cause illness and death in infant animals, their presence in such a high percentage of young and adult baboons is disturbing. Their role as aetiological agents in diarrhoea in baboons is not clear. With the ability to transfer episome material from one serotype to another, it is likely that species-specific *E. coli* may lose their specificity. Rowe and Gross¹¹ have pointed out that *E. coli* isolated from patients in an outbreak of infantile enteritis may possess O antigens which are not included in the full international serotyping scheme and that new groups may become established.

It is likely that this may also apply to the *E. coli* isolated from our baboons and that many of the strains of *E. coli* isolated would be classified as new enteropathogenic serotypes. A new enteropathogenic serotype could, therefore, arise, in which case the animals may acquire their infection by ingestion of food and water contaminated by handlers and by other animals.¹²

A dramatic increase was observed in the incidence of some organisms during the diarrhoeic phase. *P. mirabilis* and *Klebsiella* organisms showed very marked increases in incidence. *Proteus* strains have been isolated from human stools in acute dysentery and from blood in bacteraemia, and *P. morgani* may produce a disease resembling *Shigella* dysentery.¹³ *Klebsiella* has also been found to produce diarrhoea in infants.¹⁴ In animals with a strongly reduced immune capacity, enteric bacterial infection may be caused by bacterial species that are not pathogenic under normal circumstances.¹⁵ Many experimental procedures constitute trauma, and have the potential to reveal a latent infection.⁷ Similarly, non-pathogenic organisms may be the cause of disease in newly acquired baboons subjected to the stress of capture, transport, new diet and other factors associated with capti-

vity. This may be the cause of some of the cases of diarrhoea for which no specific pathogenic causal organisms could be found.

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Society for Endocrinology, Metabolism and Diabetes for Southern Africa: Abstracts of Papers

The following are abstracts of papers read at the 16th Annual Congress of the Society for Endocrinology, Metabolism and Diabetes for Southern Africa, held in Johannesburg on 8-10 September 1975.

EFFECTS OF THYROTROPHIN-RELEASING HORMONE ON PROLACTIN, PLASMA RENIN ACTIVITY, WATER AND ELECTROLYTE EXCRETION IN NORMAL MALES

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The injection of ovine prolactin (PRL) in normal man causes retention of water, sodium and potassium. Human prolactin (HPRL) has been implicated in nocturnal antidiuresis and its absence may allow saluresis and the 'escape phenomenon' in endocrine hypertension. We studied the effects of thyrotrophin-releasing hormone (TRH)-stimulated PRL release on electrolyte, water and plasma renin activity (PRA) in normal male volunteers to clarify the role of HPRL in electrolyte and water excretion. Seven subjects who were given 200 µg TRH with stimulation of prolactin (HPRL) had no alteration in urinary sodium, potassium and free water clearance. In addition, PRA remained unaffected by the elevation in HPRL. We conclude that acute elevation of HPRL does not alter water and electrolyte and PRA homeostasis in normal man, or else counter-regulatory mechanisms by unknown concomitant effects of TRH or TSH might be operative.

HYPERLIPIDAEMIA IN RENAL TRANSPLANT PATIENTS

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The prevalence of hyperlipidaemia was investigated in 94

renal transplant patients. Mean plasma triglyceride concentration was 199 ± 19 mg/100 ml (SEM) and mean plasma cholesterol 261 ± 6 mg/100 ml (normal 150 mg/100 ml and 250 mg/100 ml respectively). Mean basal insulin was 13 ± 0.6 µU/ml, and mean plasma insulin 1 hour after an oral glucose load was 55 ± 5 µU/ml (control values 6 ± 0.6 and 75 ± 1.0 µU/ml respectively). When assessed separately, no significant correlation could be found between the plasma lipids and factors likely to affect lipid levels, such as basal insulin and serum creatinine concentrations, body weight, carbohydrate tolerance and current steroid dose, although upward trends in lipid values were noted in each case. A group of 11 patients loaded with the above hyperlipidaemia-predisposing factors showed significantly higher mean plasma triglyceride and cholesterol values than a second group of 17 patients selected to eliminate these factors (plasma triglyceride 307 ± 73 versus 134 ± 11 mg/100 ml, $P < 0.01$; and plasma cholesterol 314 ± 24 versus 241 ± 10 mg/100 ml, $P < 0.005$). It is concluded that the genesis of hyperlipidaemia in renal transplant patients, though unclear, is likely to be multifactorial.

HYPERLIPIDAEMIA IN BLACKS WITH THE NEPHROTIC SYNDROME

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Plasma lipid and lipoprotein concentrations, basal insulin and insulin levels 1 hour after oral glucose load, blood glucose levels, serum albumin and creatinine, and 24-hour urinary protein concentrations were measured in 14 Black subjects with the nephrotic syndrome. Mean plasma triglyceride was 363 ± 132 mg/100 ml (SEM), and mean plasma cholesterol 356 ± 58 mg/100 ml (upper limit of normal 150 mg/100 ml and 250 mg/100 ml, respectively). Mean basal and 1-hour post-