

GOUT IN THE MAORI

BY

G. A. Q. LENNANE, B. S. ROSE, AND I. C. ISDALE

From the Queen Elizabeth Hospital, Rotorua, New Zealand

To extend knowledge of the rheumatic diseases epidemiological studies are of considerable importance, and these are of particular interest where disease incidence in different racial groups living side by side can be compared. Such opportunity occurs in New Zealand, especially in the northern part of the North Island where live most of the Maori population. At Rotorua in the centre of this area is the Queen Elizabeth Hospital, the national hospital for rheumatic diseases.

Clinical experiences at the hospital early suggested that the pattern of rheumatism differed in the Maori and European populations; the Maori people appeared to be affected less by rheumatoid arthritis (Rose, 1955), but more by rheumatic fever and gout than the inhabitants of European stock. These apparent differences were considered worthy of further investigation not only because of their intrinsic interest but also because of their possible bearing on aetiological concepts of these diseases. Further, the rate of natural increase of the Maori population is now much greater than that of the non-Maori, and the subject is one of public health importance.

This paper reports one aspect of these investigations: the prevalence of gout in the Maori population. Because the findings are so much at variance with a widely-held opinion that—"gout is much commoner among certain races, *e.g.* the English and German; it is very uncommon among native races" (Hunter, 1956), it is thought worth while to record our results.

Historical Background

Apart from a short visit by the Dutch explorer, Abel Tasman, New Zealand remained unknown to European civilization until its re-discovery by

Captain Cook in 1769 (Cook, 1951). At that time Cook found the country inhabited by a Polynesian people who appeared to be remarkably healthy, with a well-organized tribal system. Originally a sea-faring race, the Maori had settled the country in a series of migrations from the central Pacific from approximately the 9th century, culminating with a major settlement in the mid-14th century. An intelligent war-like people with a stone-age culture, they lacked any writing technique, and despite the prodigies of memory of which they were capable, much of the minutiae of their daily life is not known, though traditionally there is little mention of ill health among them (Buck, 1949).

In their voyages of migration, the Maori brought to New Zealand a number of plants and the only land mammals, the Maori dog and the Polynesian rat, which were found in New Zealand at the time of Cook's visit. Despite this lack of mammals, the pre-European Maori diet was essentially sound and well balanced in proteins, carbohydrates, and fats (Turbott, 1940). Water was the only and universal drink. The major sources of protein were the plentiful birds, and the fish and molluscs abounding in lakes, rivers, and sea. Both at catching and preserving these the Maori were expert, and in particular their skill in fishing provoked incredulous admiration from many early explorers (Best, 1929, 1942). Dogs and rats where available formed a less important part of the diet, and the bodies of enemies slain in battle were also consumed. Such cannibalism was morally accepted, being prompted not by lack of animal protein but by motives of revenge and ancient tradition (Cook, 1951; Banks, 1958). The main sources of carbohydrate were the introduced kumara (sweet potato) and the rhizome of the native bracken fern; no refined sugars were available and brewing unknown. Food was cooked by broiling on open

fires or by steaming in Polynesian earth ovens, neither method needing the addition of cooking fat. Sir Joseph Banks, a trained naturalist with Captain Cook, commented on the Maori, both young and old, as being blessed with sound health in a very high degree. Although familiar with gout, he did not record its occurrence, nor indeed that of any other rheumatic illness.

Within the next 50 years, with the advent of traders, sealers, and missionaries, came European diseases, muskets, and alcohol, which combined to produce a rapid disruption of the former way of life and a great deterioration in Maori health (Savage, 1807). In particular, smallpox, measles, scarlet fever, enteric fever, tuberculosis, and gonorrhoea led to great mortality, the last three diseases remaining common until very recent times. In evidence submitted to Commissioner Bigge's inquiry in 1821, Dr. Fairfowl stated that the most prevalent diseases were pneumonia, inflammation of the bowels, dysentery, rheumatism, ulcers, and venereal disease, but did not further specify the type of rheumatism (Fairfowl, 1908).

Disease, the wars of the 1860s, and the subsequent dispossession of much of their best land led to a marked decline of the Maori people; not only were their numbers decimated, but the survivors were also disorganized and undernourished. Newman (1880), in a valedictory presidential address to the Wellington Philosophical Society, commented on their "sparse diet of fern root and dried eels". He mentioned as common, "rheumatism in all its forms, consumption, and scrofula", but specifically stated that "no Maori ever had gout". That nutrition was then unsatisfactory is indicated by his mention of "Maori leprosy", a malnutrition syndrome seen most frequently among the poorer tribes, which responded rapidly to improved food and care.

With the introduction of domestic animals, potatoes, and cereals, there was a rapid change in diet. Fish and birds were replaced by a smaller amount of tinned and fresh meats and there was a marked increase in the amount of carbohydrate, particularly white sugar, potatoes, and cereals. The Maori early acquired a lasting fondness for beer. Milk and cheese consumption was always low among them, though as much butter was eaten as economic status allowed (Turbott, 1940).

In 1902, Dr. A. S. Wohlmann was appointed as the first medical officer to the Government Sanatorium at Rotorua in the centre of the hot mineral spring area. In his work on the mineral waters of New Zealand (Wohlmann, 1914), he recorded gout or "goutiness" in 16 per cent. of his Sanatorium cases, and in 30 per cent. of his private cases. A later

medical officer at the same institution, Dr. A. T. M. Blair, commented that many of the patients with gout would later have been classified as suffering from "fibrositis" (Blair, 1951). Nevertheless, Wohlmann mentioned two Maori patients exhibiting tophi, though he subsequently commented on the rarity of both acute and tophaceous gout in the Maori race (Herbert, 1921). This is the earliest report of gout in a Maori.

Until the last 25 years the economic status of the Maori remained low, and in this period the usual diet was low in first-class protein, vitamins, and protective foods.

The old methods of cooking had been supplanted by frying or baking in electric or coal ranges and this, with the Maori preference for fat meat, led to an increase in fat intake. In 1942, a dietary survey (McLaughlin and Wilson, 1945) was conducted among Maori families of the Arawa tribe. At that time, with full employment and generous children's allowances, the Maori was in a much more favourable financial position than ever before. The survey showed that, compared with the basic wage-earning European group, less dairy produce, fruit, and vegetables but more carbohydrates were eaten, both groups having a high meat intake. Since then no further survey has been done, but it is the general belief that these dietary patterns remain qualitatively unaltered. By European standards the total quantity of food consumed would be considered generous.

Surveys of Rheumatic Disease in the Maori Population

Material and Methods.—Since 1956 we have conducted two independent surveys of the prevalence of rheumatic disease among the Maori. In these surveys the definition of "Maori" has followed current New Zealand usage to include all those people of at least half Maori blood who consider themselves to be members of the Maori community. Only a portion of such persons are of completely unmixed Polynesian ancestry. In 1871, the darkest period of the Maori race, there were approximately 37,520 Maori; in 1896, 42,113; in 1921, 55,000; and in 1936, 82,326. In 1956 the Maori composed 137,341 of the total New Zealand population of 2,174,062. These figures give some idea of the present rate of population increase.

First Survey (Rose, 1956).—This was conducted by one of us (B.S.R.), with the help of a medical social worker and in conjunction with officers of the

Maori Affairs Department, in a tribe of comparatively "pure" blood. At the time of a personal interview with a Maori Affairs Officer, a questionnaire was completed for all the adults of this tribe (the Whanau-a-apanui), a rural community living in the most easterly part of Auckland Province.

All those with a history or symptoms of rheumatism were examined for peripheral arthritis. Gout was suspected in those who gave a history of episodic acute arthritis, usually peripheral and asymmetrical, characterized by severe pain and swelling which subsided completely, and responding to colchicine when exhibited. Blood samples were obtained when possible in suspected cases of gout, and serum uric acid level and erythrocyte sedimentation rate were estimated, and a sheep cell agglutination test (S.C.A.T.) performed. It was not possible to have many x rays done. Diagnosis was made on the typical history and clinical findings, confirmed by a raised serum uric acid level. All serum uric acid levels were determined photometrically by Beale's modification of Folin's method (Beale, 1954). The cutting line for this method is at 6 mg. per cent. for women and 7 mg. per cent. for men. Hyperuricaemia was not regarded as sufficient evidence of gout in the absence of clinical history or of typical physical signs (Goldthwait, Butler, and Stillman, 1958).

Of 462 Maori adults in the tribe, 88 made complaint of rheumatism. The results of the examination are shown in Table I; 22 were found to be suffering from clinical gout, and a further three with hyperuricaemia were possibly gouty. No classical rheumatoid arthritis was seen, although there was a group of fourteen patients with a history of recurrent arthritis or arthralgia without diagnostic signs.

TABLE I
WHANAU-A-APANUI SURVEY, 1956

No. of Persons	Interviewed Examined						
		462
		88
Rheumatic Conditions	Classical clinical gout	22
	(a) confirmed by hyperuricaemia	20	
	(b) blood not examined	2	
	Possible gout with hyperuricaemia	3
	Definite rheumatoid arthritis	0
	Recurrent arthralgia (not further defined)	14
	Degenerative joint disease and mechanical derangement	20
	Extra-articular conditions	26
Nonrheumatic Disorders	3

In the 22 cases of clinical gout, the serum uric acid was determined in all but two, and all were found to be raised (see Table II).

TABLE II
SERUM URIC ACID LEVELS IN 22 CASES

Case No.	Sex	Age (yrs)	Serum Uric Acid	S.C.A.T.
1	M	33	11.5	.
2	M	36	11.10	-ve
3	M	68	10.45	.
4	M	44	10.25	.
5	M	67	10.05	.
6	M	37	9.5	-ve
7	M	51	9.4	.
8	M	65	9.2	-ve
9	M	66	9.2	.
10	M	52	9.05	-ve
11	M	42	8.6	-ve
12	M	50	8.45	.
13	M	79	8.3	.
14	M	38	7.95	.
15	M	65	7.85	-ve
16	M	58	7.2	-ve
17	M	48	.	.
18	M	41	.	.
19	F	60	10.3	.
20	F	52	9.3	.
21	F	53	7.4	.
22	F	56	7.35	.

Second Survey.—This was carried out in 1957-58 by the staff of the Queen Elizabeth Hospital and the School of Social Sciences of Victoria University, Wellington. A 1:12 random sample was taken of both the Maori and non-Maori adults of Rotorua, a country town of approximately 15,000 people. Most of the Maori in the area are members of the Arawa tribe, who through long friendly association with European settlers are a less pure and homogeneous section of the Maori people than the subjects of the 1956 survey. The survey was performed by personal interview, at which time 827 questionnaires were completed. 361 persons complaining of rheumatic symptoms were subsequently examined clinically, and in most cases the erythrocyte sedimentation rate was estimated, a sheep-cell agglutination test was carried out, and standard x rays were taken of the hands and feet. Serum uric acid determinations were done only in those cases in which the presence of gout was considered probable on clinical grounds. The relevant diagnostic findings are shown in Table III (opposite); the detailed methodology and findings of this survey will be reported later (Rose and Isdale, in preparation).

Criteria for the diagnosis of gout were the same as those in the preceding 1956 survey with more extensive use of x rays. Of 641 non-Maori subjects, only two sufferers from gout were found, while of the 186 Maori subjects, five were gouty, a significant difference ($\chi^2 = 9.699$; $p < 0.01$).

All seven patients with gout had a raised serum uric acid level (Table IV, opposite). On this occasion some definite rheumatoid arthritis was found among the Maori subjects, but much less than among the non-Maori (see Table III).

TABLE III
ROTORUA SURVEY, 1957-58

Race		Maori	Non-Maori	Total
No. of Persons	Interviewed	186	641	827
	Examined	66	295	361
Rheumatic Condition	Classical Clinical Gout	5	2	7
	Definite Rheumatoid Arthritis	2	43	45
	Probable Rheumatoid Arthritis	4	35	39
	Degenerative Joint Disease and Mechanical Derangement	—	—	77
	Miscellaneous, including possible Rheumatoid Arthritis and Rheumatic Fever	—	—	74
No Rheumatic Diagnosis	—	—	119	

TABLE IV
URIC ACID LEVELS IN SEVEN CASES
ROTORUA SURVEY, 1957-58

Case No.	Sex	Age (yrs)	Race	Uric Acid Level	S.C.A.T.
1	M	41	European	8.45	-ve
2	M	51	European	8.1	-ve
3	M	48	Maori	7.9	-ve
4	M	49	Maori	7.6, 11.4	-ve
5	M	40	Maori	8.5	-ve
6	M	37	Maori	8.7	-ve
7	M	23	Maori	9.3	-ve

While the prevalence of gout among the Whanau-a-apanui tribe of the East Coast was found to be higher than among the Arawa people of Rotorua, this difference is not significant ($\chi^2 = 1.428$; $0.50 > p > 0.30$). A high level of significance is achieved when the prevalence in the combined tribes is compared with the non-Maori population of Rotorua ($\chi^2 = 21.77$; $p < 0.001$). In each survey group, men were much more frequently found to be gouty (Table V), an expected observation (Hench, Bauer, Dawson, Hall, Holbrook, Key, and McEwen, 1940).

TABLE V
INCIDENCE OF GOUT IN TWO SURVEYS

Centre	Survey		Sex		Total
	Race	Date	Male	Female	
Whanau-a-apanui Tribe	Maori	1956	18 of 219 8.2 per cent.	4 of 243 1.6 per cent.	22 of 462 4.7 per cent.
	Arawa Tribe Maori	1957-58	5 of 83 6.0 per cent.	0 of 103 0 per cent.	5 of 186 2.7 per cent.
Rotorua	Non-Maori	1957-58	2 of 296 0.7 per cent.	0 of 345 0 per cent.	2 of 641 0.3 per cent.

Discussion

It is widely held that gout is rare among native races (Hench and others, 1940; Brugsch, 1957; Perlman, Bernstein, Maslow, and Scatliff, 1953). The historical evidence supporting this opinion in the case of the Maori of past generations has been briefly reviewed. That such a view is no longer true among the present Maori community has been clearly demonstrated by these surveys. Among another Pacific race, the Filipinos, a high and probably increasing incidence of gout has recently been reported (Decker and Lane, 1959). In the United States, Nathan, Kubota, and Turnbull (1953) reported an incidence of gout of 0.04 to 0.2 per cent. among Negroes admitted to the Cook County Hospital between 1944 and 1952, compared with an incidence of 0.06 to 0.1 per cent. among the white patients at the same hospital.

Among the patients with gout treated at this hospital, the Maori patients show certain minor clinical differences. Tophi are less common, and when present are never large. Impaired renal function is occasionally found, but little work on the renal pathology has been possible because of the characteristic difficulty in obtaining permission for

autopsy or biopsy from the Maori. Associated hyperpiesia is not infrequent. No estimations of total uric acid pool have yet been done. In their response to the treatment of both acute and chronic gout, Maori patients differ in no way from non-Maori subjects, except that, to be effective in long-term treatment, probenecid is needed in a dosage of 2 g. daily rather than the more usual 1-g. dosage used for non-Maori patients with gout. Not uncommonly there is a past history of gonorrhoea, and in cases showing a positive gonococcal complement-fixation test, treatment with parenteral penicillin combined with pyretic baths has value as an adjunct to the usual gout treatment.

Historical evidence quoted above makes it probable that gout was uncommon, if seen at all, among the Maori in the days before their discovery by Europeans and until the present century. There seems little doubt that there has been a genuine and marked increase of gout in the Maori, and while it is difficult to be certain when this increase occurred, it has been most marked in the last 20 to 30 years.

The incidence of clinical gout is at least partially genetically determined, and in Caucasian races a reservoir of potentially gouty subjects exists. Experience in Germany (Zöllner, 1957) during and after World War II shows how the amount of clinically obvious gout may fluctuate with the general state of nutrition of the population. Acute classical gout, notoriously prevalent among the nobility and gentry in the Regency period, is steadily diminishing in England (Thompson, 1938), but is being recognized more frequently in the United States (Hench and others, 1940); changing economic status may be a factor in this transatlantic shift. The results of the surveys of the Maori reported above show that the genetic defect preceding clinical gout is present in the race, as much in the East Coast tribe with minimal racial admixture as in the less racially pure Arawa people. This strongly suggests that, though clinical gout was not earlier recognized, the Maori has always been potentially gouty. It is of interest in this context that gout is not seen among the Polynesians living in the New Zealand dependencies in the Pacific (McCarthy, 1956). These island peoples, whose anthropologic and blood group characteristics (Simmons, Graydon, Semple, and Taylor, 1951) closely resemble those of the Maori, still live to-day in a culture not unlike that of the pre-European Maori, with a simple diet consisting largely of fish and root vegetables. It will be of great interest to see whether the incidence of gout rises in these people with any future improvement of economic status.

Failure by early observers in New Zealand to

describe gout may have been due in part to under-diagnosis, though the background, training, and worth of those observers and the very magnitude of the incidence among the Maori to-day, makes it certain that there has been a very real increase in frequency. In the absence of a significant genetic change, other factors must be sought to explain this, and of these, the marked change of diet and cooking method is probably the most important. This increase of prevalence has appeared at a time of markedly increased material prosperity and life expectancy: to-day the Maori who earns well spends freely, particularly on rich foods, beer, and other creature comforts.

Summary

Population surveys show a much greater prevalence of gout among the Maori people of New Zealand than among people of European stock. Historical evidence strongly suggests a real and recent increase in incidence. It is suggested that the marked economic and dietary changes in the Maori cultural environment in the last two hundred years could sufficiently explain this without postulating any alteration in the necessary genetic constitution.

We wish to acknowledge the most courteous and generous reception to our survey teams by the Maori people, and the help given by our own social workers, Mrs. Welsh and Miss Newcombe, and the staffs of the Department of Maori Affairs and the School of Social Sciences of Victoria University, Wellington.

We are grateful to Dr. J. Cairney, Director General of the New Zealand Department of Health for permission to publish this paper.

REFERENCES

- Banks, Sir J. (1958). "Sir Joseph Banks in New Zealand from his Journal", ed. W. P. Morrell. Reed, Wellington.
- Beale, R. N. (1954). Paper read to New Zealand Rheumatism Association.
- Best, E. (1929). "Fishing Methods and Devices of the Maori." Dominion Mus. Bull. No. 12. Wellington, New Zealand.
- (1942). "Forest Lore of the Maori." Dominion Mus. Bull. No. 14. Wellington, New Zealand.
- Blair, A. T. M. (1951). *N.Z. med. J.*, 50, 85.
- Brugsch, H. G. (1957). "Rheumatic Diseases, Rheumatism, and Arthritis", p. 222. Lippincott, Philadelphia.
- Buck, Sir P. (1949). "The Coming of the Maori", p. 64. Whitcombe and Tombs, Wellington.
- Cook, J. (1951). "Captain Cook in New Zealand: Extracts from the Journals of Captain James Cook", ed. A. H. and A. W. Reed. Reed, Wellington.
- Decker, J. L., and Lane, J. J. (1959). *New Engl. J. Med.*, 261, 805.
- Fairfowl, Doctor (1908). In "Historical Records of New Zealand", ed. R. McNab, vol. 1, pp. 550-8. Government Printer, Wellington.
- Goldthwait, J. C., Butler, C. F., and Stillman, J. S. (1958). *New Engl. J. Med.*, 259, 23, 1095.
- Hench, P. S., Bauer, W., Dawson, M. H., Hall, F., Holbrook, W. P., Key, J. A., and McEwen, C. (1940). "Sixth Rheumatism Review", in *Ann. intern. Med.*, 13, 1837.
- Herbert, A. S. (1921). "The Hot Springs of New Zealand." Lewis, London.
- Hunter, D. (1956). In "Price's Textbook of the Practice of Medicine", 9th ed., ed. D. Hunter, p. 444. Oxford University Press, London.
- McCarthy, D. D. (1956). Personal communication.
- McLaughlin, E. C. G., and Wilson, I. (1945). *N.Z. med. J.*, 44, 93.

- Nathan, L. A., Kubota, C. K., and Turnbull, G. C. (1953). *J. Lab. clin. Med.*, 42, 927.
- Newman, A. K. (1880). "Transactions and Proceedings of the N.Z. Institute, 1879", vol. 12, p. 433.
- Perlman, L., Bernstein, A., Maslow, W. C., and Scatliff, J. H. (1953). *J. Amer. med. Ass.*, 151, 726.
- Rose, B. S. (1955). *Lancet*, 1, 1274.
- (1956). Paper read to N.Z. Rheumatism Association.
- and Isdale, I. C. In preparation.
- Savage, J. (1807). "Some Account of New Zealand", quoted by Turbott (1940), p. 257. Murray, London.
- Simmons, R. T., Graydon, J. J., Semple, N. M., and Taylor, C. N. D. (1951). *Med. J. Aust.*, 1, 425.
- Te Rangi Hiroa, see Buck, Sir P.
- Thompson, F. G. (1938). In "A Survey of Chronic Rheumatic Diseases", ed. R. G. Gordon and others, pp. 191-200. Oxford University Press, London.
- Turbott, H. B. (1940). In "The Maori People Today", ed. I. L. G. Sutherland, pp. 229-68. Whitcombe and Tombs, Wellington.
- Wohlmann, A. S. (1914). "The Mineral Waters and Spas of New Zealand." Government Printer, Wellington.
- Zöllner, N. (1957). *Germ. med. Monthly*, 2, 253.

La goutte chez les Maoris

RÉSUMÉ

Des enquêtes effectuées en Nouvelle Zélande ont révélé une plus grande fréquence de la goutte chez les

Maoris que chez les personnes d'origine européenne. Des données historiques indiquent fortement que cette fréquence augmentée est réelle et récente. On pense que des changements alimentaires et économiques marqués dans l'ambiance culturelle des Maoris au cours de derniers deux cents ans expliquent ce fait suffisamment, sans qu'il soit nécessaire de postuler une altération de la constitution génétique.

La gota en los Maoríes

SUMARIO

Censos en la Nueva Zelandia revelaron una mayor frecuencia de la gota en los Maoríes que en las personas de origen europeo. Datos históricos sugieren fuertemente que este aumento es auténtico y reciente. Se cree que cambios alimentarios y económicos en el ambiente cultural de los Maoríes en los últimos doscientos años bastan para explicar este hecho, sin necesidad de postular una alteración de la constitución genética.