

From decline to recovery: the Marquesan population 1886–1945



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Abstract

Population decline in the South Pacific is poorly documented. Civil-registration data from the Marquesas (French Polynesia) from 1882 to 1945 are here used to calculate most of the usual demographic indices. Deterioration of natural equilibria following the arrival of Europeans in the islands and introduction of new diseases in a non-immune population caused a steady decline in the population. Beside catastrophic epidemics such as smallpox, mortality was high mainly because of introduced respiratory diseases. The very high mortality, ranging from 45 per 1000 to 70 per 1000 at the end of the nineteenth century and the beginning of the twentieth century, combined with low fertility due to the high proportion of sterile women (infected by venereal diseases), caused an annual decline of two to three per cent up to 1924. The arrival of a physician, introduction of basic sanitation and care of venereal diseases in Tahiti, whence most of the visitors to the Marquesas came, were followed by a sudden drop in mortality, rising fertility, and population increase. The depopulation phase lasted until the 1920s although the population generally stabilized elsewhere in Polynesia around the turn of the century.

Population decline in the Pacific has often been quoted but rarely demographically studied because the available data are poor, comprising unreliable estimates of the first navigators at the turn of the eighteenth century and incomplete censuses at the end of the nineteenth and beginning of the twentieth centuries.

The *Etablissements Français d'Océanie* instituted civil registration in 1852 in Tahiti and in 1882 in the Marquesas Islands. Depopulation had ended in Tahiti when civil registration became sufficiently complete to be used for statistics. In the Marquesas civil registration was fairly complete from 1886, except for infant deaths (see Appendix), and population decline lasted until 1924. Thus, it is possible to study population decline in the Marquesas for a period of forty years, during which the population steeply decreased from about 4900 to 2250 persons according to census data (see Rallu 1990).

Cultural and historical context

The Marquesan archipelago is situated 1500 km northeast of Tahiti and consists of six inhabited islands, Nuku Hiva, Ua Pou, Ua Uka (Northern group), Hiva Oa, Tahuata and Fatu Hiva (Southern group).

The Marquesas were discovered in 1595 by Mendana. Cook visited Hiva Oa and Tahuata in 1774 and several navigators and whalers visited the archipelago in the first half of the nineteenth century. In 1842, Du Petit Thouars, on his way to Tahiti, took possession of the Marquesas for the French. The French presence in the Marquesas was not significant: only one administrator and some soldiers were present on Nuku Hiva and Tahuata. Most often in the 1860s and 1870s no administrative nor military staff was present on the islands, and missionaries were the only French 'officials'. Hiva Oa was not pacified until the mid-1880s. Nevertheless, several censuses of the population were held from 1882

showing a rapid decline of the population. Similar declines were observed from the 1860s and the smallpox epidemic of 1863, and even from 1842 according to estimates of Du Petit Thouars and Jouan.

Dr Lesson, the first physician to visit the Marquesas, arrived in 1844. Although some of the administrators were also physicians (for example Dr Clavel and Dr Marestang) the health situation of the archipelago was very poor. Two physicians should have been located in the Marquesas, one in the Northern group and one in the Southern group, but most often only one was present in the administrative centre of Taïo Hae (Nuku Hiva), and sometimes none. This was in strong contrast with the situation in Tahiti where a colonial hospital was built at the end of the nineteenth century and where there were several military and civil physicians, as well as subordinate medical staff. They were in charge of Tahiti, Moorea and the Leeward Islands which also benefited from better communications than the Marquesas.

The reports of the physicians in the nineteenth century are interesting but provide poor information from the point of view of modern medicine. The health situation of the Marquesas did not improve much until the 1920s. Then in 1923 a physician, Rollin, was posted to the Southern group. He mainly worked in Hiva Oa and Tahuata, more rarely in the distant Fatu Hiva; in 1933 he was located in Nuku Hiva and again no medical staff were present in the Southern group. Rollin (1929) wrote a book on the Marquesan culture using early sources but said nothing of his work as a medical practitioner and even less than his predecessors about the health condition of the population. Valenziani, an Italian demographer, visited the Marquesas in 1938 and made an interesting study for the time, reporting both his own and Rollin's comments on the health situation (Valenziani 1940).

On the whole, our knowledge of health in the Marquesas before 1945 is very poor. We are forced to rely on sometimes contradictory physicians' reports to understand why mortality was so high.

Data

Censuses

Censuses before 1945 provide only the total number of the population, sometimes separately for males and females and more rarely for children under 14 years. Published data on the age structure were not available before the 1946 Census. The nominative list of the 1892 Census enabled us to tabulate the age structure. The results are not very different from those obtained from the population file constituted from civil registration (Rallu 1990:55–59). The total population enumerated at censuses between 1892 and 1946 shows a declining trend.

Civil registration

In this paper, the historical demography of the Marquesas is studied using family and genealogical reconstitution from civil registration. The genealogies have been used to constitute a population file. Individual records include dates of birth, marriage and death, places of birth and death as well as filiation. As civil registration provides no information on external migration, all the persons born in the Marquesas are accounted for in the population until their death or 1945. Before 1930 or even 1935 emigration from the Marquesas was very rare (see Appendix); only the immigrants (non-Marquesan born) who arrived before 1886 are taken into account.

Births and infant deaths were corrected to take account of children who died before registration (see Appendix). The reconstitution of families shows that the registration of births and deaths of children who survived a few months, and of adult deaths, was almost complete. The correction of births and infant deaths which has been made is probably a maximum, which makes the low fertility rates shown below still more impressive.

The population reconstituted from the genealogies is always close to the population enumerated at the censuses and shows a very similar evolution. The former consistently represents about 95 per cent

of the latter when short-term migrants are not taken into account. The difference between enumerated and reconstituted populations is due to migrations (see above) and to short-term immigrants who do not appear in civil registration, the number of the latter increasing from 1920. As population trends shown by censuses are consistent with those of the reconstituted population which does not take migration into account, population decline appears to be mainly due to negative natural growth, a conclusion already made by Valenziani (1940) for the period 1914–1937.

Population trends

In the Marquesas, deaths exceeded births from 1886 to 1923 (Figure 1 and Table 1), by a factor of two. From 1886 to 1900 the crude death rate was high, over 40 per 1000¹, and the crude birth rate was very low, around 25 per 1000.² Mortality rose to over 50 per 1000 after the beginning of the twentieth century and became extremely high between 1911 and 1925, reaching almost 70 per 1000. A very dramatic epidemic hit the Southern group and in 1914, the death rate reached 156 per 1000 in Hiva Oa. Nevertheless, this epidemic was a very light charge on the population in comparison with continuous high mortality, and the epidemic deaths represented barely two per cent of deaths in the 1886–1923 period. Such levels of mortality were never recorded for periods as long as 15 years. During the mortality crisis (1910–1923), births slightly increased. In fact, mortality increased mainly in the Southern group of the Marquesas, while fertility rose in the Northern group, where mortality only slightly increased. In 1924, the number of deaths suddenly halved, probably as a result of the arrival of Dr Rollin in 1923. Between 1926 and 1930 about equal numbers of deaths and births were recorded, but mortality remained high at 44 per 1000. Mortality regularly decreased after 1931 and the birth rate continued to rise.

¹ After correction for unregistered deaths.

² After correction for unregistered births.

Figure 1
Vital rates

Table 1
Vital rates

Years	Mid-year population	CBR		CDR		Natural increase %
		Observed	Corrected	Observed	Corrected	
1886-1890	4,652	19.6	25.6	41.5	47.5	-2.2
1891-1895	4,188	17.6	23.4	37.2	42.9	-2.0
1896-1900	3,814	18.9	27.6	35.8	44.5	-1.7
1901-1905	3,440	21.5	27.4	45.3	51.2	-2.4
1906-1910	3,123	30.1	41.3	43.4	54.6	-1.3
1911-1915	2,797	28.8	38.8	59.3	69.4	-3.1
1916-1920	2,400	28.7	37.8	57.6	66.8	-2.9
1921-1925	2,111	32.0	43.7	52.0	63.7	-2.0
1926-1930	1,981	32.6	41.6	35.1	44.1	-0.3
1931-1935	2,032	42.7	49.3	27.4	34.0	+1.5
1936-1940	2,211	41.9	45.3	21.2	24.6	+2.1
1941-1945	2,483	44.6	48.1	16.6	20.1	+2.8

The levels of fertility and mortality resulted in annual negative growth rates of about two per cent until 1910, reaching three per cent in 1911-1920 (Table 1). Natural growth was still slightly negative in 1926-1930 but increased regularly thereafter.

The results for the period 1916-1935 are consistent with those obtained by Valenziani (1940) for the years 1914-1937 using annual total numbers of births and deaths (including immigrants) related to the total number of the population enumerated at censuses.

Mortality

Extremely high mortality is the most striking feature of Marquesan demography until 1924.

That age-specific mortality rates were very high is evident from the beginning of civil registration (Figures 2a, b and c).³ Adults, especially young adults, were hit by abnormally high death rates, and thus mortality only slightly increases, with age. Adult death rates were higher than the highest level of the Princeton model life tables and it is difficult to believe that such levels of mortality could prevail for decades. Moreover, death rates increased again from 1910. In the 1910–1923 period, age-specific death rates of adults were twice as high as the highest level of model life tables. It is difficult for a demographer to comment, having no other data for comparison, except during short epidemics or wars.

Female death rates were higher than those of males by 10 or 15 per cent, mainly at child-bearing ages, until 1935. Nevertheless, excess female mortality existed before ten years of age. In the 1936–1945 period excess female mortality became limited to the 10-29 years age group.⁴

Figure 2a

³ Age-specific death rates are considered from five years as underregistration of infant deaths and inaccurate date of births (due to the delay between birth and registration) prevent the analysis of death rates of younger children. Registered infant mortality rates fluctuated around 220 per 1000 for males and 170 per 1000 for females before 1924. Infant mortality decreased after 1924.

⁴ The Maori of New Zealand also experienced excess female mortality (see Pool 1991).

Figure 2b

Life expectancy at birth (Table 2) was just over 21 years in 1886-1905, and dropped to 16 or 17 years during the long mortality crisis of the 1910s and early 1920s. Life expectancy at ten years (which is not dependent on estimates of unregistered infant deaths) is lower than that of Level 1 of the West life table models, owing to the level of age-specific death rates at adult ages. Female life expectancy is systematically lower than that of males until 1935, often with a gap of two or three years. Such low levels of life expectancy over periods of 15 to 20 years reveal very poor health. Very few populations have experienced such high mortality. For example, life expectancy at birth was 24 years for males and 26 years for females in France around 1740-1749, owing to several food shortage crises, and was as low as 22-23 years in India at the beginning of the twentieth century during an epidemic of plague.

Table 2
Expectation of life at birth and age ten, by sex, various years

Years	At birth		At ten years	
	Males	Females	Males	Females
1886-1895	20.8	21.3	29.4	26.7
1896-1905	21.8	22.6	29.8	29.2
1906-1915	17.1	19.3	24.2	24.8
1916-1925	16.5	16.5	23.4	20.3
1926-1935	27.4	26.8	32.8	30.6
1936-1945	40.3	43.3	44.2	44.5

Figure 2c

Life expectancy began to increase rapidly after 1925 for both sexes, and the level reached in 1936-1945 was close to that of French Polynesia at that time. The level of mortality varied greatly between the islands. The mortality crisis of the years 1911 to 1925 was mainly located in the Southern group of the Marquesas before the effect of the first physician there in 1923. In Hiva Oa, some valleys like Hanaiapa experienced higher mortality. In 1916-1925, the observed crude death rate was 74 per 1000 in the Southern group as against 38 in the Northern group; it reached 83 in Hiva Oa and 102 in Hanaiapa in the absence of any epidemic. The annual population decrease was 5.1 per cent in the Southern group, 5.9 per cent in Hiva Oa and 8.3 per cent in Hanaiapa. Life expectancy at birth was well under 15 years in Hiva Oa in 1910-1925.

Causes of death were not registered but possible insights from other sources into the reasons for such high mortality are presented below.

High adult mortality for both sexes and excess female mortality show that violence and warfare were not a frequent cause of death since these would have caused excess male mortality. Only about one-half of excess female mortality was due to maternal deaths (death in the 60 days following a birth). The other half was due to other causes, such as that women take care of sick people and are more affected by infectious diseases. Death rates at 5–9 years were not very high and much lower than adult rates. Such high mortality was the result of imported diseases affecting adults in a population that lacked immunity against the new diseases.

Sanitation was very poor and medicine non-existent. In the absence of medicine, tuberculosis often had a fatal outcome after eight to twelve months. Alcohol also reduced resistance. Moreover, islanders were given bad alcohol made by the sailors, which was sometimes the cause of epidemics (Lesson 1981). Tuberculosis, other respiratory diseases and dysentery were the causes of death reported most often by physicians in the Marquesas, such as Marestang (1892) and Tautain (1898). They, as well as Rollin and Valenziani, also emphasized the importance of young adult deaths from respiratory diseases.⁵ Valenziani notes that a civil registration officer in Atiheu (Nuku Hiva) reported that half the adult deaths in 1918–1920 were attributable to ‘phthisis’. As the officer was not a physician this information is not very reliable, and ‘phthisis’ may include other respiratory diseases such as bronchopneumonia, as acknowledged by Valenziani. Also according to Valenziani, probably following Dr Rollin’s statements, young children’s deaths were often due to intestinal diseases as a consequence of bad food (fermented *‘popoi’*). Nevertheless, death rates at one to four years were not as grossly elevated as at young adult ages.

The steep and sudden drop in mortality is more difficult to explain. Rollin, the first physician in the Southern group, arrived in 1923. He could have used collapse therapy (pneumothorax) to reduce rapidly deaths due to consumption. BCG was used in the French colonies, but Rollin did not say in his book whether he used it. His role in the rapid decrease in the death rates is obvious: in 1924 the number of deaths in every valley of Hiva Oa and in the nearby island of Tahuata dropped by 50 per cent or more compared to 1921–1923; the drop occurred a few years later in the distant Fatu Hiva. The steep decline in deaths occurred at all ages except older ones. Changes in very basic sanitation could also have helped cause a rapid drop in mortality. A sudden underregistration of deaths in 1924 and the following years is unlikely and there is no evidence of it in the reconstituted families.

Nuptiality

Most marriages were consensual unions and thus around 80 per cent of fertile couples experienced extramarital births or premarital conceptions. Practically all adult women were married or were in a consensual union, the latter being known through the registration of extramarital births where the woman’s partner declared he was the child’s father. Thus fertility was not affected by high levels of celibacy although broken unions through widowhood or unstable unions affected the risk of conception.

Fertility

Low fertility was mainly the result of low fecundity. An ageing population structure due to population decline also had a slight deflating effect on the birth rate.

⁵ These were also frequent causes of deaths reported for the Maori of New Zealand (cf. Pool 1991).

The total fertility ratio (TFR)⁶ which was between 3.0 and 3.5 was very low in the 1886–1905 period. Then the TFR increased by steps: 4.9 in 1906–1910, 6.0 in 1921–1925 and 7.2 in 1941–1945. The shape of the curve of age-specific fertility rates in 1886–1895 was very flat. Rates were exceptionally low at young ages but almost normal at 35–39 years. In the following decades until 1920, age-specific fertility rates progressively rose before 30 years, but it is only from 1930 or 1935 that the fertility of the 20–29 age group reached the usual levels expected of a pre-transitional society.

Cohorts born before 1871 had very low fertility at ages 20–24, 25–29 and 30–34 (Figure 3); the 1876–1886 birth cohorts had slightly higher fertility at ages 20–24 or 25–29. The shape of the fertility curve by age is more usual for the birth cohorts of 1891–1906, but still low before 30 years, and women born from 1911 onwards have normal fertility. It is clear that the increases in the period TFR by steps is due to the arrival of these different groups of birth cohorts into reproductive ages.

Figure 3

⁶ Corrected for underregistration of births; corrected births were distributed by age of mother and by birth order according to registered births.

Table 3
Parity progression ratios (per cent) of birth cohorts

Birth cohorts	P ₀		P ₁	P ₂	P ₃	P ₄	P ₅	P ₆	P ₇	P ₈	Completed fertility (fertile women)
	observed	corrected									
1871-1875	40	54	79	82	72	88	93	86	89	83	4.74
1876-1880	53	72	80	87	95	95	92	76	82	84	6.08
1881-1885	57	77	76	89	86	85	93	79	96	50	5.18
1886-1890	60	80	85	87	87	77	77	92	98	92	6.14
1891-1895	68	89	87	84	96	84	86	77	99	74	6.24
1896-1900	67	85	94	82	87	69	85	83	91	88	5.84
1901-1905	64	78	88	79	82	80	84	99	97	92	6.07
1906-1910	69	81	85	90	91	91	(85)	(87)	(86)	0	
Crulai ¹			100	100	83	89	82	93	69	67	6.51
Tourouvre ²			100	97	100	92	91	84	80	82	7.70

Sources: ¹Henry 1958: Table 126; ²Charbonneau 1970:137, Table 2.

Figure 4

The analysis of fertility by birth order explains why earlier birth cohorts had so few babies. The size of the population under study is too small to analyse age-specific fertility rates by birth order, so we shall consider TFR by birth order and parity-progression ratios (Figure 4 and Table 3). First births are very few in cohorts born before 1880: between 28 and 46 per cent of the women in these birth cohorts had no child. Primary sterility was still around 20 per cent for women born in 1881-1890 and 13 per cent for women born in 1891-1900 and 1911-1920. Parity progression ratios p_1 , p_2 and p_3 are still low compared to other pre-transitional fertility levels. It is also unusual that parity-progression ratios increase with birth order up to p_3 or p_4 as seen in older birth cohorts. The p_5 and p_6 ratios are closer to pre-transitional fertility standards and p_7 and higher ratios are higher than in eighteenth-century France. The completed fertility of fertile women is 4.7 for the 1871-1875 cohort and exceeds 6.0 for the cohorts 1876-1905 (except for the 1876-1880 cohort).

Table 4
Completed fertility of various subpopulations according to birth cohort

Birth cohorts	All women	Fertile women	Currently married fertile women	(2)/(1)	(3)/(2)
1871–1875	3.19	4.74	5.93	0.67	0.80
1876–1880	4.09	6.08	8.55	0.67	0.71
1881–1885	4.22	5.18	7.40	0.81	0.70
1886–1890	4.85	6.14	9.80	0.79	0.63
1891–1895	5.72	6.24	8.10	0.92	0.77
1896–1900	5.54	5.84	7.73	0.95	0.76
1901–1905	5.26	6.07	7.92	0.87	0.77

The difference between the TFR and the completed fertility of fertile women represents the reduction of fertility due to primary sterility. Thus primary sterility reduces by one-third the completed fertility of women born in 1871-1880 (Table 4) and by 20 per cent that of women born in 1881-1890. The effect of primary sterility in later birth cohorts is much less.

Age-specific fertility rates of fertile currently 'married' (cohabiting) women, as shown in Figure 5 and Table 4, are quite close to pre-transition fertility standards for women born from 1876 and are very high even for younger generations (1906–1915). Completed fertility ranges between 7.4 and 8.6 in the birth cohorts of 1876–1905. The difference between this and the previous index represents the reduction of fertility due to unions that were broken by widowhood or separation.

It was difficult to assess if some infertile women were in a union or not. I considered them in a union if their last partner was still alive and did not appear in a subsequent union, but they could have experienced a broken union followed by no other union or an infertile consensual union. In the former case, fertility rates would be underestimated. On the other hand, some women whose last partner died or entered a new union could have experienced a subsequent infertile consensual union. Thus it is possible that I selected fertile women and this bias would probably be more important than the former one. As a result the difference between the fertility indices may include fertility reduction due to secondary infertility. The effect on fertility of broken unions and secondary infertility increases from the birth cohorts of 1871–1875 to those of 1886-1890; the latter were 20–24 years old in 1910 when the mortality crisis began and 35–39 years when it ended in 1924.

Comparison of three fertility indices (Table 4) shows that primary infertility was the main cause of low fertility in older birth cohorts (1871–1880). From the birth cohorts of 1881–1885; broken unions and secondary infertility were the most important cause of fertility reduction. Owing to secondary sterility and broken unions, the mean age at last birth was 32 years in generations born before 1900, compared to about 39 or 40 years in Europe before the beginning of the fertility transition. Thus the flat fertility curve before 30 or 35 years in older birth cohorts is due to abnormally high proportions of primarily and secondarily sterile women. Women who were not affected by diseases and broken unions had the usual pre-transitional fertility.

Figure 5

Contemporary observations are consistent with these results. For example, around 1880 Clavel surveyed 47 women over childbearing age and found that 20 of them (or 43 per cent) had not had a child, while the other 27 had borne 199 children (or 7.4 per woman). In 1890 Marestang surveyed 539 women aged 15 and older and found that 48 per cent were sterile.

Birth intervals were not corrected for underregistration of births, which probably causes the slightly longer intervals in older birth cohorts for whom underregistration was more important than for younger birth cohorts.⁷ Nevertheless, in most generations and especially those born from 1890 onwards, birth intervals are short compared to those in pre-industrial Europe (24 to 26 months in eighteenth-century France). This indicates that breastfeeding was very short, perhaps only three to six months. The comparison of the birth interval after a deceased child with that after a surviving child leads to the same conclusion.

Birth intervals between children of different fathers were about twice as long as between true siblings. This accounts for a reduction of about one birth per broken union when it is followed by a new union.

Venereal diseases were widespread in Polynesia at that time. Gonorrhoea would in general have been much more prevalent than syphilis. Gonorrhoea causes lesions that finally result in sterility, while syphilis causes abortions, stillbirths or premature births of infants who could not have been kept alive in the sanitary conditions of that time. Thus venereal diseases were the main cause of primary sterility and low fertility in older birth cohorts. Many women became secondarily sterile after only one or two births. Few women had more than eight or ten births. Overall health conditions also played a role in low fertility. It is well known that fevers due to any sickness reduce the fecundity of both males and females for the time of the sickness.

At the end of the nineteenth century, the colonial hospital in Tahiti began to treat venereal diseases and thus women born after 1886 were not so often affected. In addition, European sailors and traders coming to the Marquesas, who almost all came from or through Tahiti, were less frequently infected and did not spread the sickness so much. Young girls attended the missionary boarding schools, so they were not exposed to sexual intercourse and to venereal diseases until later than their elders had been, and a higher proportion of women bore one or two children before they became infected and sterile. Catholic missionaries wrote that Marquesan sexual behaviour (early intercourse with different partners) was the cause of low fertility.

Widowhood and separations or divorces also caused low fertility. However, these reduced fertility less than did primary and secondary sterility in older birth cohorts.

Population development and age structure

The 1886 population structure (Figure 6) shows an ageing population. There were fewer children aged 0–4 years than 5–9 years. Generally, numbers of young persons (5–19) are close to those of adults which are fairly stable from 20–24 to 35–39 years, probably owing to a long period of population decline. Children younger than 15 years make up only 27 per cent of the population while persons over 60 years comprise five per cent. The small birth cohorts between 1862 and 1866 (20–24 years in 1886), mainly evident for women⁸, are due to the catastrophic epidemic of smallpox in 1863 which the missionaries believed claimed one-third of the population in the northern group. Other epidemics occurred in 1844 (Lesson 19?), but little is known about them, especially before 1850 when few Europeans stayed in the islands and wrote reports.

⁷ If most unregistered births are due to stillbirths and linked with venereal diseases and subsequent sterility, birth intervals should not be too much affected by underregistration which would mainly affect the last births of women.

⁸ Age misreporting is very probable at the beginning of civil registration. Ages of father and mother were reported at the birth of each of their children; the most consistent age was included in the population file, which is often the age reported at births of first or second children, when the person was youngest. Nevertheless, the age of some women had to be corrected so that age at last birth should not be too high. A similar correction was impossible for males.

The stable population consistent with the 1886–1895 life table and an annual negative growth of two per cent has proportions of youthful population (28.5%) and adults aged 15 to 59 (63%) close to those observed in 1886. The sex ratio is 1.06 in the stable population against 1.04 observed in 1886, because of female excess mortality. The crude birth rate of the stable population, 13.9 per 1000, is close to the observed level in 1886–1890, taking into account a higher share of adults in the real population.

Thus, the population structure in 1886 implies a mean annual decrease of two per cent for the 50 to 55 years up to 1886. Backwards projection of the population using this rate, and taking into account the big epidemic in 1863, leads to a population of around 17,500 in 1848, which suggests that the estimate of Du Petit Thouars (20,200) appears to be too high. Famine, warfare, epidemics and new diseases had hit the population before 1848. Possibly 40,000 to 45,000 people lived in the Marquesas at the beginning of the nineteenth century but this is much lower than Forster's guess of 50,000 persons in two islands (Hiva Oa and Tahuata) at the end of the eighteenth century.

After 1886 extremely high mortality and low fertility had dramatic effects on the size and the age structure of the population. The Northern group was not affected by a mortality crisis in the 1910s and early 1920s and there fertility rose from 1900; the Southern group, mainly affected by poor sanitation and extremely high mortality, experienced fertility recovery only from 1930. The population of the Northern Marquesas was 40 per cent smaller in 1926 than in 1886 (Table 5). The population of the Southern Marquesas in the early 1930s was only a quarter of that in 1886. During the 20 years of recovery from 1926 to 1946, the population increased by 53 per cent in the Northern group but only by 17 per cent from 1930 to 1946 in the Southern group where mortality remained higher and fertility rose later.

Figure 6

Table 5
Population of the islands of the Marquesas

Year	Nuku Hiva	Ua Pou	Ua Uka	Hiva Oa	Tahuata	Fatu Hiva
1886	726	329	130	2504	348	742
1896	538	274	91	1963	291	612
1906	478	247	80	1510	216	470
1916	441	253	79	976	189	330
1926	428	228	73	543	147	247
1936	462	310	75	489	139	214
1946	540	468	106	546	160	285

Population decline caused an important ageing of the population due mainly to reduced fertility. In 1906 the proportion of children younger than 15 years was only 23 per cent in the Southern group while the proportion of persons aged 60 years and older was nine per cent. High mortality of adults caused a decline in the share of the 15–59 age group, making children under 15 years 34 per cent of the total population in 1926 in the Southern Marquesas, and 44 per cent in 1946 (48 per cent in the Northern Marquesas).

Conclusions

The Marquesas may be the worst, or one of the worst, cases of depopulation in the Pacific. Marquesan mortality and fertility levels and trends from 1886 to 1924 show the mechanisms of a rapid and continuous population decline with negative annual rates of natural increase of two to three per cent. This situation was due to very poor public health and to the breaking of natural centuries-old equilibria by the introduction of new diseases into a population lacking immunity and medical protection against them. Mortality and sterility were extremely high. Life expectancy at birth was well under 20 years from 1910 to 1923. Women born between 1871 and 1875 bore around three children on average and women born in 1876 to 1885 bore fewer than four children.

Following the arrival of a physician, the introduction of medicine with which to treat tuberculosis, and the development of basic sanitation, suddenly lowered death rates in 1924. Fertility increased progressively from the 1910s in the Northern Marquesas and from the 1930s in the Southern Marquesas, new generations being affected by venereal diseases at later ages and less frequently than their elders.

Appendix⁹

Quality of data

Incomplete registration affects infant deaths and births. Infant death rates calculated from registered deaths are obviously too low, ranging from 242 per 1000 to 114 per 1000 before 1930 and being most often under 200 per 1000. On the other hand, the fates of fewer than four per cent of persons are unknown. I conclude that both births and deaths of young babies were not recorded. If birth had been recorded but not death there would be many persons of unknown fate. Comparison of declared dates of birth at registration and baptism, and adjustment of infant deaths using the method of Bourgeois-Pichat, show that non-registration of birth and death of a child was due to a delay ranging from several days to

⁹ This summarizes Chapter 1 of Rallu (1990).

two or three months between birth and registration. If the child died before registration neither birth nor death was reported. Moreover, many stillbirths and premature births rapidly followed by death would have occurred because of syphilis, and might represent most of the underregistration of births and deaths.

Very few Marquesan people whose birth was not reported were recorded as marrying or dying in subsequent years (less than one per cent of all births) which indicates that the registration of births was almost complete so long as the child lived for a few weeks or months.

Tahitian census data and civil registration show that emigration from the Marquesas was very low until the 1930 . Very few Marquesan-born persons were enumerated in Tahiti and very few deaths or births occurred to persons born in the Marquesas and resident in Tahiti.

Correction of data

Most methods for correcting data for underregistration of infant deaths rely on the level of death rates at adult ages. Because of extremely high adult mortality before 1924, adjustment of infant death rates on adult ones leads to rates between 600 and 800 per 1000 which are obviously too high. Consequently I adjusted the infant death rate on that of level 1 of the West model life tables. After 1924 I adjusted infant death rates on levels 1 to 10 progressively by five-year periods, according to the course of adult mortality. The same number added to infant deaths was added to births. As adult mortality was exceptionally high from specific causes of death, the correction of infant deaths (using West level 1), and births, is a maximum, which also appears from the crude birth rate implied by the stable population.

The appendix table shows that the correction of births has no major influence on fertility trends. Fertility rises when the proportion of corrected births decreases. Uncorrected fertility is extremely low in early birth cohorts. As a result of syphilis most unregistered births would have been stillbirths, which raises the question of the measurement of fertility when many pregnancies do not result in live births. Should one consider pregnancies or live births when comparing the fertility of a population largely affected by syphilis to natural fertility? On the other hand, the probable predominance of gonorrhoea (on the assumption of a similar situation in the Marquesas as in Tahiti) and associated sterility is consistent with the low fertility observed after correction of births in former generations.

Registration of deaths, except for infants, is fairly complete. Estimates are also given of life expectancy at birth derived from death rates at ages ten and over which do not rely on correction of infant deaths.

Annexe table.
Corrected fertility and correction of births

Birth cohorts	Completed fertility	Correction	Period	TFR	Correction
1871–1875	3.19	1.36	1886–1890	3.25	1.30
1876–1880	4.09	1.36	1891–1895	3.01	1.33
1881–1885	4.22	1.35	1896–1900	3.43	1.46
1886–1890	4.85	1.34	1901–1905	3.22	1.28
1891–1895	5.72	1.31	1911–1915	4.87	1.37
1896–1900	5.54	1.27	1911–1915	4.89	1.35
1901–1905	5.26	1.22	1916–1920	5.08	1.32
			1921–1925	5.95	1.36
			1926–1930	5.56	1.28
			1931–1935	6.36	1.15
			1936–1940	6.22	1.08
			1941–1945	7.21	1.08

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