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Reasons for the decline in mortality in Sri Lanka immediately after the Second World War: a re-examination of the evidence*

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Abstract

Newman estimated that 42 per cent of the decline in mortality in Sri Lanka between 1930-1945 and 1946-1960 was attributable to DDT-spraying; Molineaux estimated 27 per cent; Gray judged that 23 per cent of the decline between 1936-1945 and 1946-1960 was due to DDT. Here the Newman-Gray-Molineaux approach is criticized, the main point being that they ignored a significant improvement in mortality in the early 1940s, before DDT-spraying. Bearing this, and certain other complexities of the situation, in mind, an attempt is made to assess the impact of DDT on mortality.

Immediately after the Second World War Sri Lanka (then Ceylon) embarked on a program of DDT-spraying designed at least to suppress, and if possible even to eliminate, malaria, by killing the adult mosquitoes responsible for spreading the disease. Until then malaria had figured very prominently in Sri Lankan morbidity and mortality. Death rates then improved considerably in the late 1940s, prompting the suggestion by some that DDT-spraying had been largely responsible. However, others disagreed and a debate ensued about the relative importance of the DDT campaign, on the one hand, and improved provision of health services or a variety of social and economic factors, on the other. The population of Sri Lanka, according to the 1946 census, was 6.7 million.

DDT-spraying had begun in earnest in November 1945, after a few small-scale experiments earlier that year utilizing supplies provided by the military authorities, and covered all malarial areas by 1947 (Rajendram and Jayewickreme 1951:22; Visvalingam 1961:64). According to Cullumbine the effect was dramatic: noting a marked drop in the crude death rate in Sri Lanka from 20.3 deaths per 1000 population in 1946 to 14.3 in 1947, and 13.2 in 1948, he observed: 'This sudden drop in the rate can be attributed almost entirely to the near-eradication of malaria following the successful use of D.D.T. as a control measure for mosquitoes' (Cullumbine 1950:120). Sarkar, however, disagreed, arguing that mortality was already following a long-run downward trend in Sri Lanka from well before the Second World War, and that the postwar decline might well be largely a continuation of this (Sarkar 1957:121-125). Sarkar was

> ...inclined to believe that the low death rate of today has been the result of a number of factors of which D.D.T. spraying is one, which have been operating with a cumulative effect ... The operation of this process has probably been accelerated recently by the improvement in curative and preventive medicine, in the political status of the country and in the economic situation generally (Sarkar 1957:124-125).

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Coale and Hoover (1958:62-67) concluded that DDT-spraying was responsible for 'something less than half' (p.66, footnote 13) of the decline in mortality in Sri Lanka between the period 1936-1945 and the period 1950-1952. Newman estimated that 42 per cent of the fall in the average annual crude death rate in Sri Lanka between the periods 1930-1945 and 1946-1960 was attributable to DDT-spraying, though possibly aided by quinine use (Newman 1965:3,48-49; 1970:157, note 17; Newman and Meegama 1969:285).

Meegama (1967) believed that Newman's analysis overstated the significance of the DDT campaign. He argued that Newman had overlooked a very important point: that in the immediate postwar period the malarial areas of Sri Lanka had profited not only from DDT-spraying but also from a disproportionate improvement in health services, as well as possibly in nutrition. Meegama also expressed the view, though this point was put less strongly and not pressed by him in the subsequent debate, that mortality in Sri Lanka was already clearly falling in the late 1930s and the early part of the war, so that the postwar decline in mortality should in part be seen as the resumption of an earlier trend which had then been interrupted by various difficulties in the latter part of the war.

Meade (1968) did not accept this second point of Meegama's and found it difficult to make a judgement in the case of the first. He concluded that although health measures of one kind or another were responsible for the fall in mortality in Sri Lanka (and some other countries) after the war 'the relative importance of malaria eradication by residual spraying as against other general and less specific health measures is to some extent an open question' (p.109). Very similar views to Meade's were expressed in a leading article in *The Lancet* at the time (1968, 1:899-900).

Frederiksen (1970) believed, like Meegama, that Newman had overestimated the impact of DDT in Sri Lanka. He saw the postwar improvement in mortality as largely the resumption of a long-run downward trend after an interruption in the latter part of the war; and felt that fluctuations in standard of living and especially in food availability might well be an important element in the situation (Frederiksen 1960, 1961, 1962, 1966). He thought that no more than 19 per cent of the decline in the crude death rate in Sri Lanka between 1944, the last year before spraying began, and 1954, which he considered the appropriate comparison to make, could have been due to DDT-spraying (Frederiksen 1970).

Gray (1974) estimated that 23 per cent[1] of the postwar decline in the crude death rate in Sri Lanka was attributable to DDT-spraying. His approach followed that of Newman (1965) but with two modifications. Newman's estimation depended on a linear regression equation linking the absolute fall in the average annual crude death rate between the periods 1930-1945 and 1946-1960 in the 21 districts of Sri Lanka, as dependent variable, to the average so-called 'spleen rate' in the districts for the period 1938-1941. The 'spleen rate', the proportion of school children found in surveys to have an enlarged spleen, was taken as an indication of the level of malaria in a district, an enlarged spleen being one of the possible symptoms of malaria; the years 1938-1941 were the last for which this information was available before to the DDT campaign. Since spleen rates effectively became zero throughout Sri Lanka not many years after the Second World War (Newman 1965:91-92) the rates for 1938-1941 also represent the improvement in spleen rates that took place over this period. Gray modified Newman's approach, first, by using as dependent variable in the linear regression the *proportional* rather than absolute decline in the crude death rate, which he argued was more appropriate, and secondly, by considering changes not between 1930-1945 and 1946-1960 but between 1936-1945 and 1946-1960: this latter change was because there was a very serious malaria epidemic in Sri Lanka in 1934-1935 which Gray believed might distort the analysis.

[1]According to Newman (1977:260), given Gray's approach, this figure should have been 22 per cent.

Newman subsequently seemed implicitly to accept this second point of Gray's in that he too compared the periods 1936-1945 and 1946-1960 when he re-examined this question using a Box-Cox approach; however his 'best' estimate of the contribution of the DDT-campaign to the fall in the crude death rate in Sri Lanka between these two periods, using this approach, was still 44 per cent; with his original regression approach but the periods suggested by Gray, Newman's estimate would have been 48 per cent (Newman 1977). Molineaux, on the other hand, felt that Gray's regression approach, relating the proportional decline in the district crude death rates to the 1938-1941 spleen rates, was the more realistic, though he preferred the periods of time employed originally by Newman; on this basis he calculated that 27 per cent of the reduction in the crude death rate in Sri Lanka between 1930-1945 and 1946-60 was attributable to DDT-spraying (Molineaux 1985).

Both Gray (1974) and Newman (1977) specifically rejected Meegama's assertion that in the immediate postwar period malarial areas in Sri Lanka enjoyed a disproportionate improvement in health services and possibly in nutrition. Neither of them seriously dealt with Meegama's other point, that there was a noticeable drop in mortality just before and in the early part of the Second World War, and that this must be taken into account in considering the possible reasons for the postwar mortality decline. Gray's analysis simply did not point to such a dip in mortality (Gray 1974). Newman, responding to observations made by Frederiksen about a mortality decline in that period, spoke of the 'well-known dangers in basing trends in a fluctuating series on such a short time series' (Newman 1970:153).

In what follows, the whole question of the possible reasons for the decline in mortality in Sri Lanka after the Second World War is re-examined. Possibly the most important point to emerge is that Meegama was right that there was a noticeable fall in mortality in the late 1930s and early 1940s which must be taken into account in judging subsequent developments. This is demonstrated by a more detailed analysis of year-by-year changes in mortality in Sri Lanka than has hitherto been attempted, involving the consideration of infant and maternal as well as overall mortality, and the examination of changes in different parts of the country, supported by some completely new analysis of a whole range of data relating to public health activities in Sri Lanka. A number of other significant points, overlooked in previous work, are also brought out. It should be emphasized that this matter does not just have historical significance, though that would make it important enough. Views about what happened in Sri Lanka may well inform and therefore influence present-day approaches to malaria control (see, for example, the recent review by Bradley 1993).

Malaria and malaria control in Sri Lanka before DDT-spraying

Sri Lanka is an island not far north of the equator ($5^{o}55'$ - $9^{o}50'$N), just off the south-east tip of India. It is about 140 miles across at its widest point and 270 miles from north to south. The south-central part of the island is mountainous, ranging from about 1000 feet to more than 7000 feet above sea level; this is the so-called hill country where most of Sri Lanka's tea is grown. The south-western coastal districts of the island together with this adjoining hill country are well watered and make up the 'wet zone' of Sri Lanka; the remainder of the island, which has much less rainfall, constitutes the 'dry zone' (see map presented as Figure 1). Some accounts refer also to an 'intermediate zone' on the fringes of the other two zones. Whereas the wet zone of Sri Lanka tends to experience rainfall both during the south-west monsoon, which typically blows from May to September, and the north-east monsoon, which typically blows from November to March, the dry zone usually experiences rainfall only in connection with the north-east monsoon; moreover, the south-west monsoon is generally more reliable and associated with heavier rainfall than the north-east monsoon. Both zones tend to experience thunderstorms in the inter-monsoon periods.

Figure 1
Districts of Sri Lanka subdivided into wet zone and dry zone districts

Malaria is spread by female anopheline mosquitoes: a person becomes infected when bitten by a mosquito which has itself taken in malaria parasites some time before by biting an infected person. The particularities of malaria transmission in Sri Lanka were established by Carter in the 1920s and early 1930s. He was a medical entomologist appointed by the Government of Ceylon in 1921 to study the problem. Carter discovered that although there were 15 or more species of anopheline mosquitoes in Sri Lanka, several of which were, moreover, known to be implicated in malaria transmission elsewhere, only one species was involved in malaria transmission in Sri Lanka: *Anopheles culicifacies*[2] (Carter 1927; Gill 1935; Visvalingam 1961). This was essentially a dry zone species, though in particular circumstances it proliferated in parts of the wet zone.

The dry zone of Sri Lanka had (indeed has) a very large number of water storage 'tanks' with associated irrigation systems. This, and some other features of the dry zone, meant that the kinds of pools, and puddles, and slow-moving streams in which *A. culicifacies* could breed were fairly common. Hence malaria was endemic in the dry zone, though with seasonal upsurges in the aftermath of rain. In the wet zone, on the other hand, in the ordinary course of events there were few opportunities for *A. culicifacies* to breed, though it was always present, hence little malaria. However, if there was a prolonged drought in the wet zone, pools would form in river beds, because of the fall in water levels, which provided ideal breeding sites for *A. culicifacies*, and a malaria epidemic would ensue; the impact of such an epidemic would be the greater because of the relative lack of previous exposure and therefore immunity of the population to the disease. There tended to be such epidemics of malaria in the wet, and especially the 'intermediate', zone of Sri Lanka about every five years; the worst of these came as a result of the complete failure of the usually relatively reliable south-west monsoon. Since the breeding capability of *A. culicifacies* is markedly reduced at altitudes over 2500 feet, the more elevated parts of the hill country were, however, completely free of the disease, or at least of locally-generated cases of it (see Carter 1927; Briercliffe 1935; Gill 1935; Rajendram and Jayewickreme 1951; Visvalingam 1961).

According to Rajendram and Jayewickreme (1951:2) there were malaria epidemics in Sri Lanka in 1906, 1911, 1914, 1919, 1923, 1928-1929, 1934-1935, 1939-1940, 1943 and 1945-1946.

The fact that malaria is transmitted by mosquitoes had been known since the very end of the nineteenth century following work by Ross. In Sri Lanka, from the beginning of the twentieth century, attempts were made to control malaria by eliminating mosquitoes' breeding places: through drainage of wet areas, through in-filling of depressions, through repair and proper maintenance of water channels, through thorough and regular removal of refuse, which might otherwise provide receptacles for water, etc. Later, in addition, mosquito breeding sites were sprayed with oil, or various other compounds, in order to kill mosquito larvae; larvivorous fish were introduced. There was also some use of insecticides. Beginning in a small way but increasingly as time went on, quinine was distributed, both for malaria treatment and for use prophylactically (see Rajendram and Jayewickreme 1951; Visvalingam 1961; see also the annual *Reports of the Principal Civil Medical Officer of Ceylon* and the later *Reports of the Director of Medical and Sanitary Services of Ceylon*).

The extent of such anti-malarial activity in Sri Lanka was relatively limited initially but increased during the 1920s and into the 1930s. The particularly severe malaria epidemic of 1934-1935 undoubtedly came as a shock and even an affront to Sri Lanka's Department of Medical and Sanitary Services. Following reports on this outbreak by Briercliffe (1935) and Gill (1935) the Malaria Control and Health Scheme was brought into operation in late 1936. This expressly provided for not only a whole battery of direct anti-malarial activities,

[2] *A. culicifacies* is still the only proved malaria vector in Sri Lanka.

including precautionary larvicidal spraying in areas judged to be showing pre-epidemic signs, but also a range of more general health measures: it embodied the notion that malaria could not be seen in isolation from more general health problems and that tackling these problems would also reduce the impact of malaria; the scheme was seen as having particular relevance for rural areas. In the words of the Director of Medical and Sanitary Services for Ceylon in his report for 1937,

> Malaria in rural areas cannot be dealt with in the same way as in urban areas. In the latter ... it is possible for intensive anti-larval work to be undertaken on a reasonably economical basis; but this is not possible in wide areas with scattered population and where rice cultivation depends generally on artificial irrigation. The work that is being carried out consists of direct and indirect methods for the amelioration of existing conditions in regard to malaria. The direct method is chiefly the treating of the disease and the control of the insect vector as far as possible and the indirect method deals with conditions the existence of which aggravate the incidence of malaria[,] by caring for the mother and child through maternity and child welfare work, by caring for the school child through school health work, by giving mass hookworm treatment, by treatment for parangi [yaws], by control of communicable diseases, general sanitary work, and by health education (Ceylon 1938: Part 4, C38).

The Malaria Control and Health Scheme continued in operation through the late 1930s and throughout the Second World War, though with varying activity, and was still in place at the time of the postwar DDT-spraying campaign. Beginning in 1943, and especially from 1944 onwards, in addition to larvicidal spraying of mosquito breeding sites, there was fairly extensive spraying of pyrethrum insecticide inside houses to kill adult mosquitoes.[3] This activity was superseded as the DDT-spraying campaign got under way. DDT was also used as an insecticide, in the domestic setting. However it was longer-lasting in its effect, being sprayed on walls and other surfaces and continuing to kill mosquitoes which landed there for some time, i.e. it was a 'residual' insecticide, so that much less frequent spraying was required than with pyrethrum (see Rajendram and Jayewickreme 1951; Visvalingam 1961; also the annual reports of the Principal Civil Medical Officer and the Director of Medical and Sanitary Services).

Trends and fluctuations in mortality in Sri Lanka, 1900-1954

All of the measures presented here have been calculated from Sri Lankan census and registration data. Census data were taken from the census reports: there were censuses of Sri Lanka in 1901, 1911, 1921, 1931, 1946 and 1953. Registration data were obtained from the annual *Reports of the Registrar General of Ceylon on Vital Statistics*.

Material is presented for the whole of Sri Lanka and for five areal subdivisions of the country, each subdivision comprising one or more of the (then) 21 administrative districts of the island. Sri Lanka was split first into 'wet' and 'dry' zones; the wet zone was then further subdivided into three areas and the dry zone into two areas (see Table 1 and map presented as Figure 1). The grouping of districts into wet and dry zones has been taken from United Nations (1976:35).

[3]In fact, there had been some, intermittent and small-scale, spraying of insecticide inside houses in Sri Lanka since 1934.

Table 1
Minimum, maximum and mean annual infant mortality rates over the period 1900-1944, and average spleen rates 1938-1941, for the districts of the wet zone and of the dry zone of Sri Lanka

Zone, sub-zone and district		Infant mortality rate[a]			Average spleen rate[b] 1938-1941%
		Min	Max	Mean	
Wet zone					
I	Kalutara	81	144	117	1.4
	Galle	100	159	133	2.9
II	Colombo	103	186	150	2.4
	Negombo	103	271	141	9.4
	Matara	96	178	135	16.0
III	Kandy	119	306	186	10.2
	Nuwara Eliya	121	254	193	10.7
	Ratnapura	113	254	175	12.2
	Kegalle	93	482	158	13.6
Dry Zone					
I	Jaffna	120	240	185	6.7
II	Matale	133	433	218	39.2
	Hambantota	152	393	235	62.6
	Mannar	215	574	346	35.4
	Mullaittivu[c]	200	581	298	65.3
	Batticaloa	164	306	224	37.8
	Trincomalee	164	462	245	39.5
	Kurunegala	131	716	243	39.6
	Puttalam	226	485	313	50.4
	Chilaw	99	329	153	17.3
	Anuradhapura	194	487	292	67.3
	Badulla	114	263	192	25.1

[a]Registered infant deaths in year divided by registered live births, expressed per 1000 live births.

[b]The spleen rate is the percentage of school children found in surveys to have an enlarged spleen. These figures come from Newman (1965:92).

[c]Mullaittivu district was subsequently renamed Vavuniya district and has now been split into Mallaittivu and Vavuniya districts.

This fivefold areal subdivision of Sri Lanka was adopted largely on the basis of what previous researchers had concluded about the differing importance of malaria historically in different parts of the country. The dry zone was distinguished from the wet zone since, as already noted, malaria had been found to be endemic in most of the dry zone but hardly at all in the wet zone. The district of Jaffna was, however, separated out from the rest of the dry zone, since previous work suggested that malaria levels there were more akin to those found in the wet zone than the dry zone. Kalutara and Galle districts were separated out together from the wet zone since historically these areas had been found to suffer little from malaria. The remainder of the wet zone, parts of which were subject to malaria epidemics, was divided into two, mainly on the basis of the mortality differences indicated by the data of Table 1 but also bearing in mind that one of the resulting areas, labelled as sub-zone III of the wet zone in Table 1, included many tea estates and so might have different experience from

other areas (see Carter 1927; Briercliffe 1935; Gill 1935, 1940; Abhayaratne 1950; Rajendram and Jayewickreme 1951; Visvalingam 1961; Newman 1965; Gray 1974).

The data shown in Table 1 by and large confirm the appropriateness of this subdivision of Sri Lanka. In general, dry zone districts had higher infant mortality than wet zone districts and higher spleen rates, suggesting more malaria. Jaffna, on the other hand, was more similar to some wet zone districts in these respects than to other dry zone districts. Kalutara and Galle were the most favoured districts in the island; even Colombo district, which like them had an extremely low 1938-1941 spleen rate, nevertheless had somewhat higher average infant mortality and higher peak mortality, the latter feature probably indicating a greater susceptibility to epidemic malaria. On the figures of Table 1, both Chilaw district and Badulla district might possibly have been dealt with separately from other dry zone districts. This would have complicated the analysis quite considerably, however, without any very great benefit; so this approach was not adopted.

The graphs presented in Figure 2 show changes in the crude death rate, infant mortality rate and maternal death rate for Sri Lanka over the period 1900-1954. The data on which these graphs are based are presented in an appendix; this is so for all graphs shown. Crude death rates were calculated by dividing the registered deaths in a year by the total population and expressing the result per 1000 total population; populations in non-census years were estimated by assuming a constant rate of growth between censuses. This method of estimating denominators was obviously not exact; however, the fact that, as may be seen from Figure 2, the resulting crude death rates correspond almost exactly with those produced for 1930 and later years by Newman (1965:89) using denominators estimated in a much more elaborate fashion, suggests that these data are, nevertheless, reasonable. Infant mortality rates were calculated by dividing the infant deaths registered in a year by the registered live births, and maternal death rates by dividing the maternal deaths, that is, deaths associated with pregnancy or childbirth, registered in a year by the registered live births, in each case expressing the result per 1000 live births.

It may be seen from Figure 2 that mortality in Sri Lanka fluctuated a great deal. Most of the upsurges in mortality were associated with outbreaks of malaria, though in 1918-1919 there was a very serious influenza epidemic, which was itself then further added to by a malaria outbreak (Langford and Storey 1993a). From 1900 until about 1920—attempting to set aside short-run fluctuations—the data seem to suggest a slight upward trend in mortality: this may be genuine but is more likely to be a reflection of improvements in data quality. From about 1920 onwards—again, disregarding short-run fluctuations—there was a general downward trend in mortality, though in the case of maternal mortality this might not have begun until the late 1930s. Marked fluctuations continued, however, the most notable being the terrible rise in mortality associated with the malaria epidemic of 1934-1935, until the immediate post-World War II period, when they apparently disappeared.

All of the data presented in Figure 2 indicate that there was a remarkable improvement in mortality in Sri Lanka after the Second World War. Mortality dropped in 1947 to lower levels than seen previously (since 1900) and there was further improvement subsequently. In some degree, however, the fall in 1947 is made to seem more precipitate than it truly was by the fact that mortality rose in Sri Lanka in the later years of the war and just afterwards. Moreover, before this rise, mortality in Sri Lanka had actually already been falling quite sharply, albeit only for a short period, to what were then unprecedentedly low levels, the crude death rate and infant mortality rate dropping to a low-point in 1942 and the maternal death rate in 1943. There had been a previous dip in the crude death rate, though apparently not noticeably in infant and maternal mortality, in the early 1930s.

Figure 2
Crude death rate, infant mortality rate and maternal death rate for Sri Lanka, 1900-1954

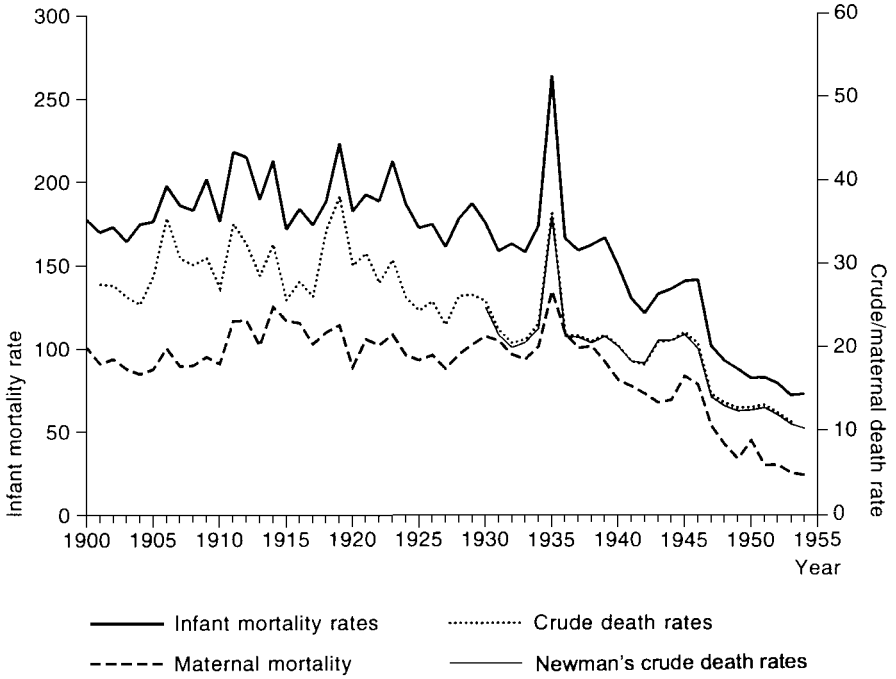

Figure 3 shows changes in the crude death rate over the period 1900-1954 for each of the five areal subdivisions of Sri Lanka; Figure 4 shows changes in the infant mortality rate. It may be seen that all these areas shared in the general downward trend in mortality after about 1920 in Sri Lanka. Areas differed, however, until the late 1940s, anyway, in terms of both the typical level of mortality and the extent of mortality fluctuations. In the wet zone, sub-zone II (see Table 1 for the districts this included) after about 1920 tended to have similar or slightly higher mortality than sub-zone I in 'good' years but a larger gap opened between the two areas when malaria struck, reflecting the greater susceptibility of sub-zone II to epidemic malaria; in sub-zone III of the wet zone mortality tended to be somewhat higher still and the fluctuations due to epidemic malaria even more marked. In the dry zone, Jaffna district had higher mortality, in general, than sub-zones I to III of the wet zone but fluctuations in mortality were not especially marked; this area seems to have been completely unscathed in the 1934-1935 malaria outbreak. The remainder of the dry zone had higher mortality generally than other areas of the island as well as marked fluctuations in mortality. In all parts of Sri Lanka, however, from 1947 onwards, fluctuations in mortality were small and differences between areas relatively slight.

Of special interest in the present connection is whether the fall in mortality in 1947 in Sri Lanka as a whole occurred also in much the same way in these different areas of the country. Considering the period from 1920 on, it may be seen from Figure 3 that, so far as the crude death rate is concerned, there is no real sign of a noticeable discontinuity of trend in 1947 in sub-zone I of the wet zone; moreover, if temporary upsurges in mortality are ignored, the same can be said in relation to sub-zones II and III of the wet zone. In the case of Jaffna

Figure 3
Crude death rate per 1000 population for the sub-zones of the wet zone and the dry zone of Sri Lanka, 1900-1954

Figure 4
Infant mortality rate per 1000 live births for the sub-zones of the wet zone and the dry zone of Sri Lanka, 1900-1954

district there was apparently some acceleration in the decline of the crude death rate in the post-World War II period, though if the improvement in the rate over the period 1934-1940 is used as a basis for judging likely future developments rather than the whole period from 1920 on, the postwar dip below trend seems rather slight. In the remainder of the dry zone there was, on the face of it, a clear downward shift in the crude death rate in 1947; however, even in this case, there is the question of whether the very short-lived, but quite marked, improvement in the rate in the early 1940s, to what was then the lowest level ever (since 1900), should be regarded as signalling the impending postwar improvement. There were, it may be noted, new low-points in the crude death rate in all the sub-zones of Sri Lanka except sub-zone I of the wet zone in the early 1940s.

The data on infant mortality presented in Figure 4 point even more strongly to the question of whether the downward trend in mortality in Sri Lanka might not have taken on a new impetus in the late 1930s or early 1940s, thus in some sense anticipating the postwar decline. There was apparently quite a noticeable fall in the infant mortality rate in the early 1940s, albeit very short-lived and rapidly reversed, in all parts of Sri Lanka except sub-zone I of the wet zone; even in that area there seems to have been some acceleration of the pre-existing downward trend in the late 1930s and early 1940s.

Changes in the intensity of public health activities in Sri Lanka, 1930-1954

A variety of data relating to public health activities in Sri Lanka over the period 1930 to 1954 is presented in Tables 2 and 3. These data have been compiled from material provided in the annual *Reports of the Director of Medical and Sanitary Services of Ceylon*, and the *Education* reports, together with some information from the census and from vital registration. Even at the very beginning of this period there was clearly a great deal going on: most infants seem to have undergone at least primary vaccination against smallpox[4]; a very large number of individuals were treated for hookworm infestation each year, that is, wormed (for an account of the debilitating effects of this disease in Sri Lanka see Langford and Storey 1993b); and the School Medical Service carried out a large number of medical examinations each year among children in schools. In the early 1930s government expenditure on health was cut, as part of a general response to the economic difficulties of that period, and there seems to have been a reduction in the number of primary smallpox vaccinations and in the number of medical examinations in schools; the anti-hookworm campaign, on the other hand, apparently increased its coverage during this time. In 1935 there was a considerable increase in government spending on health (strictly, this was in the 12-month period beginning on 1 October 1934). However, this was obviously largely, and possibly entirely, simply a response to the appalling malaria outbreak at that time. The number of malaria cases treated as inpatients in hospitals or, overwhelmingly, as outpatients in hospitals or dispensaries in 1935 (5,454,781) amounted to 97 per cent of the population of Sri Lanka at that time.[5] By contrast,

[4] Only data on primary vaccinations against smallpox have been shown in Table 2 even though numbers of secondary vaccinations are also provided in the official reports. It was felt that numbers of primary vaccinations give a better indication of the general level of vaccination activity, whereas numbers of secondary vaccinations tend to fluctuate more in response to actual or feared smallpox outbreaks.

[5] See *Administration Report of the Acting Director of Medical and Sanitary Services for 1935*, page C28. The number of cases reported is extremely high (though clearly labelled as such) and it is conceivable that the same individual could have been treated more than once in the same year, or even (as suggested by an anonymous reviewer) that the number of visits rather than cases has been reported. Even so, the number of malaria cases treated in 1935, and the expense of providing this treatment, must have been very high indeed.

Table 2
Some indicators of public health activities in Sri Lanka, 1930-1954

Year	Govt. spending on health[a] (millions)	Change from year before (%)	Govt. spending on school meals[b] (1000s)	Primary smallpox vaccinations[c]		Total hookworm treatments[d]		Typhoid inoculations[e] (1000s)	
				Number (1000s)	As % of last year's births	Number (1000s)	As % of population	1st	2nd
1930	10.7			170	86	1,294	25	5	2
1931	9.7	-9		149	73	1,406	27	4	2
1932	9.8	1		104	52	1,827	34	7	3
1933	9.3	-5		94	47	1,824	33	9	4
1934	9.4	2		143	6	1,992	36	13	7
1935	11.7	24		97	47	1,402	25	10	6
1936	11.0	-7		145	75	1,856	32	8	6
1937	11.1	1	242	163	85	2,163	37	25	18
1938	12.1	9	623	190	88	2,170	37	41	32
1939	12.9	7	972	150	72	2,112	35	-	-
1940	12.9	0	1,257	178	84	2,147	35	69	55
1941	13.4	4	1,284	177	83	1,899	31	72	56

1942	14.3	7	1,506	176	80	890	14	101	77
1943	15.7	9	1,674	196	89	813	13	122	94
1944	18.2	16	3,764	168	68	1,420	22	128	96
1945	21.6	19	5,229	153	66	1,332	20	54	39
1946	28.7	33	5,863	193	81	1,376	21	55	41
1947	37.2	30	7,049	192	75	1,806	26	87	59
1948	51.7	39	7,338	217	80	1,898	27	160	116
1949	57.0	10	8.785	225	78	1,902	26	82	61
1950	62.4	9	9,591	214	73	1,861	25	88	80
1951	67.6	8	9,492	233	77	1,880	25	77	56
1952	82.5	22	10,136	228	73	1,810	23	68	55
1953	87.0	6	10,126	237	76	1,728	21	90	66
1954	90.1	4	8	223	69	1,850		83	59

[a]Millions of rupees. Expenditure is in fact for year ending 30 September of year shown. Figures exclude cost of new buildings as well as improvement and maintenance of old buildings.

[b]Thousands of rupees. This expenditure is from education budget, not health budget. Probably for years ending 30 September rather than calendar years.

[c]Figures for 1932 and 1933 officially reported to be too low since primary vaccinations carried out as part of vaccination campaign then in response to smallpox outbreak omitted. 1941 figures may be similarly affected.

[d]These are largely what were terms ‘first’ but include subsequent treatments; hence treatments may exceed individuals treated in year.

[e]The figures for 1938 relate to inoculations carried out in schools only; no all-Sri Lanka figures were provided for that year. In the case of 1937 (first and second doses) and 1936 (second doses), disquietingly, though the figures are supposed to be for all-Sri Lanka, slightly higher numbers of inoculations were actually reported as occurring in schools alone.

the smallpox vaccination program, the anti-hookworm campaign and the activities of the School Medical Service all seem to have been curtailed in 1935. This was unquestionably due in part to the direct impact of the malaria epidemic itself, since many health workers were diverted from their usual tasks to help cope with the epidemic, and many others would no doubt have themselves suffered from malaria at that time, but it may well also have reflected continuing financial stringency.

Table 3
Number of government health centres providing maternity and child welfare services in Sri Lanka, clinics held there, and attendance at clinics by mothers and children, and number of medical examinations in schools carried out by School Medical Service, 1930-1954

Year	Number of health centres	Clinics held	Visits to clinics[a] by			School children medically examined (1000s)
			Expectant mothers (1000s)	Infants (1000s)	Pre-school children (1000s)	
1930	40	1,547	1	14	7	81
1931	59	2,089	3	18	17	74
1932	54	2,493	3	21	12	64
1933	73	3,199	5	30	16	61
1934	78	3,952	8	28	18	45
1935	86	4,702	10	28	17	36
1936	77	4,543	17	30	19	53
1937	207	8,395	40	88	40	85
1938	311	9,485	76	158	75	95
1939	408	16,354	101	224	98	69
1940	-	-	119	246	96	77
1941	-	-	155	259	88	72
1942	437	18,695	-	-	-	64
1943	444	15,655	-	-	-	78
1944	461	18,122	-	-	-	61
1945	503	17,227	-	-	-	55
1946	533	19,144	-	-	-	62
1947	572	16,591	324	173	67	90
1948	602	20,488	277	237	73	107
1949	662	23,971	296	330	97	102
1950	701	24,890	313	382	109	84
1951	714	25,509	413	361	168	87
1952	771	27,143	412	385	122	98
1953	755	26,151	333	370	105	128
1954	744	24,556	307	330	107	129

[a]The figure for visits to clinics by pre-school children in 1935 is that reported for the whole island; however, disquietingly, about 3,000 more such visits, almost 20,000, were separately reported for 'health unit areas' alone. The 1947 figures are those that appear in the 1947 Report of the Director of Medical and Sanitary Service of Ceylon; the 1948 report, without explanation, presents different figures for 1947: 289 instead of 324; 199 instead of 173; and 76 instead of 67.

In the years following the 1934-1935 malaria outbreak there was a general upsurge in public health activities in Sri Lanka (see Tables 2 and 3). Government spending on health in 1936, though somewhat lower than in the epidemic year 1935, was considerably higher than in the pre-epidemic period; moreover, this level of expenditure was maintained in 1937 and there were further increases in 1938 and 1939. The coverage of the smallpox vaccination

program improved during this period as did that of the anti-hookworm campaign. From 1937 onwards there was a noticeable increase in the numbers of anti-typhoid inoculations administered. So far as maternity and child welfare services were concerned, while there had been a steady increase in both provision and use of services between 1930 and 1936, in 1937 there was a considerable increase in activity, no doubt associated with the Malaria Control and Health Scheme, already referred to, which came into operation in late 1936. In 1937 there was a dramatic increase in the number of health centres at which maternity and child welfare services were provided, from 77 such centres in 1936 to 207 in 1937, and a considerable increase also in the number of clinics held at these centres; moreover, the numbers of visits to these clinics by expectant mothers more than doubled between 1936 and 1937, as did the number of visits by infants and by pre-school children. The extent of use of these facilities continued to grow quite strongly until at least 1941, after which there is unfortunately a gap in the data of some years. Beginning in 1937 (in fact, late 1936) the government made funds available to enable midday meals to be provided for children in schools.[6] According to the Director of Education for Ceylon in his report for 1937 this was to include (from February 1937) 'all the children in schools in distressed areas' (Ceylon 1938: Part 4, A10). This expenditure on midday meals for school children grew rapidly in the late 1930s and early 1940s.

During the early years of the Second World War public health activities in Sri Lanka were in most respects maintained or even extended; even later on, many activities continued at reasonable levels (see Tables 2 and 3). However, the coverage of the anti-hookworm campaign was drastically curtailed in 1942 and 1943 because of the lack of necessary supplies. The anti-malaria program was probably also constrained by shortage of supplies, at least before 1944. To some extent, moreover, the health of the population would undoubtedly have been undermined by the food shortages that developed following the Japanese invasion, in December 1941, of Burma, which had previously been an important source of rice imports for Sri Lanka. Unfortunately, there is no information on the very important matter of the extent of use of maternity and child welfare services during the period from 1942 to 1946.

At the end of and immediately after the Second World War, as regards most of the specific public health measures for which information is provided in Tables 2 and 3, there was apparently little or no sign of increased activity. The coverage of the smallpox vaccination program and of the anti-hookworm campaign, as well as the extent of inoculation against typhoid, were well below their earlier peak levels. In one very important respect where maternity and child welfare services are concerned there were signs of greater activity: the number of visits to clinics at health centres by expectant mothers in 1947 was substantially higher than in 1941, unfortunately the last year before 1947 for which this information is available, but having the highest recorded figure for any year up to that time. The numbers of visits to clinics by infants and by pre-school children, though, were still apparently somewhat lower in 1947 than their peak recorded levels early in the Second World War, and it was not until two or three years after 1947 that these earlier peak levels were surpassed.

It seems extremely likely, nevertheless, that there was an upsurge in public health activities in Sri Lanka towards the end of the Second World War and afterwards. The specific activities referred to in Tables 2 and 3 are only part of the picture; no data have been presented on the provision of inpatient and outpatient facilities at hospitals or dispensaries,

[6] Even before this time there were some local schemes for the provision of midday meals in schools, funded by municipalities or individuals; and post-1936 there was some local supplementation of government expenditure on school meals, as well as encouragement of school gardens to provide additional supplies.

for example, or the availability of trained public health nurses and midwives, or the involvement of these in visiting people in their homes, all areas where there might conceivably have been improvements in this period. Some data relating to these matters are available but problems of coverage and discontinuities of definition, as well as gaps in the material, make them difficult to interpret. The figures relating to government expenditure on health presented in Table 2 certainly do suggest very strongly that there were improvements in health provision towards the end of the Second World War and afterwards: it may be seen that spending grew especially rapidly in the period from 1944 (in fact, late 1943) until 1948. This does not seem to have been merely a reflection of increased expenditure on malaria eradication during this period: stripping out the costs of the anti-malaria campaign from total government spending on health affects the figures on annual percentage growth in expenditure only slightly.[7] It may also be seen from Table 2 that government spending on midday meals for children in schools, which had increased in successive years from the outset, then more than doubled between 1943 and 1944, and continued to grow quite strongly after that.

The effect of DDT-spraying on mortality in Sri Lanka

The Newman-Gray-Molineaux approach to the assessment of the effect of DDT-spraying on mortality in Sri Lanka is unsatisfactory for a number of reasons. This approach overlooks the fact that, as has been pointed out, over and above the general longer-run downward trend in mortality after about 1920 in Sri Lanka, there was also a fairly dramatic, albeit rather short-lived, fall in mortality in the early 1940s. This fall followed an upsurge in public health activities in Sri Lanka which came about as a result of the introduction of the Malaria Control and Health Scheme: this both attests to the likely genuineness of the fall and draws attention to the possible impact on mortality of such public health measures; moreover the fact that the fall occurred in every part of Sri Lanka, except perhaps sub-zone I of the wet zone, the healthiest part, strongly suggests that these measures could be effective even in the face of endemic malaria. Comparing a longish pre-DDT period of time with the post-DDT situation, which was the Newman-Gray-Molineaux approach—and Coale and Hoover's, for that matter—thus has the effect of concealing an important change in the pre-DDT period which is relevant to the interpretation of the post-DDT changes in mortality.

Another shortcoming of the Newman-Gray-Molineaux analysis is its reliance on the spleen rate as an indicator of the level of malaria in an area. A high spleen rate in an area will undoubtedly be a reflection of a high malaria prevalence in that area, but the latter will depend not only on the risk of malaria infection but also on the likelihood of an attack being serious and prolonged; this in turn will depend partly on the nutritional status and general state of health of the population, so that, in the words of Carter, 'The social status and economic conditions of ... communities ... also influence the rates'; Carter went on to observe that 'Malnutrition ... exerts an important effect in many parts of Ceylon upon ... the maintenance of high spleen rates' (Carter 1927:14). A corollary is that in some degree improvements in nutrition and general state of health would be expected to reduce spleen rates, and malaria morbidity, and malaria mortality, even if exposure to the risk of malaria remained unchanged.

[7] Visvalingam (1961:82) provides figures on the 'expenditure on anti-malaria campaign' from 1934 onwards. No source is given but he was Superintendent of the Anti-Malaria Campaign at the time he was writing. Removing these amounts from the total expenditures on health shown in Table 2 results in annual percentage changes in the remainder which are essentially the same as the percentage changes in total expenditure shown in Table 2.

A further weakness of the Newman-Gray-Molineaux approach is that, expressly or implicitly, it assumes that attempts at malaria control before DDT-spraying were ineffective. Gray simply states (1974:210): 'Before 1945 malaria control was limited to ineffectual larvacidal [sic] methods'. This judgement may well be incorrect. Particularly after the introduction of the Malaria Control and Health Scheme in late 1936, armed with the detailed knowledge of the habits and significance for malaria of *A. culicifacies* provided by Carter, an elaborate system of monitoring potential danger areas and of responding where necessary with larvicidal, and increasingly insecticidal, spraying was established; this was in addition to all the usual activities designed to avoid the formation of bodies of stagnant water, large and small. Those involved certainly did believe that these measures made a difference and in the view of Rajendram and Jayewickreme (1951:21), referring to the malaria outbreaks of 1939-1940, 1943 and 1945-1946, there was 'little doubt that if it were not for the control measures adopted in these years these epidemics might have assumed much larger proportions'. According to Visvalingam (1961:64) 'There is little doubt that the control measures adopted under the new scheme had reduced the problem of malaria and the severity of epidemics during these years'. If pre-DDT control measures did indeed have some effect, which seems very likely, this needs to be borne in mind in thinking about any possible impact that DDT might have had: first, in some degree DDT was merely supplanting earlier measures, so if these were effective then any impact of DDT would have been in some degree a replacement effect; secondly, had DDT not been available, there would undoubtedly have been a great upsurge in antimalarial activity of the pre-DDT type after the Second World War, which if such measures were indeed effective would itself have reduced mortality in some degree in any case.

Given this complexity, can any assessment be made of the impact of DDT-spraying on mortality in Sri Lanka? Consider first only the fall in mortality in the year 1947. It seems reasonable to suppose that, even in the absence of DDT-spraying, the crude death rate in Sri Lanka in 1947 would have fallen back at least to its previous minimum level, reached in 1942. The year 1947 was a favoured year in a number of respects: climatic conditions were not conducive to epidemic malaria[8] and there was a falling-back of mortality following the fairly serious malaria outbreak of 1945-1946, which may well also have 'brought forward' some deaths; in addition, by 1947 food and other wartime shortages had ended and, as noted previously, public health expenditure had noticeably increased. By contrast, the earlier low-point in the crude death rate in 1942 had been achieved despite wartime difficulties.

As may be seen from Table 4 the Sri Lankan crude death rate in 1947 was 14.4 per 1000 population whereas the previous minimum figure, reached in 1942, was 17.9. Thus, assuming that given the conditions in Sri Lanka in 1947 the crude death rate would have fallen at least to the 1942 level, even without DDT-spraying, the maximum possible reduction in the 1947 crude death rate attributable to DDT-spraying is 20 per cent. However, there was no spraying at all in sub-zone I of the wet zone at any stage; moreover, although spraying was carried out in parts of sub-zones II and III of the wet zone, this would have had little or no impact in 1947 since this was not a year with epidemic conditions. Taking account of the reduction in

[8] Rajendram and Jayewickreme (1951) might be thought to have suggested otherwise by stating that (p.46) 'in 1947 both south-west and north-east monsoons were failures'. However, elsewhere in the same article (p.31) they confined themselves to the expression 'partial failure' in relation to the south-west monsoon in 1947 by which they clearly meant that rainfall was considerably below average (their figures referred to the epidemic zone) in April and May (p.57). Since rainfall was well above average in January and in March of 1947 and reasonable, or in the case of August extremely heavy, from June to October, there would have been no real reason to expect epidemic malaria in 1947. Nor did Rajendram and Jayewickreme suggest that there were epidemic conditions in 1947; their argument was that the failure of the north-east monsoon in 1947 would have led to epidemic conditions in 1948.

mortality that occurred anyway in sub-zone I of the wet zone, which could not have been due

Table 4

The minimum crude death rate (CDR) during the period 1936-1944, the crude death rate in each year 1942-1947, and the average crude death rate over the periods 1948-1951 and 1947-1951, for Sri Lanka and the sub-zones of the wet and the dry zone of Sri Lanka[a]

Zone and sub-zone[b]	Year(s)								
	Min CDR	CDR						Average CDR	
	1936-1944[c]	1942	1943	1944	1945	1946	1947	1948-1951	1947-1951
Wet zone									
I	16.5	16.7	16.9	17.7	17.7	16.2	13.6	12.8	13.0
II	15.7	15.7	17.8	19.4	19.7	18.1	13.7	13.6	13.6
III	15.2	15.2	17.6	18.4	20.2	18.1	13.2	13.1	13.1
Dry Zone									
Jaffna	20.6	20.8	24.5	21.5	22.2	17.1	15.5	12.7	13.3
Rest	22.5	22.5	27.1	25.7	27.4	27.7	16.2	12.4	13.1
Sri Lanka	17.9	17.9	20.6	20.8	21.8	20.4	14.4	13.0	13.3

[a]See appendix for complete data.

[b]See Table 1 for the districts comprising the different sub-zones.

[c]This is also the lowest rate recorded up to that time (since 1900) except in sub-zone I of the wet zone where the CDR in 1932 was 16.4.

to DDT-spraying, the maximum possible reduction in the all-island 1947 crude death rate attributable to DDT-spraying falls to 18 per cent; assuming further that no part of the wet zone was affected by DDT in 1947 this figure falls to only 13 per cent. Moreover, even this low figure is very likely to be an overestimate since it assumes that all of the reduction in mortality occurring in the dry zone between 1942 and 1947 is attributable to DDT-spraying, which is clearly highly improbable.

All in all, then, it seems very unlikely that the striking fall in mortality in Sri Lanka in 1947 itself had very much to do with DDT-spraying. However, what of the period immediately following 1947? So far as the wet zone is concerned, it may be seen from Table 4 that mortality fell only a little further in the next four years, so on the face of it hardly requiring a radical explanation. However, this is to presume that the absence of conditions conducive to epidemic malaria characteristic of 1947 continued, whereas, at least according to Rajendram and Jayewickreme (1951:12, 22, 47, 49), both 1948 and 1950 were years in which epidemics would very probably have occurred historically, given the climatic conditions. This being so, it could obviously be argued that DDT-spraying had prevented these epidemics and hence the mortality they would have caused. However, making a judgement on this would require not only an assessment of the likelihood of a malaria epidemic in these years in the absence of any control measures but an assessment of that likelihood given only pre-DDT control measures, though possibly on a considerable scale; moreover, a judgement would also be required on the possible extent to which mortality in any epidemic which did occur might have been reduced, perhaps considerably, by the improvements in nutrition and in the availability of health services which very probably occurred in this period. So far as the dry zone is concerned, it may be seen from Table 4 that in the few years immediately following 1947 mortality fell distinctly further, below 1947 levels. Thus two questions arise in connection with the dry zone: first, what part did DDT play in reducing mortality to these new low levels? Secondly, rather as in the case of the wet zone, what part did DDT play in preventing any periodic malaria outbreaks, and consequent mortality, that would otherwise have occurred?

Suppose that, as argued previously, DDT-spraying did not affect mortality at all in the wet zone in 1947; and suppose further that one-half of the reduction in the crude death rate in the sub-zones of the dry zone between 1942 and 1947 was attributable to DDT-spraying, which seems unlikely to be an underestimate; then the crude death rate for Sri Lanka in 1947, in the absence of DDT-spraying, would have been 15.4 per 1000 population, rather than the observed rate of 14.4 per 1000. If, without DDT-spraying, the average crude death rate for Sri Lanka over the four years following 1947 would have exceeded 15.4 by the same proportion as the average crude death rate over 1943 to 1946 exceeded that for 1942, the average crude death rate for Sri Lanka over the period 1948 to 1951 would have been 18 per 1000, meaning that the average observed crude death rate for the five-year period 1947-1951 was 24 per cent below the average expected rate in the absence of DDT-spraying. However, this is likely to be a considerable overestimate of the impact of DDT-spraying in this period: as already suggested, there is every reason to believe that in the absence of DDT, pre-DDT antimalarial measures would have been deployed on a massive scale as epidemic conditions developed; moreover, for the very many reasons already given, mortality would almost certainly have been very much lower in any malaria outbreak that had occurred than during the wartime period. If the potential proportional rise in mortality in Sri Lanka during the period 1948-1951, over 1947, were judged to be half that which occurred during 1943-1946, over 1942, then the average observed crude death rate for 1947-1951 would have been 19 per cent below the average expected rate; if the potential rise were judged to be a quarter of the 1943-1946 rise, this would fall to 16 per cent. Even these figures will tend to be overestimates in that they overlook the fact that there were further noticeable improvements in mortality after 1947

in the dry zone, some part of which would undoubtedly have occurred, assuming favourable climatic conditions, even in the absence of DDT-spraying; meaning that, effectively, the baseline crude death rate of 15.4 per 1000 used in the calculations and hence also the estimates of the potential mortality in outbreaks of malaria are somewhat too high.

Conclusion

It is clearly no easy task to estimate the impact of DDT-spraying on mortality in Sri Lanka after the Second World War. The problem is not so much with the apparently dramatic fall in mortality in the year 1947 itself: it is very unlikely that DDT-spraying played much part in this. The problem is to assess its impact in the years that followed. This requires a whole range of judgements in relation to events which did not occur: the likelihood of a malaria outbreak given certain meteorological circumstances; the extent to which the probable mortality associated with such an outbreak would have been moderated by improvements in nutrition or health services; the extent and impact of the antimalarial measures that would have been adopted had DDT not been available; and so on. The results of illustrative calculations, on various assumptions, have been presented above; for what it is worth I personally suspect that the effect of DDT-spraying on the 1947-1951 crude death rate may well have been at or below the lowest assessment provided, that is, a reduction of 16 per cent; however, the hard truth is, regrettably, that there does not seem to be an adequate empirical basis for arriving at a reliable conclusion.

A number of comments should be added, finally, about analyses that were attempted without success or could not be attempted because of lack of data. Despite considerable effort, it has proved possible neither to verify nor refute Meegama's assertion that formerly malarial districts enjoyed a disproportionate improvement in health services after the war; it was concluded that there were simply too few data available on a district basis to permit this. Another potential area of analysis to which a great deal of attention has been given without success is causes of death. Although a large quantity of material on causes of death is available for Sri Lanka, it was concluded, very reluctantly, that it is not of sufficient quality to be helpful in this case; the main problem is the very large 'pyrexia' category (meaning, simply, fever); it was felt that whilst this might well often conceal largely malaria the relationship between malaria and 'pyrexia' could not be taken as fixed, indeed probably was not, over time and space. An area, lastly, where analysis which might have been important has not proved possible because of the complete absence of data, is that concerned with the timing of DDT-spraying in different parts of Sri Lanka. If information had been available, which does not seem to be the case, on the dates at which spraying began in different areas of the country, it might well have been possible to examine the relationship of this timing to changes in mortality, and so throw light on the question of a possible connection between DDT-spraying and mortality decline.

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