Measles in South Africa: A Comprehensive Interpretation of the Data. Part III.

Other Factors influencing measles morbidity and severity in SA (Protein-energy-malnutrition, age at infection, urbanisation, socio-economic status and health care — P Ferrinho, E Buch

**Summary**

In South Africa (SA) there is a commitment to, and indications that resources are being allocated for the eradication of measles. Still, there has been no comprehensive review of the epidemiology of the disease in SA. This understanding is important to identify factors and trends to guide public health practice. This series of articles tries to cover this gap.

Part I reviews briefly the international literature on the epidemiology of the disease and describes the methodology followed, the sources of data and analysis strategy. The other articles review South African reports on morbidity — mortality and relationships to age, population group, sex and geographical distribution (Part II); Part III reviews other factors influencing measles morbidity and mortality in SA (protein-energy-malnutrition, age at infection, urbanisation, socio-economic status and health care); and Part IV contains appropriate conclusions and recommendations.

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**KEYWORDS:** Measles; Epidemiology; Age Factors; Socio-economic Factors; Urbanization; Measles Vaccine; Vitamin A

**Introduction**

In this article we show that measles still remains a common problem in South Africa (SA) with a high case fatality rate (CFR) and that “race”, vaccination coverage, poverty, urbanisation and vitamin A deficiency are important factors in understanding its morbidity and mortality.

There are other possible determinants of incidence and severity of measles. Because of their importance in SA and the availability of data, we examine the influence of protein-energy-malnutrition, age at infection, urbanisation, socio-economic circumstances and availability of health care on incidence and severity for the country.

**Protein-Energy-Malnutrition and Measles**

There are South African reports that identify protein-energy-malnutrition as an important determinant of measles severity. However, all these reports are hospital-based and fail to control factors like overcrowding and micro-nutrient deficiencies, which are more likely to be the relevant determinants in socio-economic environments where malnutrition is common. As vitamin A deficiency seems to be common in SA, the high measles CFR observed could, to some extent, be due to this.
Morbidity - Mortality, Age Distribution and Population Group

Measles data by age and population group is analysed for case notifications, deaths notifications, certified deaths, active surveillance, outbreak investigations, hospital data and data community surveys.

Although the age structure for each population group in South Africa varies, the overall national age structure is that of a developing country with less than 5% of the population under one year of age, 20% below 5 years, 15% 5 to 9 years, 15% 10 to 14 years and 50% below 15 years.

Notifications

For 1985-1987 a significant proportion (26% for Coloureds, 22% for Blacks, 20% for Asians and 7% for Whites) of notified measles occurred in children under one year of age (Fig 1). A similar age pattern can be seen for the period 1988 to July 1990. Although younger age is likely to lead to increased chances of notification, due to increased hospitalisation and mortality, this is still a very high proportion.

Children 1-4 years of age account for 46% for Blacks, 36% for Asians, 31% for Coloureds and 22% of the notified disease for Whites.

In 5-9 year olds, Blacks still have the highest age specific incidence, but Coloureds and Asians now have lower rates than Whites. In children over 10 years of age Whites have the highest incidence rates. Notification data also demonstrates that in the population groups with an average household size greater than five (Asians, Blacks and Coloureds) the risk of measles infection amongst the under ones is three to six times that of older children. Regional reports from the Cape indicate similar trends to national notifications. In Cape Town the notification rate was 60 per 100 000.
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in 1986 with rates in children under 1 year of age of 816 per 100,000.24

The Cape data also suggests that urbanised children are more likely to get infected at a younger age than rural children.25 In Kangwane and Bophuthatswana excess notifications occur in children 5-9 years of age and in all the other areas in the first year of life.26

There is no report available that provides an age breakdown of notified deaths.

**Active surveillance data**

In Johannesburg-Benoni the median age for all notified measles cases was 37 months, 10% of the cases were younger than 9 months and 18% in Johannesburg and 19% in Benoni were under 12 months.27

**Outbreak investigations**

The Port Elizabeth epidemic in 1983 showed the highest and the earliest incidence of measles in the most overcrowded shanty camps. The mortality was particularly high in the socio-economically poor squatter areas. Fifty four percent (54%) of Blacks and 62% of Coloured deaths were < 1 year of age (84% and 79% respectively were < 2 years of age). The highest age specific CFR was in children under 6 months of age (44%).28

**Hospital data**

During 1985-1986, 72% of children admitted to the City Hospital with acute complicated measles were children aged 15 months and under.29 Similar figures were found in 1973 where 90% of cases were below 3 years of age, 50% were younger than 15 months, and 25% were less than 10 months old.30

A report from the intensive care unit at the Red Cross War Memorial Children's Hospital, Cape Town, identifies the median age of children as 9 months, with 9% of the admissions in children younger than 6 months (January 1985 to April 1986).31

In Clairwood and King Edward Hospitals in Natal, 28% of 111 measles cases were below 9 months (August 1986).32 In Baragwanath (in 1987) 72% of admissions were under 2 years and 36% were 9 months or younger.33

Data from the City Hospital in Cape Town and from the Red Cross War Memorial Children's Hospital illustrate the high load of measles mortality in children under five years, particularly infants34-35 while in King Edward Hospital mortality in the very young is very high with a CFR of 26% for infants under 8 months.36

In Letaba, Gazankulu, 41% of 109 admissions with measles (1985-1986) were below 5 years.37

In some rural areas there is a noteworthy trend for measles admissions to occur in older age groups, probably because of successful efforts to achieve adequate vaccination coverage. Crisp et al report that the average age of measles admissions in the Elim Health Ward of Gazankulu, increased from 29.7 months in 1976 to 60.9 months in 1986.38 In Venda in 1985 an epidemic of measles involved children of an age group "older than normal". In the Gelukspan Health Ward of Bophuthatswana measles is no longer a problem of under five children,39-40 recent outbreaks occurring in primary school children (Sutton C: personal communication, 1988).

**Miscellaneous**

Loenig and Coovadia41 used an urban community sample and samples of patients attending several health facilities (2 urban hospitals, 1 peri-urban hospital and 3 rural hospitals) to study incidence and mortality differentials for measles (1978 to 1981). Their most important finding was that over 25% of children in an urban environment get measles at a very young age, with the proportion decreasing as the population became more rural. It was unexpected to find the proportion of measles occurring at 8 months and under, to be higher in the urban community than in some
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Table I. Age Specific Mortality Rate per 100 000 Population (Certified Mortality, 1985)

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Asian</th>
<th>Black</th>
<th>Coloured</th>
<th>White</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-4 Years</td>
<td>7.92</td>
<td>38.03</td>
<td>28.86</td>
<td>0.00</td>
</tr>
<tr>
<td>5-9 Years</td>
<td>2.21</td>
<td>2.41</td>
<td>1.25</td>
<td>0.00</td>
</tr>
<tr>
<td>10-14 Years</td>
<td>0.00</td>
<td>1.13</td>
<td>0.28</td>
<td>0.00</td>
</tr>
<tr>
<td>15-19 Years</td>
<td>0.00</td>
<td>0.10</td>
<td>0.29</td>
<td>0.23</td>
</tr>
<tr>
<td>20 OR +</td>
<td>0.00</td>
<td>0.11</td>
<td>0.07</td>
<td>0.00</td>
</tr>
</tbody>
</table>

Table II. Percentage of Certified Measles' Deaths under 1 and 5 years of age for each population group

<table>
<thead>
<tr>
<th></th>
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<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>0-6 months</td>
<td>Africans*</td>
<td>NA</td>
<td>NA</td>
<td>41%</td>
<td>43%</td>
</tr>
<tr>
<td>of age</td>
<td>Asians</td>
<td>20%</td>
<td>35%</td>
<td>16%</td>
<td>44%</td>
</tr>
<tr>
<td></td>
<td>Coloureds</td>
<td>34%</td>
<td>35%</td>
<td>42%</td>
<td>43%</td>
</tr>
<tr>
<td></td>
<td>Whites</td>
<td>24%</td>
<td>30%</td>
<td>53%</td>
<td>30%</td>
</tr>
<tr>
<td>1-4 years</td>
<td>Africans*</td>
<td>NA</td>
<td>NA</td>
<td>95%</td>
<td>92%</td>
</tr>
<tr>
<td>of age</td>
<td>Asians</td>
<td>94%</td>
<td>95%</td>
<td>82%</td>
<td>80%</td>
</tr>
<tr>
<td></td>
<td>Coloureds</td>
<td>96%</td>
<td>96%</td>
<td>95%</td>
<td>95%</td>
</tr>
<tr>
<td></td>
<td>Whites</td>
<td>77%</td>
<td>79%</td>
<td>82%</td>
<td>78%</td>
</tr>
</tbody>
</table>

* For the African Population Data is only available from 1979 to 1986. This data is therefore reported in two 4 years groups, 1979-1982 and 1983-1986.

# NA = NOT AVAILABLE

- The significant increase in Coloured infant share of measles mortality has been accompanied by a significant decrease in mortality share of children 1 to 4 years old (1968 to 1986) (the trends of the two age groups differ significantly, p<0.001).
- The only statistically significant trend for Asians has been a decreasing...
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Interpretation of the Morbidity – Mortality Data
The importance of age at infection as a determinant of measles severity and mortality is not easy to interpret. Data from other countries suggest that CFR are higher in population groups with higher mean age at infection. On the other hand, age-specific CFR point to the fact that CFR are much higher in the younger infant. Also, as vaccination increases the mean age at infection increases and the CFR decreases.

A possible explanation for this conflicting information is as follows: in unvaccinated (or low vaccination coverage) populations, particularly in urbanised children get infected at a younger age than rural children.

Table III. Proportion of Cases and Deaths, in each population group, that are either less than 1 or less than 5 years of age.

<table>
<thead>
<tr>
<th>Age</th>
<th>Population Group</th>
<th>Morbidity*</th>
<th>Mortality*</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 1 year</td>
<td>Africans</td>
<td>23%</td>
<td>43%</td>
</tr>
<tr>
<td></td>
<td>Asians</td>
<td>21%</td>
<td>44%</td>
</tr>
<tr>
<td></td>
<td>Coloureds</td>
<td>31%</td>
<td>43%</td>
</tr>
<tr>
<td></td>
<td>Whites</td>
<td>8%</td>
<td>30%</td>
</tr>
<tr>
<td>&lt; 5 years</td>
<td>Africans</td>
<td>69%</td>
<td>92%</td>
</tr>
<tr>
<td></td>
<td>Asians</td>
<td>59%</td>
<td>80%</td>
</tr>
<tr>
<td></td>
<td>Coloureds</td>
<td>66%</td>
<td>95%</td>
</tr>
<tr>
<td></td>
<td>Whites</td>
<td>32%</td>
<td>78%</td>
</tr>
</tbody>
</table>

* Notified Data
# Certified Deaths
are infected with a lower infective dose, but still with a high CFR, although lower than that in the epidemic situation, where inter alia, multiple household cases may increase mortality. This explanation is still largely speculative.

To be able to advance a logical explanation of what is happening at community level in SA we need to:

Infant share of mortality in all non-white groups is on the increase

know the mean age at infection, the overall CFR and age specific CFR, the vaccination coverage and the endemic or epidemic nature of the disease. We are not aware of any single community in SA where all of these indices are known.

Race is also not a known determinant of measles incidence and severity. There is no reason to believe that the genetic factors associated with severity have a distribution amongst the different population groups such that they make a difference to the epidemiology of the disease. Rather, race as a proxy of socio-economic circumstance, the nature of the settlement (urban or rural), household size and, vaccination coverage could be the main factors interacting to determine age at infection.

Furthermore, the epidemiological patterns present today in less developed countries with socio-economic change and changed radically with the introduction of vaccination. Thus, racial differences in SA are taken as a reflection of different socio-economic realities. We suggest that age at infection seems to be a dubious direct determinant of severity. Examination of the measles data in relation to differentials of urbanisation supports (but does not necessarily prove), this interpretation.

Urbanisation

Urbanisation represents all the processes of social change in the urban environment. The relationship between urbanisation and measles, is not a simple one. Whites have a lower incidence of notifications and lower CFR than both Asians, who are more urbanised, and Coloureds and Blacks, who are less urbanised. Also Kettles' analysis of notifications in the Western Cape shows that the more urbanised Blacks have a higher incidence and background rate of notifications and more frequent measles peaks than the less urbanised. This work is consistent with data that showed that measles incidence was highest in urban blacks and lowest in rural Blacks. In Black urban areas the mean age at infection is lower than in Black rural residents. The limited data available also suggests that hospital case fatality rates are higher for urban Black residents.

What seems an obvious conclusion is that, in SA, urbanisation under poor socio-economic conditions is associated with a higher incidence of measles at a younger age and with a higher fatality rate. The reverse seems to apply to urbanisation under good socio-economic conditions. Therefore, the operative factor in the

<table>
<thead>
<tr>
<th>Area</th>
<th>Incidence/100,000 (year)</th>
<th>Vac Coverage (year)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nskazi</td>
<td>42.5 (1988)</td>
<td>56% (1988)</td>
</tr>
<tr>
<td>Alexandra</td>
<td>25 (1988)</td>
<td>78% (1990)</td>
</tr>
<tr>
<td>Odi</td>
<td>0.0 (1988)</td>
<td>78% (1989)</td>
</tr>
<tr>
<td>Malamulele</td>
<td>121.7 (1988)</td>
<td>82% (1987)</td>
</tr>
<tr>
<td>Gelukspan</td>
<td>1.9 (1988)</td>
<td>84% (1988)</td>
</tr>
<tr>
<td>Elim</td>
<td>25.7 (1988)</td>
<td>85% (1985)</td>
</tr>
</tbody>
</table>
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Table V. Vaccination Coverage with the third dose of DWT and with the first dose of Measles.

<table>
<thead>
<tr>
<th>Area</th>
<th>Year</th>
<th>DWT 3 % Coverage</th>
<th>Measles % Coverage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Molopo</td>
<td>1984</td>
<td>75%</td>
<td>59%</td>
</tr>
<tr>
<td>Elim (Ritavi)</td>
<td>1985</td>
<td>94%</td>
<td>85%</td>
</tr>
<tr>
<td>Malamulele</td>
<td>1987</td>
<td>83%</td>
<td>58%</td>
</tr>
<tr>
<td>Eersterus</td>
<td>1989</td>
<td>92%</td>
<td>90%</td>
</tr>
<tr>
<td>Hillbrow</td>
<td>1989</td>
<td>75%</td>
<td>70%</td>
</tr>
<tr>
<td>Laudium</td>
<td>1989</td>
<td>97%</td>
<td>87%</td>
</tr>
<tr>
<td>Odi (12/12)</td>
<td>1989</td>
<td>81%</td>
<td>78%</td>
</tr>
<tr>
<td>Pretoria</td>
<td>1989</td>
<td>95%</td>
<td>93%</td>
</tr>
</tbody>
</table>

DWT3 coverage less or equal to MEASLES coverage

<table>
<thead>
<tr>
<th>Area</th>
<th>Year</th>
<th>DWT3 % Coverage</th>
<th>Measles % Coverage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gehukspan</td>
<td>1985</td>
<td>81%</td>
<td>81%</td>
</tr>
<tr>
<td>Taung</td>
<td>1985</td>
<td>65%</td>
<td>81%</td>
</tr>
<tr>
<td>Bethesda (Ubombo)</td>
<td>1985</td>
<td>58%</td>
<td>58%</td>
</tr>
<tr>
<td>Gehukspan</td>
<td>1985/86</td>
<td>83%</td>
<td>88%</td>
</tr>
<tr>
<td>Ingwavuma (Mosvold)</td>
<td>1986</td>
<td>56%</td>
<td>56%</td>
</tr>
<tr>
<td>Alexandra</td>
<td>1988</td>
<td>66%</td>
<td>67%</td>
</tr>
<tr>
<td>Gehukspan</td>
<td>1988</td>
<td>82%</td>
<td>84%</td>
</tr>
<tr>
<td>Nsikazi</td>
<td>1988</td>
<td>50%</td>
<td>56%</td>
</tr>
<tr>
<td>Botshabelo</td>
<td>1989</td>
<td>31%</td>
<td>31%</td>
</tr>
<tr>
<td>Kayelitsha (Site C)</td>
<td>1989</td>
<td>57%</td>
<td>63%</td>
</tr>
<tr>
<td>Pratiseer</td>
<td>1989</td>
<td>46%</td>
<td>48%</td>
</tr>
</tbody>
</table>

1982-1983 measles epidemic in Port Elizabeth when 88% of notifications and 91% of deaths were in the Blacks who accounted for only 50% of the population. This was in comparison to only 1% of cases and 0.3% of deaths amongst Whites who comprised 26% of the population. The areas most affected were the most deprived socio-economically, with the most overcrowding and worst housing.28

In Cape Town the overall notification rate for 1986 was 60 per 100,000, but for Blacks it was 178. Seventy five per cent (75%) of cases admitted with complicated acute measles to the City Hospital were Blacks from the Peninsula.28

Health workers from Durban reported an average of 9.43 persons per Black household with measles, while the overall average for Blacks in SA is 5.94 and for Whites, 3.62.54 Notification data demonstrates that in the population groups with an average household size greater than 5 (Asians, Blacks and Coloureds) the risk of measles infection amongst the under ones is 3 to 6 times that of older children.24 Eighty per cent (80%) of cases of measles studied in Johannesburg-Benoni in 1988 stayed in houses with a crowding index of more than 2.5 persons per sleeping room.27

Overcrowding in poor urban areas the reason for high rates of severe measles in RSA

It is true that under poor socio-economic conditions, when measles is
more common and severe, protein-energy malnutrition is also more prevalent. As discussed before, protein-energy malnutrition is now believed to be a confounder in the model that explains the epidemiology of measles. What is apparent from the literature, and SA data is compatible with such literature, is that heavy dose of infection seems to be the main determinant of measles severity. It is also suggested that the dose of infection is most commonly related to the degree of overcrowding in the community, particularly overcrowding of children. The

overcrowding in the poor urban areas with poor housing and and ventilation would then contribute to a high dose of infection with the

Age at infection not really a direct determinant of severity

measles virus and be the reason behind high rates of severe measles in SA.

In SA the poor and urbanised blacks would be, because of overcrowding and poor ventilation, the group at higher risk for high dose of infection. This risk could be successfully counteracted by high vaccination rates but the urban poor, particularly the Black urban poor, are also a politically deprived group with neglected provision of health care.

Health Care Provision

Health care is an important modelling factor of measles morbidity and mortality. It is discussed under 3 headings: health
Preventive health care: vaccination against measles

Vaccination is essential to the control of measles. We already mentioned the falling incidence and CFR of measles in areas with successful vaccination campaigns. In SA there are vast areas in the homelands, in the “white” farm areas and in peri-urban squatter camps where health care is absent or, where preventive care is not aggressively promoted. Table IV reviews data from areas where we do have morbidity and or mortality data and data on vaccination coverage. It is apparent that, with the exception of Malamulele in Gazankulu, high vaccination coverage above 70%, is associated with lower incidence of measles.6,11-14

The data from Malamulele61 can be explained in 6 ways: it could be a reflection of the methodology used; high vaccination coverage in children between 12 and 23 months of age but much lower in older children with outbreaks of disease in older children; cold chain failure with concomitant failure to impart immunity to children; above average notification services when compared with other areas although this is unlikely; the prevalence of poor environmental and socio-economic conditions in Malamulele with extreme clustering of children not vaccinated, allowing for outbreaks of disease in the presence of high vaccination coverage; lastly, high density housing, would be associated with the occurrence of measles before they had the opportunity to be vaccinated.

Also apparent from the existing vaccination surveys are data that show that usually the coverage figure with measles vaccine is not higher than for DPT (Table V). There is therefore no operational advantage in recent recommendations to shift the age of vaccination to 6 months of age.76,79

Clinical care of the sick child with measles

The treatment of measles has been up to now of a supportive nature. In uncomplicated measles the emphasis has been on control of pyrexia, nutritional support and hygiene.80 The development of complicated measles has always been followed by treatment specific to the complications. Morley has, for more than 20 years, advocated the use of vitamin A supplementation in areas known to be deficient in this nutrient, to prevent the blinding complications of measles.81

More recently there has been tremendous interest in the use of vitamin A to reduce mortality.82,83 On the basis of a small randomized control trial,84 which did not achieve statistical significance for any of its findings, the WHO has been promoting the use of vitamin A supplementation to reduce mortality in areas with CFRs equal or in excess of 1%.8,82 This recommendation, first mentioned and ignored in 1932,85 has not been followed in SA.

A more recent randomized control trial in Cape Town found strong
supportive evidence for the routine use of vitamin A in children with measles complicated by pneumonia, diarrhoea or croup, admitted to hospital within 5 days of the onset of the rash. The Cape Town trial found twice as many deaths in the control group as in children given a high dose of vitamin A. The treatment group also had a more rapid recovery from pneumonia and diarrhoea, less croup and shorter admissions. Vitamin A supplementation in severe measles has therefore the potential to halve mortality and reduce morbidity by one third.\(^8\) The use of vitamin A in children with measles complicated by pneumonia, diarrhoea or croup, admitted to hospital within 5 days of the onset of the rash. The Cape Town trial found twice as many deaths in the control group as in children given a high dose of vitamin A. The treatment group also had a more rapid recovery from pneumonia and diarrhoea, less croup and shorter admissions. Vitamin A supplementation in severe measles has therefore the potential to halve mortality and reduce morbidity by one third.\(^8\)

What has not yet been proven, and requires urgent investigation, is the value of vitamin A supplementation to prevent severe measles, by supplementation before the onset of complications.

Bibliography
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81. WHO/UNICEF. Vit A for measles.

World Immunization News 1987; 3(4) 212.
