The impact of vitamin A supplementation on mortality inequalities among children in Nepal

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Objective: This paper examines gender, caste and economic differentials in child mortality in the context of a cluster-randomized trial of vitamin A distribution, in order to determine whether or not the intervention narrowed these differentials.

Design: The study involved secondary analysis of data from a placebo-controlled randomized field trial of vitamin A supplements. The study took place between 1989–1991 in rural Sarlahi District of Nepal, with 30 059 children age 6 to 60 months. The main outcome measures were differences in mortality between boys and girls, between highest Hindu castes and others, and between the poorest quintile and the four other quintiles.

Results: Without vitamin A, girls in rural Nepal experience 26.1 deaths per 1000, which is 8.3 deaths more than the comparison population of boys. With vitamin A the mortality disadvantage of girls is nearly completely attenuated, at only 1.41 additional deaths per 1000 relative to boys. Vitamin A supplementation also narrowed mortality differentials among Hindu castes, but did not lower the concentration of mortality across quintiles of asset ownership. The vitamin A-related attenuation in mortality disadvantage from gender and caste is statistically significant.

Conclusions: We conclude that universal supplementation with vitamin A narrowed differentials in child death across gender and caste in rural Nepal. Assuring high-coverage vitamin A distribution throughout Nepal could help reduce inequalities in child survival in this population.

Key words: health equity, vitamin A, child mortality, Nepal

Introduction

In evaluating policies to improve population health, there has been an emphasis on finding interventions that benefit the most vulnerable groups (Gwatkin 2000; Victora et al. 2003; Wagstaff et al. 2004). Although the poor are often singled out as vulnerable, other forms of disadvantage include membership in an ethnic minority, in a lower caste (Braveman and Tarimo 2002) or being female (Karki 1988). Social reforms that empower the vulnerable are obvious interventions to address health equity, and health policies that target services to the poor are seen as an effective way to carry out this agenda (Gwatkin 2000; Victora et al. 2003; Wagstaff et al. 2004). Because of the possibility that targeting the vulnerable with special services might stigmatize both the recipients and the services, supplementary strategies are needed. Primary health care alone does not appear sufficient to improve health equity. Policies that emphasize a primary health care strategy can potentially exacerbate health inequalities in the short term, because they may be differentially used more by households that are less vulnerable (Victora et al. 2000). This has led some to re-evaluate the equity impacts of strategies to distribute health interventions universally. For instance, measles vaccination appears to increase health equity in a population (Bishai et al. 2003). Can other universally applied health interventions improve health equity? In this paper we examine the impact of vitamin A supplementation on the equality of health outcomes across gender, caste and socio-economic gradients in rural Nepal.

Interventions to improve children’s intake and status of micronutrients such as vitamin A have risen in public health importance as scientific understanding of their role in child health has increased. Several intervention trials have confirmed that vitamin A distribution to high-risk populations can reduce early childhood mortality by as much as 20–30% (West et al. 1991; Ghana Vast Study Team 1993).

The vitamin A trials conducted in the 1980s and 1990s were focused on measuring the impact of vitamin A on the overall levels of mortality and morbidity (Sommer et al. 1986; Rahmathullah et al. 1991; Ghana Vast Study Team 1993). Yet in the last few years there has been increased interest in
discerning socio-economic differentials in mortality achieved by health and nutrition interventions (Singh and Yu 1996; Mackenbach et al. 1999; Wagstaff 2000). Further, a number of policy papers have suggested ways to evaluate differential mortality across socio-economic groups when assessing mortality effects of health strategies (Braveman 1998; Mills 1998; Sen 2001). Few policies have been identified that can successfully equalize mortality rates across socio-economic groups. The Whitehall study provided evidence from the United Kingdom that simply equalizing access to medical care might not be sufficient to equalize death rates (Marmot et al. 1991). Bishai and Koenig showed that in rural Matlab in Bangladesh, introduction of measles vaccination delivered door-to-door could actually minimize socio-economic differentials (Koenig et al. 2001; Bishai et al. 2003). A policy to increase access as in the Whitehall study still leaves ample room for household level decisions and inter-household inequalities to affect the distribution of services, whereas delivering services to the home (as in rural Matlab) may curtail the influence of household factors. An intervention to reduce health disparities among children would have to have strong effects on multiple causes of mortality and would have to have stronger effects among disadvantaged children than among more advantaged children (Koenig et al. 2001).

Mortality differentials by gender and socio-economic status are globally pervasive. A gender differential with females having a decisive survival advantage at every age is seen in most human populations. In contrast, South Asian populations exhibit the anomaly of having higher survival for males than females (Das Gupta 1987; Kishor 1993; World Health Organization—Southeast Asia Regional Office 2002). For example, in Nepal, life expectancy for females is 56 years and for males 57 years. The child mortality rate for girls aged 1–4 years in Nepal is 56.6 per 1000 compared with 45.5 per 1000 for boys age 1–4 (World Health Organization—Southeast Asia Regional Office 2002). Since son preference is widely cited as a major reason for these disparities (Karki 1988; Morgan 1995; Niraula and Morgan 1995; Stash 1996), much of the policy discussion has centred around efforts to change prevailing cultural attitudes toward child gender. There is evidence that son preference is played out in Nepali households through preferential food distribution that may discriminate against girls, depriving them of a nutritionally adequate diet that includes vitamin A rich foods (Gittelsohn 1991, 1997). Given that micronutrient malnutrition as documented by a low serum retinol level affects one of three Nepali children (Gorstein et al. 2003), it is plausible that a large-scale nutritional intervention could attenuate the cultural and societal influences on differential mortality.

In addition to gender disparities in survival, Nepali communities have shown survival disparities due to ethnic and socio-economic differences (Choe et al. 1989; Wagstaff 2000). The population of Nepal is approximately 90% Hindu and this major subgroup is stratified into castes and sub-castes which are closely linked to social status. The highest and second highest castes in Nepali society are Brahmin and Chettri, respectively. The populations in these two more elite castes have been shown to have better child survival than the lower caste Hindus and the non-Hindu, non-caste populations of Nepal (Choe et al. 1989). Evidence that social class and poverty are associated with vitamin A status and related risks in Nepal (Khatry et al. 1995) suggests that vitamin A administration could potentially reduce these survival disparities as well.

The objective of this paper is to determine whether an intervention that achieves a high rate of community coverage of large-dose vitamin A supplementation can reduce differentials in child survival due to gender, caste and socio-economic status. Given the established efficacy of vitamin A in preventing child death (Sommer and West 1996), this question is now best addressed within the context of existing population-based vitamin A trial data sets, such as that from a randomized, double-masked, placebo controlled trial in the southeastern plains District of Sarlahi, Nepal, that evaluated the efficacy of 4-monthly, high-potency vitamin A supplementation in reducing preschool child mortality from 1989 to 1991 (West et al. 1991).

Methods

Data

In 1989 the Nepal Nutrition Intervention Project-Sarlahi (NNIPS) of Johns Hopkins University initiated, in collaboration with the National Society for the Prevention of Blindness in Nepal, a randomized trial of vitamin A distribution, involving 30 059 children between 6 and 60 months of age in 261 wards in 29 rural village development committees in Sarlahi, Nepal (West et al. 1991). Children were randomly assigned by ward to receive either a placebo capsule with 300 μg retinol equivalents (1000 IU) or a capsule containing 60 000 μg retinol equivalents (200 000 IU) of vitamin A. Both the investigators and communities were masked to the random assignment. Baseline socio-economic status (SES) data were collected on: parents’ literacy, caste, land ownership, number of household servants, ownership of livestock, number of rooms in the dwelling, and ownership of carts, bicycles, radios and watches. Children were supplemented and vital status checked every 4 months over a 16-month period. For this analysis the outcome was death between the 1st and 4th round of the study observed for each of the 30 059 children. Data were entered onto computer in Kathmandu and the database archived and maintained at the Center for Human Nutrition, in the Department of International Health at Johns Hopkins University. The trial was reviewed and approved by the National Health Research Council in Nepal and the Joint Committee on Clinical Investigation at the Johns Hopkins School of Medicine, Baltimore, MD, USA.

Analysis

In this paper the proportion of children in the trial who died is compared across gender, between Brahmin or Chettri caste and other castes, and between children in the poorest SES quintile and those in four higher quintiles. For purposes of the intent-to-treat analysis presented here, all children living in wards which received high dose vitamin A every 4 months were considered to have been ‘treated with vitamin A’, and all children living in wards which received placebo
were considered ‘untreated’. In an intention-to-treat analysis, subjects are compared on the basis of their assigned treatment regardless of compliance (Rothman and Greenland 1998).

Although there are multiple castes in Nepali society, we chose to compare mortality rates of children in the two highest castes, Brahmin and Chettri, with those of children in all other, ‘lower’ caste and non-caste groups. SES was measured by using the first principal component that emerged from factor analysis (Filmer and Pritchett 2001) using parents’ ability to read and write a letter in any language, land ownership in katha (with each katha equal to approximately 0.1 acres), number of household servants, ownership of livestock, number of rooms in the dwelling, and ownership of bullock carts, bicycles, radios and watches. The full sample of 30,059 children was sorted into quintiles according to the derived SES variable.

We defined the following three groups of children as being at lower mortality risk: males, those of Brahmin or Chettri castes, and those of households in the upper four SES quintiles. Similarly, we defined three high-risk mortality groups of children as being female, neither Brahmin nor Chettri in caste, and those in the lowest SES quintile based, in part, on evidence from the Sarlahi District (West et al. 1991; Khatry et al. 1995). We used logistic regression to measure the odds ratio for mortality due to membership in a higher risk group (female, low caste, poorer asset quintile) with and without treatment with vitamin A relative to the corresponding best-off group defined as boys with vitamin A, Brahmin or Chettri with vitamin A, or least poor quintile with vitamin A. The basic equation we used for regression was:

\[
\text{Probability of death} = C + \beta_1 \times \text{Risk Group}
\]

Only two risk groups were considered at a time—the best-off group and the higher risk group, which was marked by a dummy variable. To correct for clustering at the level of panchayat (village) Huber-White standard errors were used in the logistic regressions.

For the analysis of SES by asset quintiles we computed the concentration index using the approximation:

\[
C = 1 + (1/5) - [(Q1 + 4 \times Q2 + 6 \times Q3 + 8 \times Q4 + 10 \times Q5)/25\mu]
\]

where \(\mu\) is the overall mean for mortality and \(Q1\), etc., is the mean for each quintile, where \(Q1\) is the poorest. If the concentration index has a value of 1.0, it indicates an outcome that is perfectly unequal across all five quintiles (e.g. positive in the wealthiest quintile and 0 elsewhere). Conversely, a value of −1.0 indicates a positive value in the poorest quintile and 0 elsewhere. If \(C\) has a value of 0 it indicates an outcome that has a balanced or equal distribution across all five quintiles. We estimated the confidence intervals around \(C\) using a bootstrap with 100 replications and sampling 20,000 observations each round.

\textbf{Sensitivity tests}

The first principal component of assets owned that was used to establish asset quintiles explained 32% of the variability in this collection of assets and registered an eigenvalue of 3.9. In addition to the original characterization of SES we assessed the robustness of our baseline SES measure by sequentially deleting groups of SES indicators from the full list used in factor analysis and repeating the computation of SES scales, SES quintiles and probit coefficients. This sensitivity analysis is available from the authors on request.

\textbf{Results}

Table 1 describes the data used in this analysis. The number of children enrolled into the trial was 30,059, with complete ascertainment of vital status for 28,868 children at the end of 16 months. The coverage was approximately 85% for each 4-monthly round of supplement distribution, and about 60% of the 15% of capsules that were left for those not at home were reported by parents to have been given to the children. Only seven parents refused to have their children receive a capsule at each round. There were 602 confirmed deaths during the trial. The sex ratio for the enrolled sample was approximately equal, with 49% girls. Household surveys early in the original trial showed that 9.1% of families self-reported to be of the Brahmin caste and 9.2% reported themselves to be of the Chettri caste. Other respondents classified themselves into the remaining ‘lower’ castes of Vayishya and Shudra or as non-participants in the caste system (e.g. Muslim or members of ethnic tribes).

Figure 1 and Table 2 compare the death rates per 1000 person years across gender, caste and economic status and by whether the child was treated with vitamin A or placebo. Mortality is higher for girls, children in non-Brahmin/Chettri castes, and

\begin{table}[h]
\centering
\begin{tabular}{l l}
\textbf{Variable} & \textbf{Frequency} \\
\hline
\textit{Children (n = 30,059)} & \\
Received Vitamin A & 51% \\
Child died & 26% \\
Female & 49% \\
Percentage neither Brahmin nor Chettri caste & 82% \\
Child’s age at enrolment (months) & 28 (SD 17) \\
\hline
\textit{Mothers (n = 29,789)} & \\
Mother is literate & 10% \\
Mother’s age (years) & 28 (SD 6.3) \\
\hline
\textit{Households (n = 29,837)} & \textit{Mean (SD)} \\
Lowland/irrigated (Khet) acres owned & 2.237 (5.034) \\
Sloped/rainfed (Bari) acres owned & 0.207 (1.044) \\
Distance to water supply (minutes walking) & 9.000 (12.308) \\
No. of household servants & 0.147 (0.557) \\
No. of rooms in dwelling & 1.872 (1.330) \\
No. of cattle owned & 2.593 (3.327) \\
No. of goats owned & 1.745 (2.897) \\
No. of carts owned & 0.168 (0.405) \\
No. of bicycles owned & 0.213 (0.442) \\
No. of radios owned & 0.237 (0.456) \\
No. of watches owned & 0.393 (0.832) \\
\end{tabular}
\end{table}
Table 2. Comparison of death rates across gender, caste and asset quintile. Odds ratio comparisons are always relative to the ‘best off’ group defined, depicted as the top row of each section: boys with vitamin A, Brahmin or Chettri with vitamin A, least poor quintile with vitamin A

<table>
<thead>
<tr>
<th></th>
<th>Deaths per 1000</th>
<th>Unadjusted odds ratio relative to ‘best off’ with vitamin A</th>
<th>95% confidence interval for odds ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Boys with vitamin A</td>
<td>16.53</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Boys without vitamin A</td>
<td>19.14</td>
<td>1.16 (0.91–1.48)</td>
<td></td>
</tr>
<tr>
<td>Girls without vitamin A</td>
<td>26.86</td>
<td>1.64 *** (1.26–2.14)</td>
<td></td>
</tr>
<tr>
<td>Girls with vitamin A</td>
<td>17.94</td>
<td>1.09 (0.82–1.44)</td>
<td></td>
</tr>
<tr>
<td>Caste is Brahmin or Chettri</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>with vitamin A</td>
<td>11.47</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Caste is Brahmin or Chettri</td>
<td>11.23</td>
<td>0.98 (0.60–1.59)</td>
<td></td>
</tr>
<tr>
<td>without vitamin A</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lower/other caste without vitamin A</td>
<td>25.28</td>
<td>2.24 *** (1.74–2.88)</td>
<td></td>
</tr>
<tr>
<td>Lower/other caste with vitamin A</td>
<td>18.60</td>
<td>1.63 *** (1.27–2.10)</td>
<td></td>
</tr>
<tr>
<td>5th (Highest) quintile with vitamin A</td>
<td>9.88</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>5th (Highest) quintile without vitamin A</td>
<td>12.28</td>
<td>1.25 (0.55–1.94)</td>
<td></td>
</tr>
<tr>
<td>4th quintile with vitamin A</td>
<td>12.51</td>
<td>1.27 (0.69–1.85)</td>
<td></td>
</tr>
<tr>
<td>4th quintile without vitamin A</td>
<td>13.59</td>
<td>1.38 (0.75–2.01)</td>
<td></td>
</tr>
<tr>
<td>3rd quintile with vitamin A</td>
<td>14.01</td>
<td>1.42 (0.82–2.02)</td>
<td></td>
</tr>
<tr>
<td>3rd quintile without vitamin A</td>
<td>24.10</td>
<td>2.47 *** (1.43–3.52)</td>
<td></td>
</tr>
<tr>
<td>2nd quintile with vitamin A</td>
<td>19.26</td>
<td>1.97 (0.93–3.00)</td>
<td></td>
</tr>
<tr>
<td>2nd quintile without vitamin A</td>
<td>29.34</td>
<td>3.03 (0.93–4.00)</td>
<td></td>
</tr>
<tr>
<td>1st (poorest) quintile with vitamin A</td>
<td>28.74</td>
<td>2.97 *** (1.78–4.15)</td>
<td></td>
</tr>
<tr>
<td>1st (poorest) quintile without vitamin A</td>
<td>33.12</td>
<td>3.43 *** (2.09–4.78)</td>
<td></td>
</tr>
</tbody>
</table>

*** p < 0.01. Robust standard errors adjusted for clustering at Panchayat (village) level.

Figure 1. Panel (a) compares child deaths by gender in the placebo and vitamin A arms of the trial. Panel (b) compares child deaths by caste. Panel (c) compares child deaths by economic status.
for the poorest households. Mortality is higher in the group assigned to receive placebo. In scanning Figure 1, the reader can assess qualitatively that the height of the bars is more disparate in the placebo treated population than in the vitamin A treated population, reflecting greater equality of mortality outcomes in populations where vitamin A adequacy has been experimentally secured.

Table 2 quantifies the degree to which mortality has been equalized by comparing death rates in both placebo and vitamin A treated females, low caste and poorest children with males, high caste and less poor children. The equalization of outcomes is most striking for girls. Without vitamin A, girls in rural Nepal experience 26.86 deaths per 1000, which is 8.33 deaths more than the comparison population of boys. With vitamin A the mortality disadvantage of girls is attenuated by nearly 90%, to 1.41 additional deaths (calculated as 17.94 minus 16.53) relative to boys. The odds ratios in Table 2 show that the girls disadvantage in mortality is both small (1.09) and no longer significant in a population receiving vitamin A supplementation. Girls without vitamin A show a statistically significant odds ratio of 1.64 relative to boys with vitamin A, and (not shown in table) girls without vitamin A have an odds ratio of mortality of 1.41 (95% CI: 1.09–1.83) relative to the more comparable boys without vitamin A.

Similarly, for lower caste children the mortality excess of 14.05 (25.28 minus 11.23) per 1000 without vitamin A is attenuated to total only 7.13 excess deaths (calculated as 18.60 minus 11.47). This attenuation of caste mortality differential observed with vitamin A is also reflected by the lower odds ratio of 1.63 for vitamin A treated lower and other caste children vs. 2.24 for untreated lower and other caste children. The difference between 1.63 and 2.24 is not statistically significant.

In both vitamin A and placebo groups, children exhibit a gradient in risk of mortality by quintile of socio-economic status (Table 2). In fact, Figure 2 shows that the greatest advantages of vitamin A accrue to the second and third lowest SES quintile groups. Although mortality is lower for vitamin A treated children than for the untreated within each asset quintile, neither the poorest nor the least poor quintiles experience particularly strong effects of vitamin A. Because a vitamin A treated population (solid bars in Figure 2) experiences most of the mortality improvement in the middle of the SES distribution rather than at the tails, the overall distribution of mortality becomes more concentrated at the tails of the distribution and, by a concentration index measure, more unequal. The asset quintile based concentration index for vitamin A treated population is 0.42 (95% CI: 0.23–0.55), while for the untreated population it is 0.15 (95% CI: 0.007–0.29), indicating that health disparities by asset quintile were wider in the vitamin A treated population than in the placebo population.

Our results are robust to alternative constructions of the SES measures. We computed the SES measure six different ways and compared the resulting mortality differentials and concentration indices. The five alternative measures were as follows: (1) Baseline list without parents’ education; (2) Baseline list without land, servants, number of rooms; (3) Baseline list without livestock; (4) Baseline list without transportation assets; and (5) Baseline lists without bicycles and radios. The results showed a significantly higher concentration index of mortality for the vitamin A treated population with each of the alternative specifications of SES. The full results of sensitivity tests are available from the authors on request.

**Discussion**

The evidence we have presented shows equalization of death rates between girls and boys in the villages where high-potency vitamin A was distributed on a 4-monthly basis. Public health interventions that rely on door-to-door distribution of health services essentially overcome intra-household gender biases that could otherwise be prejudiced against girls receiving such services outside the home (Gittelsohn 1991). Vitamin A prophylaxis also appeared to reduce caste-related differences in child death rates. It is interesting that vitamin A reduced differentials on the basis of caste, but not on the basis...
of asset ownership. This suggests that childhood dietary vitamin A intake (or the risk of vitamin A responsive disease) in the population was less similar across the lines of caste (more sufficient in Brahmin, Chettri) and more similar across the gradient of asset ownership.

Alternative systems for distributing health care services that rely on household members to travel to a clinic or distribution centre for services have been suspected of worsening socioeconomic differentials in health outcomes (Victora et al. 2000). Even services based in community facilities may still be differentially used by those with higher wealth and privilege. The levelling of social differences in mortality achieved with vitamin A distribution in Nepal is similar to effects achieved with measles vaccine in Bangladesh (Bishai et al. 2003). The door-to-door distribution method used during the vitamin A intervention has also been shown to level social differences in the distribution of vaccines (Bishai et al. 2002).

It is surprising that vitamin A appeared to be most effective in the middle of the income distribution. While it is easy to understand why higher SES children with possibly less exposure to infectious diseases and higher baseline nutritional status may derive less benefit from vitamin A than poorer, less well nourished children, it is unclear why the lowest SES quintile has a reduced response to vitamin A compared with children in the middle of the SES distribution. The fraction of boys in the population in quintiles 1 (poorest) to 5 (highest) was 0.50, 0.53, 0.52, 0.51, 0.52, respectively, which is not sufficient to explain the pattern on the basis of the higher vitamin A responsiveness of girls. We also examined the possibility that compliance with vitamin A might have been lower in the lowest SES households. We found that 90% of children in the poorest quintile households received the supplement in the direct presence of the health worker, identical to the direct compliance of the 2nd and 3rd quintiles. We speculate that the overall mortality hazards of children in the lowest SES quintile include several causes of death that may not be as responsive to vitamin A as diseases among children in the middle of the SES distribution. Such causes may be related to protein-energy malnutrition, pneumonia and injury. In addition, there may be antagonistic factors among the very poorest children that preclude them from deriving benefit from vitamin A.

Conclusion

Door-to-door vitamin A distribution as it occurred under field trial conditions made the survival of preschool-aged girls nearly equal to that of boys, unlike many other conditions in Nepali village life, and narrowed differentials based on caste. This effect occurred in the absence of any specific social movement to change cultural preference for sons or to reduce caste-based discrimination.

Although some health systems distribute vitamin A as part of primary health care, the approach in Nepal has relied on a system of free door-to-door distribution using a cadre of 40 000 auxiliary staff known as ‘Female Community Health Volunteers’ (FCHVs) (Fiedler 2000). In practice, the FCHVs have been quite successful in achieving extremely high coverage rates of vitamin A. The door-to-door distribution method brings the price of vitamin A faced by parents effectively to zero and minimizes economic barriers to the health intervention. The Nepal National Vitamin A Program now has FCHVs operating in every district in the country. Consequently, as vitamin A coverage of children in Nepal reaches a maximum, we predict that Nepal’s gender and caste differentials in child survival will continue to narrow throughout the country. Compared with the 1.41 to 1 mortality rate ratio for girls among the placebo group in our study (Figure 1a), the latest data from Nepal’s Demographic and Health Survey from 2001 show a rate ratio of 1.07 in under-5 mortality for girls (ORC Macro 2004). Based on our analysis, it is reasonable to attribute a substantial proportion of the improvement in gender differentials in mortality for children in Nepal to vitamin A supplementation.

What this study adds

Numerous studies have shown that nutritional interventions to provide vitamin A supplements to children in malnourished populations can have a substantial impact on the overall level of child mortality. Until now there has been no attempt to quantify the ability of vitamin A supplementation to narrow gradients in mortality across gender, ethnic groups and socioeconomic status.

This study reviews data collected during a randomized vitamin A supplementation trial in rural Nepal in 1989–1991. It shows that girls who received placebo had mortality rates that were 7 deaths per 1000 higher than boys who received placebo. In contrast, girls who received vitamin A had mortality rates that were only 1.5 deaths per 1000 higher than boys who received vitamin A. Vitamin A also narrowed mortality gradients between Hindu castes, but not across different quintiles of asset ownership. What we now know as a result of this study is that community-wide treatment with vitamin A supplements was able to narrow gender- and caste-related differentials in child survival.

References


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