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Interventions for the control of diarrhoeal diseases among young children: prevention of low birth weight*

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The effect of low birth weight (LBW) on diarrhoea morbidity and mortality is analysed and interventions to increase birth weights are reviewed. Birth weight is a major determinant of infant mortality and, in developed countries at least, its effect on neonatal mortality is independent of socioeconomic status. We have located no satisfactory data on LBW as a determinant of diarrhoea mortality or morbidity. The strong association between LBW and mortality, however, makes it likely that there is an association between LBW and diarrhoea mortality in developing countries where diarrhoea is a major cause of infant death. Poor maternal nutrition, certain infections, pre-eclampsia, arduous work after mid-pregnancy, short birth intervals, and teenage pregnancy are likely to be causally associated with LBW in developing countries. Tobacco and alcohol consumption are additional risk factors.

Of the interventions examined, maternal food supplementation has been the most studied. If targeted to mothers at nutritional risk, and if the food is consumed in addition to the usual diet, the prevalence of LBW can be expected to be reduced. However, food supplementation can be expensive and the results from carefully supervised feeding trials may be better than those that can be achieved in national programmes. The effect of supplementation with iron, zinc or folate requires further study. If it were possible to intervene in maternal nutrition, health and life-style in a developing country in a way that reduced the prevalence of LBW from around 30% to around 15%, a fall in the infant mortality rate of around 26% would be expected. The fall in infant diarrhoea mortality rate might be similar. The scarce data on relative risk of morbidity by birth weight do not allow any comparable computations for morbidity reductions to be made.

This review confirms that whatever its association with diarrhoea, LBW is an important determinant of infant mortality. For the more general goal of reducing infant mortality it is necessary to know more about the nature, etiology, and prevention of LBW in developing countries.

In this review, the effect of low birth weight on diarrhoea morbidity and mortality is analysed and interventions to increase birth weight are reviewed. While recognizing that the etiology of low birth weight is multifactorial, emphasis is given to those maternal factors that are believed to be of greatest importance in developing countries and that may be amenable to change in the short term. These include inadequate dietary intake, infections, arduous workloads, teenage pregnancy, short birth intervals, and excessive tobacco or alcohol consumption. This paper examines whether interventions that reduce the prevalence of low birth weight might be effective in reducing morbidity or mortality from diarrhoeal diseases among young children. This review is the fifth in a series of reviews of potential anti-diarrhoea interventions being published in the *Bulletin of the World Health Organization* (*33–37*).

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EFFECTIVENESS

If the prevention of low birth weight, by improving the nutritional status, health or life-style of pregnant women, is to be an effective diarrhoea control

Table 1. Percentage distribution of live births by birth weight in four countries

	Percentage of live births														
												India			Guatemala
Birth weight (g)	Oregon (Portland)	USA (5 regions)	USA (white)	California	USA	New York (white)	New York	USA (non-white)	Brazil (Recife)	Colorado (Denver)	New York (black)	New Delhi	North Arcot	Palghar	(Santa Maria Cauqué)
< 1501	0.8	0.8	1.0	1.0	1.2	0.9	1.6	2.2	1.7	3.9	3.0	2.8	1.1	5.2	1.2
1501-2000	0.9	1.1	1.3	1.5	1.4	1.3	1.8	2.5	2.8	3.4	3.0		5.5	9.4	6.5
2001-2500	3.5	4.2	4.5	5.1	5.1	5.7	6.6	8.3	10.0	8.0	9.8	20.2	25.3	23.2	34.0
2501-3000	15.6	17.8	17.2	19.1	18.5	22.7	23.9	25.3	33.0	24.9	28.4	45.8	41.2	37.4	48.1
3001-3500	38.1	39.2	38.1	39.5	38.0	40.6	39.6	37.1	35.0	36.9	36.6	25.8	21.5	22.4	10.0
3501-4000	30.4	27.7	28.2	26.0	26.8	22.6	21.0	18.9	14.8	18.4	15.8	4.8	4.8	2.4	0.2
> 4000	10.6	9.1	9.6	7.8	9.0	6.2	5.5	5.8	2.6	4.5	3.5	0.6	0.7	0.0	
% LBW	5.2	6.1	6.8	7.6	7.8	8.0	10.0	12.9	14.5	15.2	15.7	23.0	31.9	37.8	41.7
Reference	*10*	*110*	*20*	*91*	*20*	*21*	*21*	*20*	*82*	*55*	*21*	*91*	*93*	*103*	*70*

intervention, it must be true that:

either

a considerable proportion of diarrhoea morbidity or mortality in young children in developing countries is due to low birth weight	*hypothesis 1*

and

improving the nutritional status, health or life-style of pregnant women can reduce the prevalence of low birth weight	*hypothesis 2*

or

improving the nutritional status, health or life-style of pregnant women can reduce diarrhoea morbidity or mortality rates in young children	*hypothesis 3*

The literature on hypothesis 2 is extensive, whereas hypotheses 1 and 3 have been little studied. The effectiveness of low-birth-weight prevention as an intervention to reduce diarrhoea morbidity or mortality would be suggested by a demonstration either of the correctness of hypotheses 1 and 2 or of the correctness of hypothesis 3. The evidence for and against these hypotheses is examined below.

Hypothesis 1. *A considerable proportion of diarrhoea morbidity or mortality in young children in developing countries is due to low birth weight.*

Prevalence and distribution of low birth weight. The term low birth weight (LBW) is used to describe infants who weigh less than 2500 g at birth. From sample surveys and country reports, WHO has estimated that over 20 million LBW infants are born each year. At the global level this represents 16% of all births, but the proportion is not uniform and national rates range from 4% in Scandinavia to around 50% in parts of India and Bangladesh (*124*, *125*). Table 1 compares birth weight distributions in 15 populations with different LBW prevalence rates, ranging from 5% to 42%.

Infants with a low birth weight may be divided into two broad subgroups: (*a*) those who are born preterm, that is of less than 37 weeks' gestation; (*b*) those who are growth-retarded *in utero* and are born small for gestational age (SGA). Investigators are not consistent in their definition of SGA, but it may be defined as a birth weight of 2SD or more below the mean birth weight for gestational age. Although most preterm infants are appropriate for gestational age (AGA), some LBW infants are both preterm and SGA. In the developed countries the majority of LBW infants are the result of a preterm delivery. In contrast, in developing countries it would seem from the limited information available that the majority of LBW infants are small for gestational age. For example, in 18 reports from the Indian subcontinent, south-east Asia and Latin America, between 65% and 96% of LBW infants were small for gestational age (*124*). In Africa, there are data for only 5 cities and these show a more diverse pattern with between 34%

Table 2. Association of low birth weight and infant mortality in developing countries

Region or country	Place	Prevalence of LBW (%)	Percentage of deaths weighing < 2500 g at birth			Reference
			Neonatal	Post-neonatal	Infant	
Latin America and Caribbean		—[a]	—	—	47	*89*
Nigeria	Igbo-Ora	11	—	—	42	*4*
India	Hyderabad	21	84	—	—	*80*
India	North Arcot	32	56	42	48	*93*
Guatemala	Santa Maria Cauqué	42	87	58	70	*69*

[a] — denotes data not available.

Table 3. Relative risks of neonatal mortality by birth weight compared to birth weight of 2501-3000 g

	Relative risk of neonatal mortality												
Birth weight (g)	California (1977)	USA (5 regions (1974-75)	Norway (1967-78)	California (1969-70)	Oregon (Portland) (1959-66)	USA (white) (1960)	Colorado (Denver) (1974-80)	USA (1960)	India (Delhi) (1969-72)	USA (non-white) (1960)	Brazil (Ribeirao Preto) (1968-70)	India (N. Arcot) (1969-75)	Guatemala (Santa Maria Cauqué) (1964-72)
1001-1500	52.7	99.9[a]	41.1	80.5	47.5	55.0	32.8	52.6	94.2	46.2	41.4	16.7[a]	
1501-2000	13.5	17.6	16.6	19.8	20.6	19.6	9.4	18.2	30.8	13.9	23.3	4.4	27.3
2001-2500	3.0	3.8	4.5	5.2	4.4	4.4	2.1	4.2	4.5	3.3	4.0	1.4	3.4
2501-3000	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
3001-3500	0.3	0.5	0.4	0.5	0.4	0.4	0.3	0.5		0.7	0.5	0.8	
3501-4000	0.3	0.3	0.2	0.4	0.3	0.3	0.3	0.4	1.1	0.7	0.4	0.6	
4001-4500	0.3	0.4	0.2	0.6	0.2	0.4		0.4		1.1	0.5	1.4	
> 4500	0.4		0.3		0.3	0.8		0.9		1.7			
Neonatal mortality rate[b]	5.6	8.2	8.5	12.7	13.8	16.9	17.5	18.4	21.2	26.7	28.2	34.8	39.0
% LBW	unstated	6.1	5.2	unstated	5.2	6.8	15.2	7.8	23.2	12.9	8.7	31.9	41.7
No. of births	290 000	234 000	700 000	44 700	40 000	3.6 million	14 400	4.26 million	4 590	657 100	18 200	4 220	416
Reference	*123*	*110*	*30*	*90*	*10*	*20*	*55*	*20*	*41*	*20*	*90*	*93*	*69*

[a] Includes all births < 1500 g.

[b] Deaths per 1000 live births, except for ref. *123* (per 1000 single vaginal births), ref. *30* (per 1000 total births), and ref. *10* (per 1000 single, white, live births).

Table 4. Relative risks of post-neonatal mortality by birth weight compared to birth weight of 2501-3000 g

Birth weight (g)	Relative risk of post-neonatal mortality							
	Norway (1967-68)	USA (5 regions) (1974-75)	USA (white) (1960)	USA (1960)	USA (non-white) (1960)	India (Delhi) (1969-72)	India (N. Arcot) (1969-75)	Guatemala (Santa Maria Cauqué) (1964-72)
1001-1500	2.6	12.2[a]	6.3	6.1	4.8	9.3	4.5[a]	
1501-2000	3.0	3.7	3.5	3.4	3.0	5.3	2.3	7.0
2001-2500	1.8	1.9	1.9	1.9	1.8	2.2	1.1	0.8
2501-3000	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
3001-3500	0.6	0.6	0.6	0.6	0.7		0.6	0.5
3501-4000	0.5	0.5	0.5	0.5	0.7	0.7	0.4	
4001-4500	0.4	0.5	0.5	0.4	0.7			
> 4500	0.5		0.5	0.5	0.7			
Post-neonatal mortality rate[b]	3.2	4.3	5.4	6.9	15.1	25.3	51.6	60.0
% LBW	5.2	6.1	6.8	7.8	12.9	23.2	31.9	41.7
No. of births	700 000	234 000	3.6 million	4.2 million	657 100	4 590	4 220	416
Reference	*30*	*110*	*20*	*20*	*20*	*41*	*93*	*69*

[a] Includes all births < 1500 g.

[b] Deaths per 1000 survivors, except for ref. *30* (per 1000 total births) and ref. *110* (per 1000 live births).

and 73% of LBW infants being small for gestational age. More research is required to determine the relative proportion of these two subgroups of LBW infants in developing countries.

Association of low birth weight with mortality. In developed countries, LBW infants comprise the majority of infant deaths. For example, in a recent report from the USA, although LBW infants represented only 6% of live births they comprised 55% of infant deaths (*110*). Data from developing countries are limited since death registration is frequently incomplete and birth weights are rarely recorded. Indications are, however, that LBW infants similarly comprise a large proportion of infant deaths as shown in Table 2.

Birth weight has a marked association with neonatal mortality rates in both developed and developing countries. The relative risks of neonatal death among infants of different birth weights, compared with infants weighing 2501-3000 g, are shown in Table 3. The most favourable birth weights were in the range of 3500-4500 g. Although there were 7-fold differences in overall neonatal mortality rates among the 13 studies, the relative risks in each 500-g birth-weight interval were similar, except for Denver and North Arcot, where infants with birth weights of < 2500 g had lower relative risks than in the other places studied.

The association between birth weight and mortality extends into the post-neonatal period and birth weights of 3500-4500 g were similarly associated with the lowest risk. Table 4 shows that among LBW infants the relative risk of post-neonatal death in North Arcot was somewhat lower than that observed in developed countries, whereas in Delhi the relative risk among LBW infants was higher than in developed countries. In Santa Maria Cauqué, the relative risks were somewhat anomalous, perhaps because of the small number of subjects in this study.

It is possible that there is also an association between birth weight and mortality in the 1-4-year age group. In the Gambia, child mortality is significantly higher in children born in the wet season (when the incidence of LBW is high) than in children born in the dry season (*85*), and there is some indication from Guatemala that SGA infants may have an increased 1-4-year mortality risk (*68*, *71*). In view of the limited data available, however, it has been conservatively assumed in the calculations which follow that low birth weight confers no increased risk of death beyond the first year of life.

In populations where the whole Gaussian distribution of birth weights is shifted to the left, leading to a lower mean birth weight, it is possible that the definition of LBW based on a cut-off point of 2500 g is

Table 5. Neonatal and post-neonatal mortality rates by birth weight and gestational age

Birth weight (g)	Gestational age	Mortality rate			
		Santa Maria Cauqué	Delhi	North Arcot	New York City
		Neonatal mortality rate (deaths per 1000 live births)			
< 2500	Preterm	323	87	73	93
	Term (SGA)	28	27	41	44
> 2500	Preterm	—[a]	10	30	—[b]
	Term	8	4	21	—[b]
		Post-neonatal mortality rate (deaths per 1000 survivors)			
< 2500	Preterm	286	46	66	16
	Term (SGA)	58	35	66	13
> 2500	Preterm	—[a]	18	50	—[b]
	Term	42	13	42	—[b]
Reference		*69*	*41*	*93*	*117*

[a] No infants in this category.
[b] No data available.

too rigid (*95*). In such populations the relative risk of neonatal or post-neonatal death among births of < 2500 g may be lower than in populations with higher mean birth weights. There is evidence for this from the USA in populations living at high altitude (e.g., Colorado, Tables 1 and 3) and from the United Kingdom in populations differing in racial origin (*27*). At present, however, there are insufficient birth-weight-specific mortality data to determine whether, or to what extent, the relative risk of neonatal and post-neonatal death among LBW infants in developing countries differs from that in developed countries.

Many of the factors affecting mortality are interrelated. Few studies have attempted to determine the effect of one factor while controlling for the interrelated factors. In Baltimore, during 1960–64, a cohort of 108 852 single live births of known birth weight were followed for one year after birth. When the effects of birth weight, race, socioeconomic status, maternal age, birth order, and prenatal care were separated, birth weight was the most important factor in neonatal mortality, and factors such as race, socioeconomic status and maternal age were important only because they were related to birth weight (*102*). Likewise birth weight was the most important factor in post-neonatal mortality. However, in this period the other factors (except for race) were also important. In New York City during 1976–78, socioeconomic circumstances were also found to have little effect on the probability of neonatal death once birth weight was controlled (*83*). No comparable analyses from developing countries have been located.

Mortality among preterm versus SGA infants. Although the data are limited, it would appear that in both developed and developing countries preterm infants have a considerably higher risk of neonatal death than term SGA infants of similar birth weight (*9*, *110* and Table 5). The reason for this is the greater difficulty experienced by preterm infants in adapting to the extrauterine environment owing to immaturity of many of the body's systems (*117*).

In the post-neonatal period, preterm infants may also experience higher mortality rates than term SGA infants (Table 5), but the difference is less marked and less consistent than in the neonatal period.

The data on childhood mortality are very limited. However, in New York, term SGA infants appeared to be at slightly greater risk, their 12–23-month mortality rate being 2.7 per 1000 compared with 2.3 for preterm infants (*117*). In Santa Maria Cauqué, term SGA infants had a higher mortality rate during the second, third, and fourth years of life than preterm infants (*68*).

Studies in the USA and United Kingdom have shown that term SGA infants grow more slowly than

preterm infants (*25, 32, 81, 117*), the difference in their mean weight being 0.7 kg at 12 months (*25, 117*) and 5 kg at 10 years of age (*32*). Slower growth in SGA infants compared with preterm infants has also been reported from New Delhi (*100*). In conclusion, it would seem that preterm infants are at greater risk of death, especially neonatal death, in the first year of life than term SGA infants, but that preterm infants who survive may grow better than SGA infants and have lower mortality rates after 1 year of age.

Association of low birth weight with infant morbidity. At present only limited data are available, although several long-term prospective studies of preterm infants are in progress in developed countries. In Oakland, California, LBW infants did not experience significantly more episodes of acute diseases in the first two years of life than infants of higher birth weight (*116*).

Association of low birth weight with diarrhoea morbidity or mortality. The association, if any, between LBW and diarrhoea morbidity or mortality is poorly documented. In the Inter-American Investigation of Mortality in Childhood (*89*), LBW was examined as an associated cause of diarrhoea mortality, but only in relation to the neonatal period. Of 1269 neonatal diarrhoea deaths recorded in 8 countries, LBW was a contributory cause in 49%, with a range from 28% in Bolivia to 80% in Jamaica. In a prospective study from 1964 to 1972 in the Guatemalan village of Santa Maria Cauqué, LBW infants showed greater rates of diarrhoeal disease and greater prevalence rates of infection with *Shigella*, *Entamoeba histolytica* and *Giardia* in the first six months of life than infants of higher birth weight (*68, 69*). These findings are not reported in a way that allows diarrhoea incidence by birth weight to be calculated.

Since epidemiological data are limited, an alternative approach is to examine physiological function. It is known that the immune response of LBW infants is severely compromised (*19, 38*) and is more adversely impaired than that of postnatally malnourished infants (*38*). In addition to defects in cellular immunity, LBW infants have been found to have a significant reduction in maternal–fetal transfer of IgG (*19, 100*) and impaired synthesis of IgA, IgM, and the C_3 component of complement (*100*). In a follow-up study of 30 LBW infants in New Delhi, impaired immunity was associated with recurrent attacks of diarrhoea in the first 6 months of life. The impairment was most marked in SGA infants (*100*). It may be tentatively concluded from these limited data that LBW infants, especially if they are small for gestational age, may be predisposed to increased diarrhoea morbidity rates. Furthermore, since malnourished infants are more likely than well nourished infants to die from diarrhoea (*33*), it is reasonable to expect that LBW infants, especially if they are SGA, will experience increased diarrhoea mortality rates. Further research is warranted to confirm these tentative conclusions.

Evidence suggests that breast-feeding protects young infants from diarrhoea (*36*). In some countries, especially where babies are born in hospital, a greater proportion of LBW infants may be fed artificially compared with infants of heavier birth weight. Feeding mode may therefore also contribute to an increased risk of diarrhoea among LBW infants.

Conclusions on hypothesis 1. Birth weight is a major determinant of infant mortality and, in developed countries at least, the effect of birth weight on neonatal mortality is independent of socioeconomic status. Where information is available, and this shows considerable local and regional variations, the majority of LBW infants in developed countries are preterm, whereas in developing countries the majority are small for gestational age. Preterm AGA infants suffer higher infant mortality rates than SGA infants, and this difference is especially marked in the neonatal period. On the other hand, preterm AGA infants may grow better post-neonatally than SGA infants.

We have located no satisfactory data on LBW as a determinant of diarrhoea mortality or morbidity. The strong association between LBW and mortality (Tables 3 and 4) makes it likely that there is an association between LBW and diarrhoea mortality in developing countries where diarrhoea is a major cause of infant death. Immunity is severely impaired in LBW infants, especially SGA infants. Data from India and Guatemala indicate that SGA infants may be predisposed to increased diarrhoea morbidity.

Hypothesis 2. *Improving the nutritional status, health or life-style of pregnant women can reduce the prevalence of low birth weight.*

Etiology of low birth weight. Low birth weight is caused by factors that shorten the length of gestation and/or impair fetal growth. Factors affecting the latter are more pertinent to this review since SGA infants probably comprise the majority of low-weight births in developing countries. Some factors, for example altitude, are not amenable to change. Other factors including socioeconomic status, multiple pregnancy, maternal height, and pregravid weight can only be influenced by long-term interventions. In this review, only factors that are more readily amenable to change and might respond to short-term

interventions in developing countries are examined. The factors selected are poor nutritional status, physically demanding work, maternal health, maternal age, birth interval, and tobacco and alcohol consumption. Only the first of these is discussed in any detail.

Nutritional status. There is ample evidence that an inadequate dietary intake during pregnancy adversely affects birth weight. In the conditions of severe food shortage that occurred in parts of Europe during the Second World War, average birth weights fell by 185 g to 600 g (*3*, *28*, *105*).

In developing countries with seasonal food shortages, considerable fluctuations in birth weight occur. In the village of Keneba in the Gambia, for example, the mean monthly weight gains of pregnant women were 0.4 kg in the wet season (July–October) and 1.4 kg in the dry season, and the corresponding prevalences of LBW were 35% and 13% (*86*). In July the average birth weight was 480 g less than in May (*94*). However, the rainy season is not simply a time of food shortage, it is also a period of increased prevalence of maternal infection, including malaria, and a period of increased agricultural activity. The seasonal decrease in birth weight is thus likely to be the result of several adverse factors which together affect both maternal nutritional status and health.

A major focus of maternal and child health programmes has been the provision of additional food to pregnant women. Unfortunately, few such interventions have been evaluated. One exception is the nationwide special supplemental food programme for low-income American women, infants and children (the WIC programme), which encourages clinic attendance and provides individual nutrition counselling in addition to the WIC foods (milk or cheese, eggs, iron-fortified cereal, and fruit juices) through the Food and Nutrition Service of the US Department of Agriculture. This programme has been associated with an increased weight gain in pregnancy, an increase in birth weight (+136 g, if supplemented for more than 6 months), and a decrease in the prevalence of LBW from 10% to 6% (*11*, *29*, *54*). Of the services provided, only food supplementation had a significant effect on birth weight (*54*).

Several small-scale feeding trials have been evaluated and the results of nine such studies are summarized in Table 6. In four of the studies the observed improvement in birth weight was statistically significant although not always large (Mexico, +213 g; Keneba, +120 g; Guatemala, +117 g; and Montreal, +40 g). In the three developing countries where the prevalence of LBW was >15% (the Gambia, Guatemala, and Mexico), supplementation led to a statistically significant reduction in the proportion of low-weight births.[a] In China (Province of Taiwan), the existing prevalence of LBW was low and pre-supplementation birth weights were relatively good, especially if one takes into account maternal short stature (*65*). The participants in this study may therefore not have been at much nutritional risk. It may be concluded that, where maternal nutritional status is poor, food supplementation that effectively increases net intakes can improve birth weights and reduce the prevalence of LBW.

In Harlem (New York City) a decrease in mean birth weight was observed with a high-protein, high-mineral supplement (*99*). Since similar findings have been reported from San Francisco with the same supplement (*2*) and from Motherwell (Scotland) when pregnant women were advised to eat a predominantly protein diet (*44*), caution has been expressed about possible deleterious effects of excessively high protein intakes in pregnancy. The apparent decrease in mean birth weight with a protein-free supplement in Birmingham (England) was probably due to imperfect matching of the groups (*119*).

The provision of a food supplement, however, does not necessarily lead to its consumption. In some societies pregnant women may purposely restrict their intake in anticipation of an easier delivery, and thus traditional dietary customs may limit any effort to improve maternal nutrition, whether by dietary advice or by direct food supplementation. For example, in Project Poshak in India, food collection rates were poor in pregnancy and 90% of the ration which was collected was subsequently dispersed among other family members (*43*). In Colombia where food supplements were provided for each family member, pregnant women consumed only 57% of their allocation. Moreover, the supplement replaced some of their regular diet so that despite very generous rations there was a relatively small increment in net intake (*50*). In contrast, in the Gambia a high uptake of the supplement was achieved which was attributed to its palatability and to the fact that it was offered in the early morning when the women would not normally have eaten at home (*88*). Its high-energy density assisted in achieving a substantial net increase in energy intake.

The magnitude of the response in birth weight to supplementation may appear disappointingly low. However, where the data have been disaggregated, the response among inadequately nourished mothers is substantial. For example, supplementation increased the mean birth weight during the wet season in the Gambia by 225 g and decreased the prevalence of LBW from 28% to 5% (*88*). In Colombia,

[a] Similar results have also been reported from India, where supplementation in the third trimester was associated with a significant increase in birth weight (+170 g) and a 48% reduction in the prevalence of LBW (*129*).

Table 6. Effect of food supplementation on mean birth weight and prevalence of LBW

Country	Place	Habitual daily intake		Daily allocation of supplement		Net increase in energy intake (kcal/day)	Duration of supplementation (weeks)	Increase in mean birth weight (g)	Reduction in LBW births (%)	Dose response g/10 000 kcal or 41.84 MJ[e]	Reference
		kcal	protein (g)	kcal	protein (g)						
Canada	Montreal	2250 (9.41)[a]	68	individualized		—[f]	self-selected	40[b]	16	—[f]	*97*
Colombia	Bogota	1610 (6.74)	35	860 (3.60)[a]	38	155 (0.65)[a]	13	51	21	41	*50, 76*
Gambia	Keneba	1470 (6.15)	—[f]	1000 (4.18)	37	430 (1.80)	24	120[b]	68[c]	17	*86–88*
Guatemala	4 villages	1500 (6.28)	40	self-selected		149 (0.62)	self-selected	117[b]	41[d]	29	*46, 58, 60, 61*
Mexico	Rural areas	1950 (8.16)	50	300 (1.26)	20	275 (1.15)	34	213[b]	80[c]	28	*22, 23*
China (Province of Taiwan)	Sui-Lin	—	—	800 (3.35)	40	—	> 40	16	48	—	*12, 65*
United Kingdom	Aberdeen	2060 (8.62)	70	290 (1.21)	15	189 (0.79)	12	37	—	—	*17*
	Birmingham	—	—	425 (1.78)	0	—	10	−120	—	—	*119*
				425 (1.78)	11	—	10	330	—	—	
USA	Harlem	2060 (8.62)	80	320 (1.34)	6	207 (0.87)	> 10	41	28	—	*98, 99*
				470 (1.97)	40	261 (1.09)	> 10	−32	—	—	

[a] Figures in parentheses are equivalent units in megajoules (MJ), which is the approved SI unit to replace the thermochemical kilocalorie (kcal).
[b] Statistically significant increase in mean birth weight ($P < 0.05$).
[c] Statistically significant decrease in % of LBW births ($P < 0.01$).
[d] Statistically significant decrease in % of LBW births ($P < 0.05$).
[e] Grams of birth weight per 10 000 additional kcal (or 41.84 MJ) consumed.
[f] — denotes data not available.

supplementation increased the mean birth weight by 181 g among mothers of low weight-for-height (*50*). When the Guatemalan data were disaggregated by socioeconomic score, supplementation reduced the prevalence of LBW only among the more disadvantaged mothers. The reduction was from 29% to 13% (*59*). These examples emphasize the importance of targeting food supplements to those most at risk. Unfortunately, relatively little attention has been directed towards identifying meaningful and practical criteria for selecting pregnant women who would benefit from dietary supplementation. Anthropometric indices are likely to be the most appropriate criteria (*46*, *121*). Dynamic criteria such as weight gain are preferable, but the necessity for at least two measurements limits their feasibility.

Energy, rather than protein, appears to be the main factor limiting fetal growth, and the total additional calories consumed during pregnancy seem more important than the trimester in which supplementation is initiated (*62*). Where data permit, dose-response values have been calculated in terms of the mean increase in birth weight for every additional 10 000 kilocalories (41.84 MJ) consumed during pregnancy (Table 6). The average values vary between 17 g and 44 g per 41.84 MJ (10 000 kcal). Where data are disaggregated, the response increases with decreasing nutritional status of the mother.

Space does not permit a full discussion of the possible relationships between other specific nutritional deficiencies and birth weight here. Anaemia in pregnancy is common in developing countries (*5*) and severe anaemia (haemoglobin of < 3.7 mmol/l or 60 g/l) is associated with LBW (*104*). Although there may be a relationship between haematological status and birth weight (*40*, *104*, *122*, *128*), studies in developed countries (*49*) and in India (*109*) suggest that the administration of iron during pregnancy has no detectable effect on birth weight or length of gestation. These studies, however, have not examined the effect of iron supplementation on birth weight in severe anaemia, as opposed to mild anaemia. The relationship between maternal folate status and birth weight is not clear (*96*, *122*, *128*), but folate supplementation may enhance birth weight (*8*, *52*) and further research is warranted. Poor maternal zinc status is associated with SGA births (*72*, *84*). Prenatal supplemental iron or folate may adversely affect maternal zinc status (*47*, *74*, *108*).

Physically demanding work. A seasonal increase in the energy expenditure of pregnant women may have a greater impact on birth weight than a seasonal decrease in energy intake. For example, in Keneba in the Gambia, the decrease in birth weight preceded the decrease in energy intake and mirrored the increase in physical work (*94*), and in the village of Ikwiriri in the United Republic of Tanzania no seasonal decrease in birth weight was observed in 1979 when flood rains delayed field work (*7*). In a review of the pattern of work during pregnancy in 112 traditional societies, in 45% of them the women continued full duties until the onset of labour (*53*).

In the US Collaborative Perinatal Project, continuation of employment during the third trimester was associated with a reduction in mean birth weight of between 150 g and 400 g. The effect was greatest for women whose work involved standing, and those with a low pregravid weight, hypertension, or a low weight gain (*78*). In a Bombay cotton mill during 1925–28, the mean birth weight was 139 g lower among women mill-workers than among the non-working wives of mill-workers, all of whom lived in similar grossly overcrowded conditions. During 1928–29, when there was a general strike for 6 months and the women workers had an enforced rest, the mean birth weight increased by 151 g and the prevalence of LBW (< 5 pounds or 2268 g) decreased from 23% to 13% (*6*). In Dakar, birth weights were found to be relatively low in women involved in strenuous physical work but whose nutritional status was considered adequate (*13*). It is known that exercise and an upright posture adversely affect placental blood flow and it is for this reason that statutory maternity leave was first introduced in the United Kingdom. If exercise is undertaken in the heat, blood has to be diverted to the skin as well as to the muscles, thus reducing placental perfusion still further. Thus certain types of work after mid-pregnancy may impair fetal growth by reducing placental blood flow and/or by affecting energy balance.

In many developing countries it is likely that a reduction in the work load of pregnant women would reduce the prevalence of LBW. Clearly this would involve technological innovations and a radical change in social attitudes and in the work patterns of both men and women (*31*). Changes in social attitudes and work patterns are unlikely to be achieved in the short term.

Maternal health. Untreated maternal infections, hypotension, and hypertension may affect fetal growth. In endemic regions malaria is associated with LBW. In a study in southern Nigeria, 24% of parturient women had malarial infection of the placenta and the mean birth weight of their infants was 145 g lighter than the infants of non-infected women (*14*). Parasitaemia rates are highest among primigravidae (*120*). In the Gambia, the difference in mean birth weight between the infants of infected and non-infected women was approximately 150 g for firstborn infants and 60 g for all other births (*67*). In the Solomon Islands, the mean birth weight rose substantially within months of starting anti-*Anopheles*

spraying (*66*). Between 1969 and 1971 the mean birth weight increased by 252 g for babies of primigravidae and by 153 g for all other babies. The overall prevalence of LBW fell from 21% to 12%. Infections of the urinary tract are associated with preterm delivery (*77*) and with SGA births (*48*). In rural Guatemala, the incidence of urinary tract infection was 27 per 100 pregnancies (*115*). A number of studies have reported a reduction in the prevalence of LBW by antibiotic treatment of pregnant bacteriuric women (*1*). Amniotic fluid infections are common especially in undernourished gravidae, and infection within the amniotic cavity may initiate preterm labour (*75*). Poor maternal nutrition appears to interfere with the normal antibiotic activity of the amniotic fluid. In Ethiopia, antimicrobial activity was absent in 75% of randomly selected urban women at term, possibly as a result of zinc deficiency (*113*), and 31% of women had localized chorioamnionitis at delivery (*79*).

Maternal hypotension and hypertension are associated with reduced uteroplacental perfusion. In the British Perinatal Mortality Survey, severe pre-eclampsia (diastolic pressure of 14.7 kPa (110 mmHg) or more, or of 12 kPa (90 mmHg) or more with proteinuria) was associated with a 3.3 relative risk of a LBW delivery and with a mean birth weight reduction of 225 g (*15*). In Ethiopia, severe growth retardation was found in fetuses whose deaths were ascribed to pre-eclampsia (*112*).

Antenatal care of good quality is likely to improve maternal health and thereby reduce the prevalence of LBW. Such an effect has been documented in India (*80, 103*), the United Republic of Tanzania (*7*), and the USA (*45*). Unfortunately the existing coverage of antenatal services is very low in most developing countries and of the few women who use the services, most do so only in the last trimester.

Maternal age. Teenage mothers have a higher frequency of low-weight births for any given parity (*90*). In 1976 in the USA, teenage mothers had a relative risk of LBW of approximately 1.5 compared with mothers aged 20–24 years (*114*). In New Delhi the relative risk was 1.4 (*91*). A reduction in the proportion of teenage pregnancies should reduce the prevalence of LBW, but will be difficult to achieve in the short term in some societies.

Birth interval. Studies in the United Kingdom (*18*), the USA (Table 7), and Guatemala (*69*) have shown that a short birth interval, especially of < 12 months, is associated with an increased risk of LBW. In developing countries, short birth intervals are becoming more common especially in urbanized areas where reduced breast-feeding is associated with a shortened duration of postpartum amenorrhoea (*118*).

Table 7. Percentage of low-weight births among Black and White populations, by interval since last birth, in 43 States of the USA, 1976[a]

Birth interval (months)	Prevalence of LBW (%)	
	Black	White
< 12	26.9	14.8
12–23	12.1	5.0
24–35	9.9	3.9
36–47	9.7	3.8
⩾ 48	9.8	4.7

[a] All data from ref. *114*.

Tobacco smoking and chewing. It is well established that maternal smoking reduces birth weight (*42*) and that there is a linear dose–effect relationship in which the more cigarettes are smoked, the greater is the reduction (*16, 39*). The putative mechanisms responsible are discussed elsewhere (*26, 51, 57, 63, 64, 73, 127*). Smoking more than 15 cigarettes/day doubles the incidence of LBW. It has been shown that the adverse effect of smoking is independent of social class, maternal age, and parity (*16, 24, 42*). In a recent review, average reductions in birth weight ranging from 120 g to 430 g are reported (*57*). Tobacco chewing also reduces birth weight (*56*), although this has been little studied. In a prospective, randomized experiment (*101*), a specific antismoking campaign directed at pregnant smokers was associated with a significant increase in mean birth weight (+92 g). There was also a 24% reduction in the prevalence of LBW, from 8.9% to 6.8%, although this was not statistically significant.

Alcohol consumption. Animal studies suggest that alcohol is embryotoxic and teratogenic. The 'fetal alcohol syndrome' is found only among infants of mothers who regularly consume over 80 g alcohol/day. Characteristically these infants are growth retarded at birth and show a consistent pattern of congenital anomalies (*107*). Whether more moderate alcohol consumption, before or during pregnancy, adversely affects birth weight is less clear. Failure to allow for confounding variables and difficulties in obtaining accurate drinking histories may have contributed to the apparently conflicting results. A recent prospective investigation of 900 pregnancies in London, which was well controlled for confounding variables, has found that women consuming more than 100 g alcohol/week (1–2 drinks/day), around the time of conception, were more than twice as likely as light drinkers to have a LBW infant (*126*). There was no apparent benefit, in terms of birth weight,

from reducing such drinking once pregnancy was confirmed. The effects of heavy binge drinking are currently being analysed. Moderate drinking (50–100 g alcohol/week) was not associated with a significantly increased risk of LBW.

Conclusions on hypothesis 2. Poor maternal nutrition, certain infections, pre-eclampsia, arduous work after mid-pregnancy, short birth intervals, and teenage pregnancy are likely to be causally associated with LBW in developing countries. Tobacco and alcohol consumption are additional risk factors. It follows, therefore, that interventions that reduce the prevalence of these 'causes' or their relative risks will reduce the prevalence of LBW. Of the interventions examined, maternal food supplementation has been the most studied. If targeted to mothers at nutritional risk, and if the food is consumed in addition to the usual diet, the prevalence of LBW can be expected to be reduced. However, food supplementation can be expensive and the results from carefully supervised feeding trials may be better than those that can be achieved in national programmes. The effect of supplementation with iron, zinc, or folate requires further study.

Hypothesis 3. *Improving the nutritional status, health or life-style of pregnant women can reduce diarrhoea morbidity or mortality rates in young children.*

The only true test of this hypothesis would come from a study in which pregnant women received food supplementation, improved health care, or some other relevant intervention, and where the impact of this on LBW and on the diarrhoea rates in their children was monitored. A randomized controlled trial would be ideal, but might encounter ethical and logistical difficulties. No study of this type has been located, although in the Gambian prenatal supplementation study the preliminary findings[b] show that there has been a sustained improvement in the nutritional status of the wet season cohort. The most likely explanation is that their increased birth weight resulted in fewer, or shorter, episodes of diarrhoea. A few studies have been reported in which food supplementation for pregnant women, combined with other interventions, was introduced and the impact of the combined intervention on diarrhoea rates was recorded. These results cannot be used to test hypothesis 3 because the other interventions, such as food supplementation for infants, are ones that may well have independent effects upon diarrhoea rates. Hypothesis 3 must be examined, therefore, by theoretical calculations of the reductions in diarrhoea morbidity and mortality rates that might be achieved by levels of birth-weight enhancement that may result from improved maternal nutritional status, health or life-style.

Three hypothetical populations are defined. First, a relatively wealthy population having a prevalence rate of LBW of < 10%; we call this population 1. Second, an intermediate population, such as a poor community in a developed country or a more wealthy urban community in a developing country, having a prevalence rate of LBW of 10–20%; we call this population 2. Third, a relatively poor community having a

[b] PRENTICE, A. M. ET AL. *Effect of prenatal supplementation on birth weight and subsequent growth of infants.* Paper presented at the Fourth Asian Congress of Nutrition, Bangkok, 1–4 November 1983.

Table 8. Standardized birth-weight distributions for populations at three different socioeconomic levels

Birth weight (g)	Percentage of live births					
	Population 1 (relatively wealthy) % LBW = < 10		Population 2 (intermediate) % LBW = 10–20		Population 3 (relatively poor) % LBW = > 20	
< 1501	1.0	7.5	1.5	14.5	1.5	31.5
1501–2000	1.5		3.0		5.0	
2001–2500	5.0		10.0		25.0	
2501–3000	20.0		28.0		45.0	
3001–3500	40.0		36.0		20.0	
3501–4000	25.0		17.0		3.0	
> 4000	7.5		4.5		0.5	
Estimated mean birth weight (g)	3200		3000		2700	

Table 9. Standardized relative risks of neonatal mortality and post-neonatal mortality by birth weight compared to birth weight of 2501-3000 g

Birth weight (g)	Relative risk	
	Neonatal mortality[a]	Post-neonatal mortality[b]
< 1501	50.0	6.1
1501-2000	18.0	3.4
2001-2500	4.0	1.8
2501-3000	1.0	1.0
3001-3500	0.5	0.6
3501-4000	0.4	0.5
> 4000	0.6	0.5

[a] Median values from Table 3.
[b] Median values from Table 4.

prevalence rate of LBW of > 20%; we call this population 3. Using the data on birth-weight distributions in Table 1, each of these three populations is assumed to have a birth-weight distribution as set out in Table 8.

The reductions in infant mortality rates in populations 2 and 3 that may result from interventions that increase birth weight can now be calculated on the basis of the following assumptions.

(1) The effect of birth weight on risk of death does not extend beyond the first year of life. This may not be true but the assumption is necessary owing to lack of data. It is a conservative assumption.

(2) A LBW prevention programme in population 3 will shift the birth-weight distribution to that of population 2 (Table 8).

(3) A LBW prevention programme in population 2 will shift the birth-weight distribution to that of population 1 (Table 8). This is much less likely than assumption 2, as discussed below.

(4) The relative risks of neonatal and post-neonatal mortality by birth weight are as set out in Table 9, derived from the median values in Tables 3 and 4. This may bias the relative risk towards that prevailing in developed countries where LBW infants are predominantly preterm. Using the median value may overestimate the impact of a LBW prevention programme in North Arcot for example, where the relative risks appear to be less than the median. Until more data are available, this assumption is unavoidable.

(5) A LBW prevention programme changes the distribution of birth weight (see assumptions 2 and 3) but the neonatal and post-neonatal death rates for a specified birth weight remain constant. In the medium term this will clearly not be true and as LBW prevalence rates decline, so also will birth-weight-specific mortality rates. It is the correct assumption to make here, however, in order to separate out the impact of interventions to increase birth weight from the impact of other curative and preventive interventions.

These calculations do not take full account of the fact that the amenability of LBW to change, and the choice of interventions that will cause that change, depend on three characteristics of the low-weight births under consideration. First, amenability to change and choice of intervention will depend on the degree of LBW. Births of < 1500 g are unlikely to be reduced by the improvements in maternal nutritional status, health and life-style discussed here. To account for this, the prevalence rates of < 1500 g births have been set at 1.0% in population 1, and 1.5% in populations 2 and 3 (Table 8), despite the fact that the rates are actually > 2% among non-whites in the USA (Table 1). Second, amenability to change and choice of intervention will depend on the ratio of preterm to SGA births. SGA births are likely to be more responsive to the interventions discussed here than are preterm births. Third, and closely related to the last point, as the overall LBW prevalence falls, the ratio of preterm to SGA births will rise and so the LBW pattern will become less amenable to change through improvements in maternal nutritional status, health and life-style. Thus, it is likely to be much easier to shift the birth-weight distribution of population 3 to population 2, than to shift that of population 2 to population 1 (Table 8).

No data on the risk of diarrhoeal death by birth weight have been located. It is therefore assumed that the relative risks of diarrhoeal death by birth weight are the same as those for all death (Tables 3, 4 and 9). This assumption is unlikely to be correct for infants of very low birth weight (say, < 1500 g). However, such babies are assumed to make up a small and relatively unchanging proportion of all births in the 3 populations being considered (Table 8). For higher birth weights, and especially for post-neonatal mortality, the assumption that all deaths and diarrhoeal deaths have the same risk pattern by birth weight is not unreasonable and is supported by data from the Dutch famine study where infant diarrhoeal deaths and overall infant mortality increased in a similar manner in the SGA cohort born during the famine (*111*).

The effects of shifting the birth-weight distribution of population 3 to population 2, and that of population 2 to population 1, on the neonatal, post-neonatal, infant, and 0–4-year age group diarrhoea death rates are shown in Table 10. A population 3 to population 2 shift is what might be anticipated by a LBW prevention programme in a poor community in a

Table 10. The impact on diarrhoea mortality rates of shifting the distribution of birth weights[a]

Birth-weight distribution shift[b]	% reduction in diarrhoea			% of 0–4 years diarrhoea deaths that occur in infants	% reduction in diarrhoea mortality among 0–59-month-old children[d]
	Neonatal mortality	Post-neonatal mortality	Infant mortality[c]		
Population 3 to population 2	30	25	26	40	10
				60	16
				80	21
Population 2 to population 1	32	17	19	40	8
				60	11
				80	15

[a] All calculations assume that the relative risks of diarrhoea death by birth weight are the same as the relative risks of all death by birth weight (see text) and are as set out in Table 9.

[b] See Table 8 for the birth-weight distributions of populations 1, 2 and 3.

[c] Calculated by assuming that 14% of infant diarrhoea deaths occur in the neonatal period (median figure obtained in 8 studies).

[d] Calculated by assuming that either 40%, 60% or 80% of diarrhoea deaths in children under 5 years old occur in children under 1 year, and that birth weight has no effect on the risk of diarrhoea death beyond 1 year of age (see text).

developing country. It presumes a reduction in LBW from 31.5% to 14.5% (a 54% reduction, see Table 8), which has been achieved in the Gambia and Mexico (Table 6). It further presumes an increase in mean birth weight of 300 g, something that appears far harder to achieve on the evidence presented in Table 6. A population 2 to population 1 shift is what might be anticipated by a LBW prevention programme in a Third World city or in a relatively wealthy, developing country. It presumes a reduction in LBW from 14.5% to 7.5% (a 48% reduction, see Table 8), which has been achieved in China (Province of Taiwan) (Table 6). It further presumes an increase in mean birth weight of 200 g, something that appears harder to achieve on the evidence of Table 6.

Expected reductions in diarrhoea mortality rates are 30–32% in the neonatal period, 17–25% in the post-neonatal period, and 19–26% for the entire first year of life (Table 10). Expected reductions in diarrhoea mortality rate in the 0–4-year age group are 8–21%, assuming that LBW confers no excess risk of diarrhoea death after 12 months of age (Table 10). This last expected reduction is sensitive to the proportion of 0–4-year diarrhoea deaths that occur in the first year of life. Widely differing proportions are reported; for instance, 90% in Recife, Brazil (*91*), 81% in Latin America (*89*), 68% in Ludhiana, India (*91*), 55% in North Arcot, India (*92*), and around 40% in a number of studies in Asia and Latin America where active surveillance was employed (*106*). Three proportions (40%, 60% and 80%) are adopted for comparison in Table 10.

There is no good evidence to suppose that either a population 3 to population 2, or a population 2 to population 1, birth-weight-distribution shift is achievable on a national scale by interventions in maternal nutrition, health and life-style. As noted above, the second of these shifts is especially unlikely in the short term. The impacts computed in Table 10 are those expected *if* the birth-weight changes under discussion were achieved. Lesser birth-weight increases would result in lesser reductions in mortality.

Conclusion on hypothesis 3. If it were possible to intervene in maternal nutrition, health and life-style in a developing country in a way that reduced the prevalence of LBW from around 30% to around 15% (Table 8), a fall in infant mortality rate of around 26% would be expected (Table 10). The fall in infant diarrhoea mortality rate might be similar. The scarce data on the relative risk of morbidity by birth weight do not allow any comparable computations for morbidity reductions to be made.

FEASIBILITY AND COST

In the consideration of hypothesis 2 above, a range of interventions were shown to be likely to decrease the prevalence of LBW. These interventions would seek to increase energy intake, decrease arduous work, improve antenatal health care, and reduce tobacco and alcohol consumption in pregnant women. Interventions would also seek to discourage teenage pregnancies and increase the birth intervals. We have reviewed five small-scale feeding trials in developing countries (Table 6). We have no know-

ledge of any regional or nationwide programmes for LBW prevention in developing countries using any of the interventions mentioned above that have been evaluated. We can therefore say little about the feasibility and cost of such programmes.

Energy supplementation for pregnant mothers at nutritional risk is perhaps the most likely of the interventions listed above to achieve reductions in LBW prevalence in the short term. However, such an intervention suffers from the very considerable cost, logistical and other disadvantages of all supplementary feeding programmes (*33*). Changes in work patterns and attitudes towards work during pregnancy may be extremely resistant to change in the short term. Antenatal care is gradually improving in most developing countries, and it is unlikely that a LBW prevention programme could significantly accelerate this process. Vigorous educational programmes may be able to reduce tobacco and alcohol intake during pregnancy, but we know of no documented examples of such campaigns in developing countries. Discouraging teenage pregnancy, and promoting longer birth intervals, are part of existing birth control programmes in several developing countries. They should have a beneficial effect upon birth weights but this is secondary to the primary goal of reducing the birth rate.

CONCLUSIONS

A substantial reduction in the prevalence of low birth weight is theoretically possible and would lead to a substantial reduction in infant mortality rate (Table 10). The effect on diarrhoeal mortality or morbidity rates is unknown, however. In this paper it has been assumed that the relative risk of diarrhoeal death by birth weight is the same as that for all death (Table 9). Data are so scarce on the risk of morbidity, diarrhoeal or other, by birth weight that no computations have been possible.

Prospective studies are required that record from birth the diarrhoea morbidity rates, and if circumstances permit, the diarrhoea mortality rates, of groups of infants having known birth weights. These infants should be followed for at least 12 months, and preferably 24 months to determine if LBW confers an excess risk of diarrhoea in the second year of life. The relative risk values obtained may be used to compute the reductions in diarrhoea morbidity and mortality that would follow from a given improvement in birthweight distribution.

At the same time, greater attention should be paid to the problem of LBW in developing countries. This review has confirmed that, whatever its association with diarrhoea, LBW is an important determinant of infant mortality. For the more general goal of reducing infant mortality it is necessary to know more about the nature (preterm vs small for gestational age), etiology, and prevention of LBW in developing countries. In particular, it is necessary to know whether interventions to reduce LBW are feasible at a national or subnational scale in developing countries and whether such interventions are cost-effective in comparison with other strategies for reducing infant mortality rates.

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RÉSUMÉ

INTERVENTIONS CONTRE LES MALADIES DIARRHÉIQUES CHEZ LE JEUNE ENFANT: PRÉVENTION D'UN FAIBLE POIDS DE NAISSAANCE

Le présent article constitue la cinquième mise au point d'une série sur les interventions possibles en vue de réduire la morbidité et la mortalité associées aux maladies diarrhéiques chez les enfants de moins de cinq ans dans les pays en développement. On étudie ici l'influence d'un faible poids de naissance (FPN) sur la morbidité et la mortalité par diarrhées ainsi que les interventions visant à augmenter ce poids. Tout en reconnaissant que l'étiologie du FPN est multifactorielle, l'accent est mis sur les facteurs maternels dont on pense qu'ils ont le plus d'importance dans les pays

en développement et qu'ils sont susceptibles d'évoluer dans un proche avenir, moyennant des interventions convenables.

Sur l'ensemble des naissances, on compte 16% de FPN (< 2500 g). D'après les observations, la majorité des cas de FPN seraient des prématurés dans les pays développés, et des enfants petits par rapport à leur âge gestationnel (PAG) dans les pays en développement. Le poids de naissance conditionne largement la mortalité infantile et, au moins dans les pays développés, son influence sur la mortalité néonatale ne dépend pas de la situation socio-économique. Chez les prématurés de poids normal pour leur âge gestationnel (NAG), on observe un taux de mortalité infantile plus élevé que chez les nourrissons PAG mis à terme, la différence étant particulièrement marquée à la période néonatale. Aucune donnée convaincante n'a pu être trouvée quant au rôle étiologique du FPN dans la morbidité ou la mortalité d'origine diarrhéique. L'association étroite entre FPN et mortalité fait qu'il existe probablement un lien entre FPN et mortalité d'origine diarrhéique dans les pays en développement où les diarrhées sont l'une des grandes causes de mortalité infantile. L'immunité est gravement amoindrie chez les nourrissons de faible poids de naissance, spécialement les nourrissons PAG.

Une nutrition médiocre chez la mère, certaines infections, la néphropathie gravidique, un travail pénible au-delà du milieu de la grossesse, des naissances rapprochées et le jeune âge de la future mère, encore adolescente — tous ces facteurs ont certainement un lien de cause à effet avec le FPN dans les pays en développement. L'usage du tabac et la consommation d'alcool sont des facteurs de risque supplémentaires. Il s'ensuit que les interventions qui rendent ces "causes" moins fréquentes ou qui abaissent le risque relatif correspondant, sont de nature à réduire la prévalence du FPN. Parmi les interventions examinées, l'administration à la mère de suppléments nutritionnels a été la plus étudiée. Cette intervention est en principe efficace si elle vise les femmes en cause du fait de leur nutrition et si les aliments distribués viennent compléter leur ration habituelle. Cependant, la distribution de suppléments nutritionnels peut être coûteuse, et des essais soigneusement supervisés pourraient donner de meilleures résultats que ceux qu'on obtient dans les programmes nationaux. L'effet de suppléments de fer, de zinc ou de folate nécessite des études complémentaires.

Si l'on pouvait agir sur la nutrition, l'état de santé et le mode de vie des mères dans un pays en développement de façon à ramener la prévalence du FPN d'environ 30% à 15%, on devrait obtenir une baisse du taux de mortalité infantile de l'ordre de 26%. Le taux de mortalité infantile par diarrhée devrait enregistrer une chute similaire. Les rares données dont on dispose sur le risque relatif de morbidité associé à un faible poids de naissance ne permettent pas de faire des calculs analogues quant à la réduction à attendre pour le taux de morbidité.

Il est indispensable de réaliser des études prospectives où seront enregistrés dès la naissance le taux de morbidité par diarrhée (et, si les circonstances le permettent, le taux de mortalité correspondant) dans des groupes de nourrissons ayant un faible poids de naissance, de valeur connue. En même temps, il faudra se préoccuper davantage du problème du FPN dans les pays en développement. La présente étude a confirmé que le FPN constitue, quel que soit son lien avec la diarrhée, un déterminant important de la mortalité infantile. S'agissant de l'objectif, plus général, d'un recul de la mortalité infantile, il faudrait en savoir plus sur la nature (prématurés ou enfants petits pour leur âge gestationnel), l'étiologie et la prévention du FPN dans les pays en développement. En particulier, il convient de savoir si les interventions visant à limiter les cas de FPN sont praticables dans ces pays, à l'échelle nationale ou infranationale, et si elles ont un rapport coût/efficacité favorable par comparaison à d'autres stratégies envisageables pour faire reculer la mortalité infantile.

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