

Mortality rates in displaced and resident populations of central Somalia during 1992 famine

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Famine and civil war have resulted in high mortality rates and large population displacements in Somalia. To assess mortality rates and risk factors for mortality, we carried out surveys in the central Somali towns of Afgoi and Baidoa in November and December, 1992. In Baidoa we surveyed displaced persons living in camps; the average daily crude mortality rate was 16.8 (95% CI 14.6–19.1) per 10 000 population during the 232 days before the survey. An estimated 74% of children under 5 years living in displaced persons camps died during this period. In Afgoi, where both displaced and resident populations were surveyed, the crude mortality rate was 4.7 (3.9–5.5) deaths per 10 000 per day. Although mortality rates for all displaced persons were high, people living in temporary camps were at highest risk of death. As in other famine-related disasters, preventable infectious diseases such as measles and diarrhoea were the primary causes of death in both towns. These mortality rates are among the highest documented for a civilian population over a long period. Community-based public health interventions to prevent and control common infectious diseases are needed to reduce these exceptionally high mortality rates in Somalia.

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Introduction

Refugees and people displaced within their own countries suffer high mortality rates during the early phases of population displacement.¹ Massive population displacements are generally due to war, famine, drought, or a combination of these factors. Displaced populations face health problems compounded by the deprivations that initiated the migration and the lack of public health infrastructure in refugee camps.² Malnutrition, crowding, lack of drinking water, and lack of sanitation can lead to intense epidemics with high mortality rates.³

Over the past few years, Somalia has suffered from a civil war, which has interrupted farming and destroyed major sources of income for both urban and rural populations. In 1991–92, the Horn of Africa experienced a severe drought, which further diminished food production and led to widespread famine in southern and central Somalia. The famine resulted in massive population displacements to feeding centres in urban areas and to refugee camps in Ethiopia and Kenya. Relief agencies worked in Somalia

throughout the period of civil unrest and regular airlifts of food began in August, 1992.

To assess mortality rates, major causes of death, and risk factors for mortality in displaced and resident populations of central Somalia, we carried out population-based surveys in the towns of Baidoa and Afgoi on Nov 20–25 and Dec 5–6, 1992, immediately before the multinational military intervention on Dec 9, 1992.

Subjects and methods

The surveys were designed to assess causes of death in nine central Somali towns by means of a standard thirty-cluster survey design,⁴ but conditions were too dangerous for completion of most of the surveys. The study was therefore modified to assess recent mortality by a stratified survey design in two Somali population groups—a camp for displaced persons in Baidoa and the town of Afgoi, which included both displaced persons and long-term residents. There are no reliable census data for Baidoa or Afgoi and population estimates were made by relief agencies working in the towns. People in our survey were taken as displaced persons only if they had moved to their present location during the previous 24 months because of war or famine.

Baidoa, the regional capital of the Bay Region, lies 250 km west of the Somali capital, Mogadishu. The population of Baidoa fell substantially between August and November, 1992, owing to high mortality and emigration, and the estimated population in November, 1992, was between 21 000 and 40 000. Access to the whole town was not possible because it was unsafe, so we surveyed only people in displaced persons camps. Based on hut counts, the number of displaced persons in Baidoa living in temporary camps was estimated to be 5200. The population was divided into seven sections, and a random starting point was selected from the centre of each section. Seven consecutive huts were visited at each starting point (except at one starting point where only five huts could be safely visited), and the head of household in each hut was interviewed by a trained interpreter and one of the study organisers. If the head of household was not available, questions were addressed to another adult living in the household.

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TABLE I—DEATHS AND MORTALITY RATE BY AGE GROUP FROM APRIL TO NOVEMBER, 1992

	Starting population	No (%) deaths	Mortality rate per 10 000 per day (95% CI)
<i>Baidoa</i>			
≥5 yr	276	86 (31)	13.4 (11.1–15.8)
<5 yr	62	46 (74)	32.0 (27.3–36.7)
All ages	338	132 (39)	16.8 (14.6–19.1)
<i>Afgoi</i>			
≥5 yr	808	62 (8)	3.2 (2.4–3.9)
<5 yr	211	53 (25)	10.4 (8.0–12.9)
All ages	1019	115 (11)	4.7 (3.9–5.5)

The Baidoa figures for displaced persons only, Afgoi for both displaced and resident populations. The survey intervals for Baidoa and Afgoi were 232 and 242 days, respectively.

Afgoi lies 40 km west of Mogadishu and had an estimated population of 35 000 in November, 1992. About 1000 displaced persons lived in temporary camps in the town, and others in houses in the town. The town was divided into nineteen sections containing approximately equal numbers of houses; survey starting points were randomly chosen from the centre of each section. Eight consecutive households were interviewed at each starting point. One of the nineteen starting points was in a displaced persons camp, and the other sites were randomly chosen from among the settled population of Afgoi.

An adult in each household was asked to identify all living household members and whether any household members had died between the end of Ramadan (April 3, 1992) and the survey date (232 days for Baidoa and 242 days for Afgoi). Deaths during the 30-day period before the interview were recorded separately. To improve recall accuracy, an interviewee who reported a death was interviewed in detail about the date and circumstances of death.

To determine causes of death, a structured post-mortem interview was done. We first asked an open question about the presumed cause of death and then a series of questions on specific symptoms before death. Five causes of death were assessed—measles, diarrhoeal disease, acute respiratory infection, trauma (both war related and not war related), and maternal death during childbirth. Some common causes of death, such as malnutrition and malaria, were not specified because it is difficult to determine specific symptoms accurately from post-mortem interviews. Deaths not clearly attributable to one of the specified causes were classified as "other/unknown".

By convention for refugee and displaced person assessments, crude and age-specific mortality rates were calculated as the average daily rate per 10 000 persons during the survey periods with the starting population as the denominator.⁵ Each sector in either Afgoi or Baidoa contained about the same number of households; therefore, surveying a fixed number of households in each sector provided a selection proportional to the population within each sector. Confidence intervals for mortality rates were calculated by the normal approximation to the binomial confidence interval method. Risk ratios and 95% CI (Taylor series approximation) were calculated with Epi Info;⁶ simple random sampling was assumed. Adjusted risk ratios were calculated for Afgoi participants only by stratification on the basis of residence status by the Mantel-Haenszel method with Robins-Greenland confidence intervals.⁷

Results

In Baidoa, survey data were collected for 338 persons reported to be alive on April 3, 1992, in 47 displaced households. Of these, 62 (18.3%) were children under 5 years. Households in April, 1992, contained an average of 7 members (range 1–16). Between April 3 and Nov 21, 1992, 132 (39%) of the 338 people died; the average crude mortality rate was 16.8 deaths per 10 000 daily (95% CI 14.6–19.1, table 1). Displaced children under 5 years old had a disproportionately high mortality rate (32.0 [27.3–36.7] per 10 000 per day). There was no improvement in the

TABLE II—HOUSEHOLD RISK FACTORS FOR MORTALITY

	n*	No (%) deaths	Mantel-Haenszel risk ratio (95% CI)*
<i>Household status</i>			
Resident	764	67 (9)	1.0
Displaced in houses	193	28 (14)	1.7 (1.1–2.5)
Displaced in camps	62	20 (32)	3.7 (2.4–5.6)
<i>Livestock ownership</i>			
Yes	36	2 (6)	1.0
No	983	113 (11)	2.5 (0.6–10)
<i>Owens chickens</i>			
Yes	181	10 (6)	1.0
No	838	105 (13)	2.0 (1.0–3.3)
<i>Income from work outside home</i>			
Yes	404	44 (11)	1.0
No	610	71 (12)	1.1 (0.7–1.4)
<i>Male head of household†</i>			
Absent	144	15 (10)	1.0
Present	709	77 (11)	1.0 (0.6–1.7)
<i>Received general rations in previous 2 wk</i>			
Yes	118	16 (14)	1.0
No	888	98 (11)	0.8 (0.5–1.3)

*April 3, 1992.

†For variables other than household status, adjusted for this factor.

‡Deaths of male heads of household excluded.

overall crude mortality rate in Baidoa for the month before the survey; 17 of 223 persons died in the 30 days before the survey and 115 during the preceding 202 days (crude mortality rate 25.4 [13.8–37.0] vs 16.8 [14.3–19.3] per 10 000 per day).

In Afgoi we collected data for 1019 displaced persons and residents in 152 households who were alive on April 3 (table 1). The average number of members of each household in April was 7 (range 1–21). Of the 1019 people, 115 (11%) died between April 3, 1992, and the beginning of the survey (crude mortality rate 4.7 per 10 000 per day [3.9–5.5]). As in Baidoa, children under 5 years old had a higher mortality rate than the rest of the population. Again as in Baidoa, there was no improvement in mortality rate in the month before the survey compared with the previous 212 days (6.2 [3.3–9.1] vs 4.5 [3.7–5.4] deaths per 10 000 per day).

We assessed risk factors for mortality among Afgoi participants for the whole 242-day survey period. Although mortality rates were high for all groups, displaced persons were at higher risk of dying than residents, and displaced persons in temporary camps were at highest risk (table II). Overall, 48 (18%) of 255 displaced persons died compared with 67 (9%) of 763 permanent Afgoi residents (risk ratio 2.2, 95% CI 1.5–3.0).

People living in households that owned chickens, a possible indicator of higher socioeconomic status, had a significantly lower risk of dying than people from households without chickens, after adjustment for residence status (table II). The association between lower mortality

TABLE III—CAUSE-SPECIFIC MORTALITY RATES

Cause	Baidoa		Afgoi	
	No (%) deaths	Cause-specific mortality rate*	No (%) deaths	Cause-specific mortality rate*
Measles	33 (25)	4.2	39 (34)	1.6
Diarrhoeal disease	74 (56)	9.4	22 (19)	0.9
Acute respiratory infection	3 (2)	0.4	4 (3)	0.2
Trauma	2 (2)	0.3	9 (8)	0.4
Childbirth	0	..	0	..
Other/unknown	20 (15)	2.6	41 (36)	1.7

*Deaths per 10 000 per day, for April–November, 1992.

and ownership of livestock was not significant, perhaps because of small numbers. Other socioeconomic factors did not predict mortality risk.

Measles and diarrhoea together accounted for 81% of deaths in Baidoa and 53% of deaths in Afgoi (table III). Only 2 of 132 deaths in Baidoa and 9 of 115 in Afgoi were directly attributed to war-related trauma. Although malnutrition was not included as a single cause of death in our survey, many deaths were said to be associated with profound oedema, a common terminal symptom of severe protein-calorie malnutrition. These deaths were classified as other/unknown unless specific symptoms were noted.

Discussion

In most countries of sub-Saharan Africa, reported national mortality rates are between 20 and 24 deaths per 1000 population per year, equivalent to 0.55–0.65 deaths per 10 000 per day.^{8,9} Although displaced persons commonly have higher mortality rates than non-displaced persons,¹ the mortality rates found in our survey are among the highest recorded for civilian populations over a long period.^{3,8} Among displaced households surveyed in camps in Baidoa, mortality rates were about thirty times higher than expected in peacetime. Nearly 75% of displaced Baidoa children under 5 years died between April and November, 1992, and the proportion of children under 5 in the displaced population fell from 18.3% to 7.8% during this period. Independent evidence corroborating these mortality rates was obtained from daily records of bodies collected for burial by the Somali Red Crescent Society; between Aug 9 and Nov 14, 1992, 12 255 bodies were collected from both displaced and resident areas of the town.¹⁰ Deaths peaked from late August to early September during simultaneous epidemics of measles and *Shigella dysenteriae* infection.

The survey in Afgoi among both displaced and resident populations may better reflect population-based mortality rates in central Somalia. Mortality rates for the whole Afgoi population were lower than those for displaced persons in Baidoa, but were eight times higher than would be expected for a similar population under non-famine conditions. Our mortality rate is more than double that in Manoncourt and colleagues' mortality survey¹¹ in the Merca-Qorioley area of central Somalia from April, 1991, to April, 1992. In both surveys displaced persons living in camps were about three times more likely to die than non-displaced residents. With the exception of poultry ownership and residence status, no socioeconomic risk factors assessed were associated with mortality risk in our survey. Since displaced persons may migrate because of pre-existing poor health, nutrition, and socioeconomic conditions, these high mortality rates are likely to be due to poor public health conditions both before and after migration.

The high mortality even among the resident population of Afgoi shows the enormity of the public health disaster in Somalia. There is no evidence that mortality rates improved during the 30 days before the survey in either town despite the continuing relief effort. Lack of security and limited transport hindered effective distribution of food and medicines between August and December, 1992. It is not yet clear whether the recent multinational military intervention will lower mortality through improved relief efforts.

Manoncourt et al¹¹ found that malnutrition and war-related trauma were the main causes of death in the Merca-Qorioley area in 1991–92, but preventable infectious diseases were the main causes of death in our study in Afgoi and Baidoa. This difference may be due partly to differences

in the survey instruments used, but it is likely that there has been a real increase in cause-specific mortality due to measles, diarrhoea, and dysentery since April, 1992. Multidrug-resistant *Shigella dysenteriae* has been isolated from patients in Baidoa (S. Toussi, International Medical Corps), and there have been widespread outbreaks of measles in both Baidoa and Afgoi during the past 6 months. This shift in cause-specific mortality may be due to breakdown of public health infrastructure in a setting of widespread malnutrition.

Because of the limitations of our surveys, care should be taken in projecting mortality rates from this study to the whole Somali population. The emigration of healthy people from Baidoa and Afgoi during the survey period would increase apparent mortality rates, whereas deaths of whole families would lead to underestimation of mortality rates. In Baidoa, only the displaced population living in camps was surveyed, and no direct information is available for non-displaced Baidoa residents. Although the Afgoi survey sampled both displaced and non-displaced residents, mortality rates in this town may not be representative of rural areas, towns with intense fighting, or areas without active feeding programmes. Mortality rates for displaced persons may reflect a combination of mortality rates before and after migration, and perhaps should not be taken to indicate mortality after displacement. Other surveys of populations throughout the country are needed so that major public health priorities for Somalia during the post-intervention period can be determined.

The main causes of death in our survey were similar to those found in other famine disaster settings.¹ The highest proportions of deaths were due to two common and preventable conditions, measles and diarrhoeal diseases. Our findings reinforce the need for aggressive prevention and management of common infectious diseases in displaced populations, especially during periods of widespread famine. Mortality can be substantially reduced by establishment of measles immunisation,¹² vitamin A supplementation,¹³ and oral rehydration clinics¹⁴ at food distribution and registration centres during refugee operations. In addition to food relief, immunisation and community health worker programmes should have high priority during emergency relief operations to provide prevention and treatment of measles, diarrhoea, malaria, and acute respiratory infections. These programmes have been difficult to implement in Somalia because of the widespread fighting. Finally, high priority should be placed on implementing a simple public health surveillance system to monitor the effect of relief efforts on affected populations.^{15,16}

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Fetal nutrition and cardiovascular disease in adult life

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Babies who are small at birth or during infancy have increased rates of cardiovascular disease and non-insulin-dependent diabetes as adults. Some of these babies have low birthweights, some are small in relation to the size of their placentas, some are thin at birth, and some are short at birth and fail to gain weight in infancy. This paper shows how fetal undernutrition at different stages of gestation can be linked to these patterns of early growth. The fetuses' adaptations to undernutrition are associated with changes in the concentrations of fetal and placental hormones. Persisting changes in the levels of hormone secretion, and in the sensitivity of tissues to them, may link fetal undernutrition with abnormal structure, function, and disease in adult life.

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Association between early growth pattern and disease in adults

Two English surveys have indicated that low growth rates in utero and during infancy are associated with high death rates from cardiovascular disease. One of them, of 1586 men born in a maternity hospital in Sheffield during 1907-25, showed that death rates from cardiovascular disease fell progressively with increasing weight, head circumference, and ponderal index (weight/length³) at birth.¹ In the other, of 5654 men born in Hertfordshire during 1911-30, death rates from coronary heart disease were almost three times higher among those who weighed 18 lb (8.2 kg) or less at age 1 year than among those who weighed 27 lb (12.3 kg) or more.²

Examination of men and women in different populations in Britain has shown that low growth rates up to the age of one year are associated with increased prevalence of known risk factors for cardiovascular disease, including blood pressure,³ and plasma concentrations of glucose, insulin,⁴

fibrinogen,⁵ factor VII,⁵ and apolipoprotein B.⁶ These associations parallel those with death rates from cardiovascular disease. The associations are seen in babies who are born small for their gestational age rather than those born prematurely.^{1,3} They are found not only among babies with intrauterine growth retardation, defined by birthweight at the lowest centiles, but are also seen in babies of average or even above average weight at birth. Some of the subjects were small at birth in relation to the size of their placentas;³ others were thin at birth;^{1,7} and yet others, though of average birthweight, were short in relation to head size and had below average infant weight gain.^{2,5}

Numerous animal experiments have shown that poor nutrition, and other influences that impair growth during critical periods of early life, may permanently affect (programme) the structure and physiology of a range of organs and tissues, including the endocrine pancreas, liver, and blood vessels.^{8,9} For example, retardation of intrauterine growth in the guinea pig causes life-long elevation of blood pressure.¹⁰

A simple example of programming in human beings is the permanent deformity of the pelvic bones caused by rickets in infancy. Since different tissues mature during different, often brief, periods of fetal life and infancy, the long-term consequences of altered nutrition depend on its timing and its duration. Consistent with this, different patterns of early growth are associated with different adult abnormalities. For example, those who are thin at birth, as measured by a low ponderal index (weight/length³), tend to develop the

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Famine in Somalia

SIR,—Dr Moore et al (April 10, p 935) advocate other surveys to determine public health priorities in Somalia. At the same time as they were doing their work we measured the nutritional status of 482 individuals in three areas of Mogadishu: a village on the outskirts (29 households), a hamlet in the north (36 households), and one camp for displaced persons (85 households). We measured weight, height, and upper arm circumference of all present members of randomly selected households, and recorded clinical signs of nutritional deficiency.

The mean weight for height was -1.2 (range -2.9-1.6) Z score units in children 0-23 months, -1.4 (range -4.9-0.8) in children aged 24-59 months, and -1.4 (range -4.4-0.7) in those aged 60-119 months. There was moderate wasting (weight for height < -2 Z) in 16% of children under 5 and 13% of the 5-10 year-olds. The mean BMI of adults (20-60) was 19.7 (range 14.9-25.1) in men and 19.8 (13.2-27.5) in women. It decreased in the elderly (>60) to 17.8 (12.2-22) in men and 19 (12.1-28.2) in women. A body-mass index (BMI) below 17, indicating moderate-to-severe wasting,² was seen in 25 (15%). The medium upper arm circumference of these wasted individuals was 20 (SD 2) cm. Clinical examination showed iron and vitamin deficiencies: bleeding gums in 7 (3%) children under 10, and in 20 (7%) older children and adults. Bitot spots were seen in 3 (1%) children under 10, all of whom had had measles in the weeks before the survey. Pale mucosae (tongue and conjunctiva) were seen in 52 (25%) children, and 143 (51%) older children and adults.

There were no differences in mean weight for height, BMI, or clinical signs of nutritional deficiencies between the local and the displaced households, nor differences among the displaced according to where they come from. Children's weight for height had almost reached the pre-war values for rural areas (-0.7 to -1.3).² Comparisons with pre-war records from the town of Mogadishu can only be made with weight for age. At the time of this survey the median weight for age corresponded to the 2nd centile of the National Childhood Health Survey reference, while in the 1970s, the weight for age of Mogadishu children was between the 10th and the 25th.³ The BMI of adults was similar to that of other African populations outside famine periods.

Despite a proportion of severe cases who still need intensive nutritional support, the survey data show that the nutritional emergency has been overcome in Mogadishu, where international aid has been delivered fairly regularly. It is likely that only the fittest have managed to survive and reach the refugee camps there, leaving behind those among whom Moore et al observed high mortality. As local production has not yet been resumed, food aid is still needed, with special attention paid to the content of iron, folate, vitamin A, and vitamin C.

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SIR,—Dr Moore and colleagues report high mortality in Afgooye town and other areas in central Somalia during the 1992 war and famine. During 1987-89 we studied farmers in the rural area close to Afgooye. A demographic surveillance system was run in close collaboration with village leaders, community health workers, and traditional midwives.¹ 9610 person-years of follow-up are included in this analysis. An increasing under-five mortality was already noted during the 3 years 1987-89, probably reflecting a deteriorating society (table). A comparison between the 1992 data reported by Moore et al and the pre-war situation in 1987 shows that the yearly crude under-five mortality rate had increased 8 times.

MORTALITY BEFORE AND DURING SOMALI CIVIL WAR

	Rates per 1000 per year			
	Afgooye rural**			Afgooye urban
	1987	1988	1989	1987†
Age (yr)				
<5	49.0	81.4	117.1	379.6
>5	18.9	29.4	29.1	116.8
Cause				
Measles	0.8	0.5	2.2	38.4
Diarrhoea	4.2	3.1	10.8	32.9
Acute respiratory infection	7.6	9.7	8.9	7.3
All causes	24.8	38.3	41.3	171.6

*Figures age-adjusted with 1987 as reference.

†Calculated from data of Moore et al.

The cumulative under-five mortality rates were 217, 312, and 366 per 1000 during 1987, 1988, and 1989, respectively. The figure from 1989 is about the same as that reported from Mozambique during the civil war.² The major killer in children as well as in the total population in 1987 was respiratory infection (table). However, during the period of successive social and economical collapse the diarrhoeal and vaccine-preventable diseases emerged as major threats. Vaccines were no longer available and simple drugs for village health services were difficult to obtain. Our estimate is that 30% of child deaths could have been prevented by a continued good vaccination coverage, lowering the cumulative under-five mortality for the whole period from 299 to 193 per 1000. The mortality from acute respiratory infections remained the same, and is similar to that reported by Moore et al from the 1992 war and famine.

Our data underline the increasing mortality risks for children in a collapsing society—even before any shot is fired. The pattern of causes of mortality is different from the pre-war situation. Almost two-thirds of the increase in mortality risks in the general population was caused by measles and diarrhoea. This stresses the need for a massive prevention and management assistance programme parallel to the peace-keeping efforts.

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SIR,—The article by Dr Moore et al shows the lack of attention paid to famine by the scientific community. Moore and colleagues report the death rates in Baidoa as "some of the highest ever recorded". However, their survey consisted of a sample of just 47 displaced households. This was the only epidemiological survey done in Baidoa and the whole Bay region of Somalia during 1992. Their conclusion (based upon a sample of only 212 adults and 19 under fives) was that the death rate did not improve in November. Those of us living and working in Baidoa had very different impressions. In November there was a dramatic decrease in the numbers of dead people lying in the streets and the number of bodies collected by the Somali Red Crescent death cart was only a fifth of the September figure. Neither was attention drawn to the fact that from October onwards more than 70% of the reported deaths were of adults, a potentially relevant and helpful observation in a town that until the end of October had no facilities for severely malnourished adults.

The CDC survey relied upon standard epidemiological questions which are not always appropriate. The consistently high numbers of deaths reported in response to the question "how many of your family died in the last month?"^{1,2} is a good example. Most people in Baidoa were aware that expatriates were associated with the distribution of food and clothing, consequently there was a considerable incentive to over-report recent deaths and hardship.

Reliance on one methodology without verification via other important sources of data, such as the Somali Red Crescent death cart, greatly reduced the usefulness of Moore and colleagues' visit. Preliminary analysis of data taken at the Concern Worldwide adult therapeutic centre in Baidoa underlines the poor state of knowledge about adult malnutrition. The average body mass index of the first 650 severely malnourished adults and adolescents was 13. This is substantially lower than 16, recently proposed as the level to mark the most severe grade of adult starvation.³ Others survived with a body mass index below 10 on admission. Famine oedema occurred in 30% of admissions and was associated with a poorer prognosis.

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1. Merceur A. *Medecins Sans Frontieres* (Holland), Health surveillance review, Baidoa, Somalia, 1993.
2. Collins S. Concern Worldwide Survey of nutrition and food distribution in North West Baidoa district February 1993.
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SIR,—Your April 23 editorial "Do epidemiologists cause epidemics?" brought to mind a similar speculation: "Do nutritionists cause mass starvation?" Your April 9 issue carries a paper by Dr Moore and colleagues on microdemographic research in Somalia undertaken by the Centers for Disease Control (CDC). This study detailed high mortality among two populations of displaced people, with death rates of 4.7 and 16.8 per 10 000 per day, respectively, over a period of 8 months. These rates are high, but well within the recorded range for famine relief shelters, particularly in view of the fact that the displaced people in Baidoa, who suffered the higher rate, were the remnant of a larger population subject to much out-migration. As in all recorded famines, the overwhelming causes of death were preventable infectious diseases such as measles and diarrhoea.

However, when these results were first announced in December, 1992,¹ the media claimed the rate to be the highest ever recorded and attributed the deaths exclusively to starvation. This was because, in addition to the 8 month death rate, CDC included death rates in the month before, when, in Baidoa, 5 children out of 21 had died—equivalent to 69.4 per 10 000 per day. These five unfortunate children received a wholly unwarranted international notoriety: "Somalia has amongst the highest death rate from starvation ever recorded... deaths among children under five in one village reached 69.4 per day for every 10 000 children—more than three times that of the 1984-85 Ethiopian famine".² This claim is absurd, but, coming on the day after US Marines stormed ashore in Somalia with the stated aim of saving two million Somalis from death by starvation, it helped justify that unprecedented action, and also the continuing preoccupation with delivering food among most of the international agencies active in the country.

By December, 1992, the price of staple grain in Somalia's markets had fallen below the cost of production, and farmers were beginning to complain that the imported food aid was making it impossible for them to sell their harvests. Two million Somalis did not face death by starvation; the food scarcity was almost over. Nonetheless, relief agencies continued to open feeding centres and distribution points: the World Food Programme opened 35 distribution points in Mogadishu in February alone. Meanwhile, contrary to the warnings of Somali health professionals, the public health requirements of the country have remained largely neglected. Measles, malaria, tuberculosis, dysentery, and acute respiratory infections are still killing large numbers of Somali children. As a result, we concluded in our recent evaluation of Operation Restore Hope³ that the international intervention had done little to save lives in Somalia.

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1. Population-based mortality assessment—Baidoa and Afgoi, Somalia, 1992. *MMWR* 1992; 41: 913-17.
2. *The Guardian* London, UK, Dec 11, 1992.
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Informed consent

SIR,—Your news item on informed consent (May 1, p 1141) can be read as giving currency to the notion that there are grades of consent—consent and informed consent. There are not.

The Royal College of Physicians explains in its guidelines:¹ "Potential research subjects are entitled to choose whether or not they will participate in research, and obtaining valid (informed, understanding, voluntary) consent is central to the ethical conduct of clinical investigation. The terms 'valid', 'informed' and 'voluntary' imply that subjects have enough information, in a form that is comprehensible, to enable them to make an autonomous, deliberated (proper) judgement whether or not to participate. The word 'consent' encompasses these requirements, for if they are not met there is no consent. The use of qualifying adjectives is unnecessary and may even be confusing. Therefore the word 'consent' will be used without qualification in this document".

However, the term informed consent is now standard usage and it is a continuing reminder that participants in research trials must be given adequate information. But there is no such thing as uninformed consent. Consent is consent is consent, as the late Gertrude Stein might have said.

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1. Royal College of Physicians. *Guidelines on the practice of ethics committees in medical research involving human subjects*. London: RCP, 1990.

Future of oral iron chelator deferiprone (L1)

SIR,—The decision by Ciba-Geigy to stop development of the oral iron chelator L1 (deferiprone) has already prompted a response from the International Study Group on Oral Iron Chelators (April 24, p 1088). After the 4th International Conference on Oral Chelators (ICOC) in Cyprus on March 26-29, at which the controversy was aired, the following recommendations were made by the ICOC committee.

Some patients receive no chelation therapy, because of non-compliance with desferrioxamine or cost. Over 90% of the world's patients with thalassaemia cannot afford desferrioxamine. The options seem to be that these patients receive no treatment or that, where clinical trials are feasible with L1, these should be set up and patients recruited, with the permission of the local health authorities or, in view of the vast amount of information on this drug,^{1,2} attempts should be made to assist organisations or companies to register and use this drug under surveillance. Regularly transfused patients are at risk, in the absence of chelation therapy of irreversible organ damage due to iron overload. Marketing of L1 under controlled clinical use should be considered once preclinical and clinical data satisfy the local health authorities, and this should also apply where desferrioxamine is freely available but cannot be used because of toxicity or non-compliance.

Collected data on all aspects of L1, including chemistry, toxicology, and pharmacology, should be made available, and information on adverse effects of L1 and other iron chelators should be sent to the WHO Collaborating Centre for International Drug Monitoring, Uppsala, Sweden.

The ICOC committee took this view in the light of preclinical studies and clinical investigations in over 450 patients who have taken L1 in fifteen countries. Some patients have taken L1 daily for over 4 years, over 100 having taken it daily for more than 3 years.³ All the centres have reported that compliance with L1 is high, iron excretion is comparable with that caused by desferrioxamine, while liver iron and serum ferritin is being reduced substantially in most cases after 6 months to a year. Side-effects during the trials have usually been transient. They include 5 cases of transient agranulocytosis, transient musculoskeletal/joint pains in about 20%, gastric intolerance in 6%, and zinc deficiency in 2%. Most toxic effects were associated with daily doses of 100 mg/kg but were substantially reduced by lowering the dose to 75 mg/kg. Although a few patients have had to stop taking L1 because of side-effects no clinical trial has been suspended because of toxicity or lack of efficacy. The therapeutic margin, in animal models and in patients, does not seem to be different for L1 and desferrioxamine.