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“Human nutrition in Mongolia: maternal mortality and rickets”

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Human nutrition in Mongolia: maternal mortality and rickets

Simon S. Strickland

Reported rates of maternal mortality and childhood rickets in Mongolia are comparatively high and are a major health problem. This paper reviews recent literature on the causes of high maternal mortality and childhood rickets, and suggests that seasonally variable nutrition underlies risk factors for these conditions. These factors are likely to operate over an inter-generational time-scale.

Introduction

The purpose of this paper is to review the nutritional context of two current health problems reported for Mongolia. These are high maternal mortality rates and prevalences of childhood rickets. Explanatory hypotheses which can be tested by future research are proposed.

The general problem of maternal mortality

Maternal mortality is a significant but hitherto comparatively neglected subject of research in the developing world (Maine 1991). Incidences have been reported to vary between 55 and 700 deaths per 100,000 livebirths (Kwast 1989), with isolated reports of around 1,000/100,000 (Graham *et al.* 1989). The five major direct causes are obstructed labour, postpartum haemorrhage, hypertension, sepsis, and complications following abortion (Rosenfield 1989). Generally seen as contributory factors are age, parity, birth interval, accessibility of antenatal care, and socio-economic status (Kwast 1989). An analytical framework integrating these clinical, demographic and socio-economic variables has recently been proposed (McCarthy & Maine 1992), but remains untested under field conditions.

In Mongolia, maternal mortality ratios ranged between 120 and 204/100,000 over the period 1985-1992, peaking in 1992. Up until and including 1991, maternal death statistics did not include deaths to women during pregnancy before 28 weeks' gestation. The substantial increase in 1992 is therefore largely due to a change in classification in order to include these deaths (Randall, pers. comm. and this volume). If deaths of women while pregnant before 28 weeks' gestation are excluded from the 1992 data, the rate for that year (159/100,000) reflects a rise over the preceding five years to a level comparable with that of 1985-86. While moderate compared to many other regions of the developing world, these levels are nevertheless 3-5 times the 40/100,000 reported for other Central Asian Republics (Kazakhstan, Kirghizstan, Tajikistan, Turkmenistan, and Uzbekistan) (Chen *et al.* 1992), which approach those characteristic of western industrialised countries (30/100,000; Kwast 1989). The little time-series evidence available suggests that major falls of 50-80 percent in the maternal mortality ratio (MMR) can be achieved by improving access to antenatal and perinatal care, for example by training of traditional birth attendants, as in Taiwan (Kwast 1989) and Nigeria (Harrison 1989).

Mongolia has until now maintained what is by international standards an ex-

ceptionally high degree of health service coverage, with 1 physician per 357 head of population (Neupert 1992), and an estimated 95 percent of births occurring in clinics. These facilities have included maternity waiting homes, which elsewhere have since the nineteenth century been among the more successful means of improving maternal outcome but have been systematically supported by few countries (Harrison 1989). Recent economic upheavals resulting from the collapse of the Soviet Union have placed Mongolian health services under increasing pressure, at a time when families in many areas are being forced back onto self-provisioning (Mearns 1993). A rise in the number of home deliveries is thought to have contributed to the recent increase in MMR in 1992, but cannot explain the formerly high levels.

Nutrition and maternal mortality in Mongolia

The major proximate causes of maternal mortality in Mongolia are reported to be haemorrhage (32 percent), sepsis and uterine rupture (26 percent), toxæmia (21 percent), and 'medical complications' (21 percent) (Ministry of Health 1992). The incidences of maternal deaths associated with haemorrhage and toxæmia are higher than those reported for developing countries on average (28 percent and 17 percent respectively; Kwast 1989). However, the available Mongolian data are not presented in a way which permits separation of hypertension from other medical complications of pregnancy.

There are several ways in which nutrition may influence maternal mortality risk. In general, childbearing before cessation of pelvic growth carries risks of cephalo-pelvic disproportion (CPD) (Rosenfield 1989). In Mongolia, CPD may also be related to pelvic distortion resulting from childhood rickets, which is reported to occur in 43–47 percent of children aged 6–48 months (UNICEF/Ministry of Health 1992) and is

considered in more detail below. Relationships of CPD with short stature have been reported elsewhere (Sokal *et al.* 1991, Kwawukume *et al.* 1993). More generally, relationships between stature and fertility have been found in both sexes in a number of populations of poorer and richer parts of the world, but their interaction with socioeconomic status has not been systematically investigated (Eveleth 1985, Ulijaszek and Strickland 1993).

Large birthweight babies (macrosomia) may also be a source of CPD in Mongolia. The prevalence of low birth weights (< 2.5 kg) was reported to be 4.5 percent in 1990 (Ministry of Health 1992). This is a comparatively low figure compared to adequately nourished women ($W/H^2 \geq 18.5 \text{ kg/m}^2$) in the developing world where values of around 5 percent would be expected (Kusin *et al.* 1993). In a number of studies, birthweights exceeding 4 kg have been associated with maternal obesity and impaired glucose tolerance ('gestational diabetes'; Jarrett 1993), major obstetric haemorrhage (Stones *et al.* 1993), and with consequential high risk of maternal mortality in some developing countries (Megafu and Ozumba 1988, Thonneau *et al.* 1992). Although there are no published reports of non-insulin dependent diabetes mellitus (NIDDM) in Mongolia, undiagnosed gestational diabetes in association with obesity may be implicated in high Mongolian levels of maternal mortality.

Obesity in Mongolian nomads exhibits a centripetal distribution of fat which is probably related to seasonal climatic extremes and variation in food supplies (Beall and Goldstein 1992). In rural areas, body weight and fatness could be expected to show periodic cycling with rises in the summer months, when food resources are relatively diverse and accessible, and declines in winter and spring when pastoralist families are forced to depend on their livestock holdings as the sole or principal source of subsistence. Mongolia has started to undergo a transition from a socialist 'command' economy to a 'free market' economy,

and the amplitude of seasonal fluctuations in food supply can be expected to increase, particularly among those whose circumstances have worsened with the privatisation of pastoral collectives.

In some recent research studies, periodic weight cycling has been associated with increased body fat deposition (Saris 1989), and greater energy efficiency during exercise in the non-obese (Manore *et al.* 1991). Instability of energy intake may induce hormonal changes which raise efficiency of energy turnover in the active tissue mass (Shetty 1990). However, reports of reduced resting metabolic rate (RMR) in weight cycling individuals (Brownell *et al.* 1986, Steen *et al.* 1988) have not always been substantiated in the experimental literature (Melby *et al.* 1990, Manore *et al.* 1991, Schmidt *et al.* 1993). When there is frequent uncertainty of food supply, mechanisms enhancing energetic efficiency may be an appropriate evolutionary adaptation. Weight cycling in affluent countries, however, is also related to obesity and increased risk of coronary heart disease (CHD) (Saris 1989, Garrow 1992). In Mongolia, the incidence of CHD has risen 2.5-fold between 1986 and 1991, although to a greater extent in urban than rural areas (Strickland 1993), suggesting that there may be common factors underlying this of which the implications for maternal health in Mongolia need to be explored.

The prevalence of hypertension in Mongolia also appears to have risen, doubling over the period 1986–1991 and with particularly high increases of 4 to 11-fold in the cities (Strickland 1993). Hypertension during pregnancy is one of the major risk factors for maternal mortality in the developing world (Kwast 1989). It could be speculated that this is linked to insulin resistance, macrosomia and CPD-related maternal mortality.

Insulin promotes sodium retention, and insulin resistance and hyperinsulinaemia may contribute to the pathogenesis of hypertension (Reaven 1988). However, correlations between hypertension and insulin

resistance tend to disappear when body weight and fatness or fat distribution have been taken into account, and there is little substantive evidence for a mechanism linking insulin resistance directly to hypertension and obesity (Jarrett 1992). There may be effects of the foetal nutritional environment. Recent studies indicate that raised blood pressure in adults is inversely related to birth weight, suggesting that hypertension is initiated during foetal life and is amplified with ageing and obesity thereafter (Law *et al.* 1993). However, the theory that intrauterine growth retardation provides a mechanism linking hypertension, non-insulin dependent diabetes and impaired glucose tolerance in adulthood (Hales *et al.* 1991) remains controversial and in need of further research (Cook *et al.* 1993).

The role of the current Mongolian dietary pattern in maternal mortality is unclear. There are few quantitative data on Mongolian diets, but macronutrient intakes appear at present to be seasonally or chronically high in protein and fat (Strickland 1993). Qualitative information suggests that, with recent economic liberalisation emerging since 1989, there has been greater dependence on meat and dairy produce as the main sources of energy and nutrients throughout the year (Cooper and Gelezhamstin 1993). Recent experimental studies on rodents indicate that low protein diets raise risks of hypertension in later life (Langley and Jackson 1993 *in press*). It is possible that the causes of current patterns of nutritional disease and maternal mortality therefore need to be sought in changes in diet following the expansion of Soviet-subsidised wheat farming in Mongolia, which dates from the 'Virgin Lands' programme of the mid-1950s (Chalmers 1993) and which has now begun to decline.

These considerations underlie the need to explore the theory that, in Mongolian women of reproductive age, body fatness, insulin resistance, high protein diets, and weight cycling together conspire to raise risk of maternal mortality in this population.

Table 1. *Prevalence of rachitic signs by area and age group.*

	Prevalence (percent)	Range (percent)
Cities	46.8	42.4-68.4
Provinces	43.4	38.7-48.6
Age (mo) <6	34.8	
7-12	48.5	
13-24	54.8	
25-48	38.2	

Source: UNICEF/Ministry of Health 1992.

Rickets

Childhood rickets is thought to be a major problem, primarily affecting those under 3 years' age. Estimates of the prevalence in the whole state were 8.0 percent in 1985 and 7.0 percent in 1988, figures which were based on samples of around 60,000 infants (FAO 1992). Table 1 shows the findings of the most recent sample survey. There are slightly higher rates in cities than in provincial areas. The prevalence peaks in the second year of life. Rickets in girls may result in pelvic deformities which later cause difficulties in childbirth, and thus contribute to the high maternal mortality rate.

The diagnostic criteria on which these prevalence data were based are shown in Table 2. A large proportion of the rachitic children exhibited bowed legs, the remainder craniotabes with or without leg deformities. However, these diagnostic criteria are far from perfect. For example, in a recent study of Egyptian children aged below 3 y whose rachitic state was identified radiologically by poor skeletal mineralization and by low plasma concentrations of vita-

min D, leg deformities identified only 30 percent of subjects; 4 percent of control subjects without rickets also showed such deformities; and none of the rachitic subjects presented with craniotabes (Lawson *et al.* 1987). Convincing estimates of the prevalence of this condition in Mongolia must therefore remain a matter of further work.

However, it is possible to speculate about the causes of rickets in Mongolia. There are two main routes of availability of the principal circulating form of vitamin D (25-hydroxycholecalciferol, calcidiol): the oral route; and, endogenous synthesis in the skin by the action of UV light on the precursor 7-dehydrocholesterol. Endogenous vitamin D diffuses slowly into the blood and is taken up by the liver less rapidly than the oral form, which appears to be preferentially lost by excretion in bile. In contrast to dietary vitamin D, the endogenous supply is continuous, prolonged and the most efficient means of supply (Fraser 1983). Plasma calcidiol varies with seasonal intensity of sunlight independently of dietary vitamin D (Cockburn *et al.* 1980), and is influenced

Table 2. *Prevalence of rachitic signs in pre-school children*

Sign	Total prevalence (percent)	N
Craniotabes	11.7	197
Leg deformities	21.7	380
Both combined	10.3	172
Total	43.7	749

Source: derived from data in UNICEF/Ministry of Health 1992.

by skin pigmentation (Clemens *et al.* 1982, Fairney *et al.* 1987, Meulenmeester *et al.* 1990). Extensive irradiation can raise plasma calcidiol to the level of 80 ng/ml, which is harmless; but high oral doses (> 250 g/d, occasionally > 45 g/d) can lead to levels of >400 ng/ml and toxicity shown by hypercalcaemia (Bender 1992). Where the intensity of sunlight falls below about 20 mJ/cm², beyond a latitude of about 40° N or S, there is unlikely to be significant cutaneous synthesis of calcidiol during the winter months of the year. This factor, together with an age-dependent decline in 7-dehydrocholesterol in the skin during adulthood, combine to raise risk of a seasonal pattern of vitamin D deficiency particularly in the elderly (*ibid.*).

Production of the biologically most active form (1,25-dihydroxycholecalciferol, calcitriol) from calcidiol in the kidney is stimulated by a rise in circulating parathyroid hormone which results when the concentration of calcium in the extra-cellular fluid falls below about 10 mg/100 ml (Deluca 1982). Parathyroid hormone, calcitriol and calcitonin regulate calcium homeostasis, keeping plasma calcium within the narrow range of 2.2–2.55 mmol/L by promoting absorption of calcium from the gut and bony tissues (Bender 1992). Hence bone demineralization (rickets in children, osteomalacia in the ageing) is associated with depleted calcidiol, though low levels of this form do not always in themselves indicate inadequate vitamin D status (Lawson *et al.* 1979). Vitamin D is therefore more a hormone, regulating homeostasis of calcium, than a micro-nutrient. However, it is thought to act in about 30 target tissues (Norman and Lowe 1992); and it is known to influence patterns of insulin secretion, the synthesis and release of thyroid hormones, the proliferation, differentiation and immune function of lymphocytes and monocytes, and a number of other cell types (Bender 1992). A relationship between vitamin D status, reported high prevalence rates of iodine deficiency disorders (palpable goitres) (Gunsendorj

and Oyunbileg 1992) and susceptibility to infection cannot therefore be ruled out.

Hypocalcaemia is likely to be present in more advanced stages of vitamin D deficiency (Cockburn *et al.* 1980, Gupta *et al.* 1974). This may result from low calcium intakes or from high dietary phytate, which chelates calcium and reduce its bio-availability. A mechanism has been proposed to explain this through the consequent hepatic inactivation and degradation of calcidiol which has been shown experimentally to induce deficiency states (Clements *et al.* 1987). However, some studies of the aetiology of rickets have failed to show clear evidence of phytate-effects (O'Hara-May and Widdowson 1974, Lawson *et al.* 1987), so there may be considerable interindividual (? genetic) variation in calcium requirements.

The apparently high prevalence of rickets in Mongolia has been thought to result from low calcium in breast milk and tight infant swaddling (Kachondam 1993). A fear of exposing small children to winds may also limit exposure to sunlight (Randall, personal communication), suggesting a cultural dimension which needs further study. These considerations suggest a seasonal pattern in the aetiology of childhood rickets. Season of birth could be important, since infant vitamin D derives largely from placental transfer (Fraser 1983, Fairney *et al.* 1987), and maternal stores may be depleted in winter. Some studies have found that breast milk does, however, protect against neonatal hypocalcaemia and that vitamin D supplementation may offset hypocalcaemic effects of artificial feeds (Cockburn *et al.* 1980). In Mongolia, heightened risk of rickets could also be related to time of birth and subsequent likelihood of exposure to adequate UV-radiation. Analysis of future data on rickets in Mongolia will need to take seasonality into account.

It has been argued that a diet containing substantial quantities of whole milk may reduce calcium availability in populations exhibiting high frequencies of lactose intolerance, and therefore promote the spread

of genotypes favouring persistence of lactase into adulthood in high latitude populations at risk of rickets (Durham 1990:279). Mongolians (including Kazakhs) appear to show frequencies of the gene for persistence of high intestinal lactase of about 0.062–0.126, among the lowest in the world (Roberts 1985). However, they maintain high intakes of dairy products, and it could be surmised that this factor is significant in the aetiology of rickets. However, experimental studies indicate conflicting evidence: some report no adverse effects of lactose intolerance on calcium absorption (Leichter 1981, Torun *et al.* 1984), or report inconsistent findings (Phillips 1981, Scrimshaw and Murray 1988), suggesting the need for further investigation of this hypothesis.

The Mongolian data on dietary intakes of calcium are limited, although extensive use of dairy produce would make low calcium intakes unlikely. It has been estimated that pregnant women consume 626 ± 18 mg/d, children under 3 y 122 mg/d (Ministry of Health, unpublished data from 1978–1987). Estimated requirements in the West are of the order of 400–600 mg/d in children and adults, and 1000–1200 mg/d in pregnant and lactating women. Some populations nevertheless subsist apparently adequately on intakes of 200–300 mg/d in Sri Lanka and regions of Africa; and this is approximately the level of calcium intake from milk in healthy, exclusively breast-feeding babies under 6 months' age (Davidson *et al.* 1979). The age pattern of Mongolian rickets suggests that the peak occurs during the second year of life, after which it diminishes. Lactase is expected to decline over several years after birth. For example, one American study reported prevalences of lactose malabsorption of 27 percent, 33 percent and 74 percent respectively in age-groups 1–2 y, 5–6 y, and 11–12 y, and found a threshold age of 8 years after which the blood glucose response to a lactose load fell below that of younger children (Paige 1981). The population in this case may have had a PLA gene frequency

of about 0.116 (Roberts 1985), and similar to that of Mongolians. However, the physiological challenge dose in such studies is 2 g lactose per kg body weight, consumed after an overnight fast; and effects tend to be mitigated or eliminated when whole milk or other items are consumed with lactose. In addition, the effect of Mongolian milk processing techniques on lactose is unclear. In general, therefore, an explanation for Mongolian rickets based on the consequences of lactose intolerance seems implausible.

An alternative hypothesis is that Mongolian diets containing high concentrations of protein, whether seasonally or throughout the year, may contribute to poor calcium availability through calciuretic effects of this nutrient (Margen *et al.* 1974, Hegsted and Linkswiler 1981). However, studies on purified protein diets ignore the effect of phosphorus, which promotes calcium absorption and is present in meat-containing diets. Thus, even if families are forced to depend increasingly on highly proteinaceous diets of meat and the products of milk from their livestock throughout the year, this factor is unlikely to explain the prevalence of rickets.

Of these three hypotheses, the most convincing is that of seasonally poor exposure to UV-radiation.

Concluding remarks

Where childhood rickets results in pelvic deformity, it is plausible to suppose that the problems of maternal mortality and rickets are connected. However, as this paper illustrates, understanding nutritional factors contributing to these health problems will require substantive cross-disciplinary research.

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