Birth weight and non-insulin dependent diabetes: thrifty genotype, thrifty phenotype, or surviving small baby genotype?

David R McCance, David J Pettitt, Robert L Hanson, Lennart T H Jacobsson, William C Knowler, Peter H Bennett

Abstract

***Objective*—To determine the prevalence of diabetes in relation to birth weight in Pima Indians.**

***Design*—Follow up study of infants born during 1940-72 who had undergone a glucose tolerance test at ages 20-39 years.**

***Setting*—Gila River Indian community, Arizona.**

***Subjects*—1179 American Indians.**

***Main outcome measure*—Prevalence of non-insulin dependent diabetes mellitus (plasma glucose concentration ≥11·1 mmol/l two hours after ingestion of carbohydrate).**

***Results*—The prevalence was greatest in those with the lowest and highest birth weights. The age adjusted prevalences for birth weights <2500 g, 2500-4499 g, and ≥4500 g were 30%, 17%, and 32%, respectively. When age, sex, body mass index, maternal diabetes during pregnancy, and birth year were controlled for, subjects with birth weights <2500 g had a higher rate than those with weights 2500-4499 g (odds ratio 3·81; 95% confidence interval 1·70 to 8·52). The risk for subsequent diabetes among higher birthweight infants (≥4500 g) was associated with maternal diabetes during pregnancy. Most diabetes, however, occurred in subjects with intermediate birth weights (2500-4500 g).**

***Conclusions*—The relation of the prevalence of diabetes to birth weight in the Pima Indians is U shaped and is related to parental diabetes. Low birth weight is associated with non-insulin dependent diabetes. Given the high mortality of low birthweight infants selective survival in infancy of those genetically predisposed to insulin resistance and diabetes provides an explanation for the observed relation between low birth weight and diabetes and the high prevalence of diabetes in many populations.**

Diabetes and Arthritis Epidemiology Section, Phoenix Epidemiology and Clinical Research Branch, National Institute of Diabetes and Digestive and Kidney Diseases, Phoenix, Arizona 85014, United States

David R McCance, *visiting associate*
David J Pettitt, *assistant chief of section*
Robert L Hanson, *medical staff fellow*
Lennart T H Jacobsson, *visiting associate*
William C Knowler, *section chief*
Peter H Bennett, *branch chief*

Correspondence to:
Dr D R McCance, Sir George E Clark Metabolic Unit, Royal Victoria Hospital, Belfast BT12 6BA.

BMJ 1994;308:942-5

Introduction

Recent findings have suggested that low birth weight and weight at 1 year are related to the development of glucose intolerance.[1-3] In men aged 64 years in Hertfordshire, England, the prevalence of non-insulin dependent diabetes or impaired glucose tolerance fell progressively from 40% in those subjects who weighed 2500 g or less at birth to 14% in those whose birth weight was 4310 g or above.[1] Subsequently a similar inverse association between birth weight and plasma glucose concentration 30 minutes after carbohydrate ingestion was reported for men aged 18-25 years,[2] and between birth weight and either impaired glucose tolerance or diabetes in subjects of both sexes aged 50 years.[3] The trends seemed to be independent of current body mass index and social class. These findings have been interpreted as long term effects of nutritional factors which reduce fetal and infant growth and impair the development of the endocrine pancreas and other tissues.[4]

The Pima Indian residents of the Gila River Indian community in Arizona have the highest reported prevalence of non-insulin dependent diabetes, which often has its onset at an early age.[5-7] The importance of maternal glucose intolerance on the intrauterine environment, in addition to the effects of genetic factors, for increased birth weight and subsequent diabetes in this population has been shown previously.[8-10] We examined the relation between birth weight and subsequent glucose tolerance, how this is influenced by parental diabetes, and whether low birth weight is predictive of non-insulin dependent diabetes in this population.

Subjects and methods

A longitudinal study of diabetes and its complications has been conducted among the American Indian population of the Gila River Indian community in Arizona.[11] This analysis includes 1179 subjects born between 1940 and 1972 with recorded birth weight, whose heritage was at least half Pima or Tohono O'odham (Papago) or a mixture of these two closely related tribes, who were singleton births, and whose glucose tolerance was evaluated at ages 20-39 years.

For those born before 1965 birth weights were obtained from the medical records of the babies of all women aged 25-44 years who had participated in a population based epidemiological study between 1 March 1965 and 1 March 1967.[12] From 1965-72 birth weights were collected prospectively as part of an ongoing population based study of pregnancy.

About every two years all residents of the community aged 5 years and over are asked to participate in a standardised medical examination which includes the determination of venous plasma glucose concentration two hours after ingesting 75 g carbohydrate. Diabetes was diagnosed according to World Health Organisation criteria if the plasma glucose concentration two hours after ingestion was at least 11·1 mmol/l[13] at a survey examination or if a casual plasma glucose concentration ≥11·1 mmol/l was observed in the course of routine medical care. Blood pressure was measured in supine subjects with a large adult cuff and a mercury sphygmomanometer (W A Baum, Copiaque, New York). Korotkoff sounds phase I and IV were used for systolic and diastolic pressures, respectively. Plasma glucose concentration was estimated by an Autoanalyzer using the potassium ferricyanide method (Technicon Instruments Corporation, Tarrytown, New York).

As parental diabetes is known to be associated with the development of diabetes in young adults and diabetes or glucose intolerance in pregnancy is a risk factor for both high birth weight and diabetes in this population,[8 10-14] the association of birth weight and diabetes was also examined in relation to the presence or absence of parental diabetes and among subjects whose mothers were known not to have diabetes during the pregnancy. Parental diabetes at the latest examination was defined as absent (neither parent diabetic) or present in at least one parent.

The significance of differences in prevalence of diabetes and abnormal glucose tolerance according to age and birth weight was analysed by a Mantel-Haenszel χ^2 test and logistic regression analysis. Age

adjustment was made either by the direct method using the age distribution of the 1179 subjects at the time of their glucose tolerance assessment as the reference population (see table II) or by logistic regression using a series of indicator variables for the various categories of birth weight and body mass index for offspring aged 30 years (see table III). As the results did not vary by sex of the offspring the sexes were combined. There were no significant interactions (product terms) in the model. For comparability with previous studies the results presented are confined to singleton pregnancies, and eight sets of twins and one set of triplets were excluded. Inclusion of these multiple births in the analysis, however, did not alter the findings.

Results

Of 2512 singleton births recorded in 1940-72 birth weight was known in 2028, 1179 of whom subsequently had at least one glucose tolerance test when aged 20-39 years. Table I shows the prevalences of abnormal glucose tolerance (plasma glucose concentration at two hours $\geq 7{\cdot}8$ mmol/l) and diabetes according to age group and birth weight. Table II shows the age adjusted prevalence of diabetes. The prevalence of diabetes showed a U shaped relation with birth weight, with the highest rates in those with the highest and those with the lowest birth weights. The age adjusted prevalences of diabetes for birth weights <2500 g, 2500-2999 g, 3000-3499 g, 3500-3999 g, 4000-4499 g, and ≥ 4500 g were 30%, 18%, 16%, 17%, 18%, and 32%, respectively. When examined as a continuous variable in a logistic regression model, age, concurrent body mass index, birth weight, and the quadratic term of birth weight (to allow for a parabolic or U shaped relation) contributed significantly to the prediction of the prevalence of diabetes ($P<0{\cdot}05$).

TABLE I—*Prevalences of diabetes and abnormal glucose tolerance according to age group and category of birth weight in 1179 Pima Indians at last biennial examination*

Age group (years)	No (%) within birth weight groups (g)			
	<2500	2500-3499	3500-4499	≥4500
	Plasma glucose concentration at two hours ≥11·1 mmol/l			
20-24	3/25 (12·0)	18/272 (6·6)	10/173 (5·8)	3/12 (25·0)
25-29	3/10 (30·0)	27/162 (16·7)	20/129 (15·5)	3/11 (27·3)
30-34	3/6 (50·0)	35/145 (24·1)	26/95 (27·4)	3/7 (42·9)
35-39	3/5 (60·0)	32/82 (39·0)	20/43 (46·5)	1/2 (50·0)
Total	12/46 (26·1)	112/661(16·9)	76/440 (17·3)	10/32 (31·3)
	Plasma glucose concentration at two hours ≥7·8 mmol/l			
20-24	7/25 (28·5)	45/272 (16·5)	24/173 (13·9)	5/12 (41·7)
25-29	3/10 (30·0)	56/162 (34·6)	34/129 (26·4)	3/11 (27·3)
30-34	5/6 (83·3)	58/145 (40·0)	46/95 (48·4)	3/7 (42·9)
35-39	4/5 (80·0)	46/82 (56·1)	29/43 (67·4)	1/2 (50·0)
Total	19/46 (41·3)	205/661 (31·0)	133/440 (30·2)	12/32 (37·5)

TABLE II—*Age adjusted prevalences (68% confidence intervals) of diabetes according to birth weight among 1179 Pima Indians aged 20-39 years. Age adjusted by direct method with age distribution of all subjects as reference population*

Birth weight (g)	Age adjusted prevalence (68% confidence interval)
<2500	30·2 (23·4 to 37·0)
2500-2999	18·4 (15·6 to 21·2)
3000-3499	16·3 (14·6 to 17·9)
3500-3999	17·1 (15·3 to 19·0)
4000-4499	18·2 (13·9 to 22·5)
≥4500	32·2 (23·8 to 40·6)

As the prevalence of diabetes is related to concurrent obesity, the association with low birth weight was examined according to obesity. Within each third of body mass index (weight (kg)/(height (m))^2) the highest age adjusted prevalences in general occurred in subjects with the lowest and in those with the highest birth weights. The effect of birth weight on the subsequent prevalence, however, was most evident in the lower third of body mass index ($<29{\cdot}8$; table III).

Among subjects with at least one diabetic parent, the age adjusted prevalences for birth weights <2500 g, 2500-4499 g, and ≥ 4500 g were 38%, 20%, and 35%, respectively. By contrast, diabetes was present in only four of the 155 subjects with two non-diabetic parents, and thus the effect of birth weight on the prevalence in this group could not be assessed. Nevertheless, this illustrates that the relation of low birth weight and subsequent diabetes is seen primarily in subjects with a parental history of diabetes.

TABLE III—*Age-adjusted prevalences (68% confidence intervals) of diabetes according to birth weight and category of current body mass index among 1179 Pima Indians. Age adjusted to a mean age of 30 years by using logistic regression*

Body mass index (kg/m^2)	Birth weight (g)	Age adjusted prevalence (68% confidence interval)
Lowest third	<2500	35·4 (23·2 to 49·9)
	2500-3499	13·0 (10·7 to 15·7)
	3500-4499	12·2 (9·3 to 15·8)
	≥4500	52·4 (36·1 to 68·2)
Middle third	<2500	35·6 (23·0 to 50·6)
	2500-3499	18·6 (16·0 to 21·5)
	3500-4499	21·4 (17·8 to 25·6)
	<4500	22·7 (8·8 to 47·0)
Upper third	<2500	32·6 (19·5 to 49·5)
	2500-3499	25·8 (22·5 to 29·3)
	3500-4499	23·4 (20·1 to 27·1)
	≥4500	36·0 (23·8 to 50·2)

TABLE IV—*Multiple logistic regression analysis for diabetes in 1179 Pima Indians controlled for birth weight, age, body mass index, maternal diabetes during pregnancy, and birth year*

Variable	Odds ratio (95% confidence interval)	P value
Age (per 10 years)	3·08 (1·96 to 4·86)	0·0001
Body mass index	1·06 (1·04 to 1·09)	0·0001
Low birth weight* (low/normal)	3·81 (1·70 to 8·52)	0·001
High birth weight* (high/normal)	1·80 (0·63 to 5·10)	0·269
Maternal diabetes during pregnancy (yes/no)	10·73 (4·76 to 24·17)	0·0001
Birth year (per year)	0·94 (0·90 to 0·97)	0·001

*Low birthweight and high birthweight variables compared weights below 2500 g and above 4500 g, respectively, with those 2500-4499 g. Sex was not significant and therefore was not included as a covariate.

As diabetes in pregnancy is an established risk factor for both high birth weight and diabetes in this population,[8 10-14] the relation between prevalence and birth weight was examined in offspring of women who did not have diabetes during pregnancy. The age adjusted prevalence in this group of 903 subjects according to birth weights <2500 g, 2500-4499 g, and ≥ 4500 g was 34%, 15%, and 20%, respectively. A birth weight of <2500 g was significantly associated with a higher prevalence (age adjusted Mantel-Haenszel odds ratio 3·37; 95% confidence interval 1·58 to 7·14; $P=0{\cdot}002$) than a birth weight of 2500-4499 g. Exclusion of subjects whose mothers may have had diabetes in pregnancy clearly reduced the proportion with diabetes in the highest birthweight category (≥ 4500 g).

The independent association of birth weight and diabetes was also examined in all 1179 subjects by using logistic regression analysis. When age, sex, body mass index, maternal diabetes during pregnancy, and birth year were controlled for by logistic regression analysis, subjects with low birth weights (<2500 g) were significantly more likely to have diabetes than those with birth weights of 2500-4499 g (3·81; 1·70 to 8·52; table IV). The risk of diabetes in subjects with high birth weights (≥ 4500 g) seems to be attributable primarily to maternal diabetes during pregnancy because after we included diabetes in pregnancy in the model the association of high birth weight with diabetes was no longer significant, but the strong significant relation between low birth weight and subsequent diabetes remained.

Mean systolic and diastolic blood pressure did not differ significantly among any of the birthweight categories.

Discussion

In Pima Indian adults, as in British populations,[1-3] low birth weight is associated with an increased prevalence of non-insulin dependent diabetes. In the

Pima this relation is evident in subjects as young as 20-30 years of age. In contrast with the reports of Hales and Barker and their colleagues, which showed much higher rates of diabetes in low than in high birthweight subjects,[1-3] the overall relation of glucose tolerance with birth weight in Pima Indians is U shaped, with higher rates of diabetes in those with high as well as in those with low birth weights.

The increased risk of diabetes among Pima Indians with high birth weight can largely be explained by the presence of maternal diabetes during pregnancy,[8 9] perhaps mediated by metabolic fuel abnormalities in utero.[15] We have shown previously how this effect may give rise to an increasing prevalence of diabetes in successive generations.[8] Exclusion of offspring of mothers who developed diabetes during pregnancy or controlling for maternal diabetes in pregnancy reduced the prevalence of diabetes in subjects with birth weights of $\geq$4500 g to levels similar to those seen in subjects with normal birth weights. The lack of an association between high birth weight and glucose intolerance in British studies probably reflects a lower prevalence of diabetes during pregnancy.

Although our study is a longitudinal population based study, birth weights were known for only 81% of the subjects born between 1940 and 1972, and only 58% of these had had glucose tolerance tests at ages 20-39 years. Most of the others had received glucose tolerance tests but had not been examined at 20-39 years of age and were therefore not considered in the present analysis. Unless the birth weights of low birthweight infants destined to develop diabetes 20 or more years later were selectively recorded, it seems unlikely that incomplete ascertainment would influence our results. Other possible sources of bias are temporal trends of socioeconomic status, health care, and an increasing prevalence of diabetes, but when we controlled for year of birth with logistic regression low birth weight was still independently associated with a higher prevalence of diabetes. Furthermore, the relation of birth weight to prevalence of diabetes remains after current body mass index was controlled for and are seen primarily among subjects with at least one parent with diabetes.

THRIFTY GENOTYPE, THRIFTY PHENOTYPE

In 1962, as an explanation for the high prevalence of diabetes, Neel hypothesised the evolution of a "thrifty genotype" which resulted in selective survival advantage in times of fluctuating feast and famine by allowing storage of calories in times of plenty.[16] He argued that such a thrifty genotype became detrimental when food supplies were constant and abundant and led to an increased prevalence of obesity and non-insulin dependent diabetes in certain populations such as the Pima.[17 18] Recently Hales and Barker have hypothesised that low birth weight, which is a reflection of nutritional deprivation in utero[19] and which impairs the development of the fetal pancreas,[20-23] predisposes to diabetes in later life.[4] They have suggested that non-insulin dependent diabetes is mainly the result of environmental factors and that genetic factors play little or no role in its development and called this the "thrifty phenotype" hypothesis.[4] In their studies of birth weight, as in ours, follow up into adulthood is of necessity limited to survivors, and their hypothesis takes no account of the high mortality associated with low birth weight.

In spite of the excess of diabetes associated with low and high birth weight in the Pima, only 6% who developed diabetes by 20-39 years of age had a birth weight $<$2500 g; a similar proportion had birth weights $\geq$4500 g. Consequently, most Pimas who develop diabetes have birth weights that fall within the usual range. Thus, the high prevalence of diabetes in this population cannot reasonably be attributed to nutritional deficiencies in utero of the type that led to low birth weight, and other explanations for the relation of diabetes to birth weight and the high prevalence of the disease in the Pima and other populations must be sought.

Clinical implications

- Low birth weight has been shown to predict subsequent diabetes in several British populations and interpreted as a reflection of nutritional deprivation in utero
- This study showed that in Pima Indians aged 20-39 years the association of birth weight and diabetes was U shaped, with the highest prevalence of diabetes occurring in both high and low birthweight infants
- The association of diabetes with high birth weight is largely explained by the presence of maternal diabetes during pregnancy
- Despite the excess of diabetes associated with low birth weight this accounted for only 6% of diabetes in this population
- An alternative hypothesis is proposed—namely, that the association of diabetes and low birth weight may reflect selective survival of small infants genetically predisposed to diabetes and other insulin resistance syndromes

SURVIVING SMALL BABY GENOTYPE

As an alternative we propose that the increase in prevalence of diabetes among subjects with low birth weight, now shown in several studies, could reflect the selective survival of low birthweight infants genetically susceptible to developing diabetes. As insulin resistance seems to be an important abnormality preceding non-insulin dependent diabetes in the Pima Indians[24 25] and in other populations[26] low (and high) birth weight might be expected to be associated with insulin resistance. We propose therefore that genetic predisposition to insulin resistance may represent the mechanism which facilitates such selective survival advantage and over many generations ultimately leads to high prevalences of diabetes and other manifestations of insulin resistance in contemporary populations.

There are several studies consistent with this hypothesis. Recently Barker *et al* have shown a high prevalence of the insulin resistance syndrome in adults who had been low birthweight infants.[27] In addition, highly significant inverse relations between birth weight and insulin concentrations after fasting and two hours after carbohydrate ingestion, blood pressure, truncal obesity, and an index of the insulin resistance syndrome have been reported in Mexican-Americans and non-Hispanic white people from San Antonio.[28] Thus, several characteristics related to insulin resistance, besides diabetes and glucose intolerance, are found more often in subjects with low birth weights.

CONCLUSIONS

The present study in Pima Indians provides further evidence for an association between low birth weight and glucose tolerance. Unlike the findings of previous British studies, however, the association of birth weight and diabetes is U shaped, with a higher prevalence of diabetes occurring in subjects with both low and high birth weights. Given the high mortality among low birthweight infants and that most diabetes occurs in Pima Indians with birth weights in the usual range, we propose that the excess of diabetes seen in subjects with low birth weight in the Pima and other

populations may reflect the selective survival of infants genetically susceptible to insulin resistance and subsequently to developing non-insulin dependent diabetes.

We thank the members of the Gila River Indian community for taking part in the study and the staff of the diabetes and arthritis epidemiology section for performing the clinical examinations and laboratory tests.

1 Hales CN, Barker DJP, Clark PMS, Cox LJ, Fall C, Osmond C, *et al.* Fetal growth and impaired glucose tolerance at age 64. *BMJ* 1991;303:1019-22.
2 Robinson S, Walton RJ, Clark PM, Barker DJP, Hales CN, Osmond C. The relation of fetal growth to plasma glucose in young men. *Diabetologia* 1992;35:444-6.
3 Phipps K, Barker DJP, Hales CN, Fall CHD, Osmond C, Clark PMS. Fetal growth and impaired glucose tolerance in men and women. *Diabetologia* 1993;36:225-8.
4 Hales CN, Barker DJP. Type 2 (non-insulin-dependent) diabetes mellitus: the thrifty phenotype hypothesis. *Diabetologia* 1992;35:595-601.
5 Knowler WC, Bennett PH, Hamman RF, Miller M. Diabetes incidence and prevalence in Pima Indians: a 19-fold greater incidence than in Rochester, Minnesota. *Am J Epidemiol* 1978;108:497-505.
6 Knowler WC, Pettitt DJ, Saad MF, Bennett PH. Diabetes mellitus in the Pima Indians: incidence, risk factors and pathogenesis. *Diabetes Metab Rev* 1990;6:1-27.
7 Savage PJ, Bennett PH, Senter RG, Miller M. High prevalence of diabetes in young Pima Indians: evidence of phenotypic variation in a genetically isolated population. *Diabetes* 1979;28:939-42.
8 Pettitt DJ, Aleck KA, Baird HR, Carraher MJ, Bennett PH, Knowler WC. Congenital susceptibility to NIDDM: role of intrauterine environment. *Diabetes* 1988;37:622-8.
9 Pettitt DJ, Bennett PH, Saad MF, Charles MA, Nelson RJ, Knowler WC. Abnormal glucose tolerance during pregnancy in Pima Indian women: long-term effects on offspring. *Diabetes* 1991;40:126-30.
10 Pettitt DJ, Baird HR, Aleck KA, Bennett PH, Knowler WC. Excessive obesity in offspring of Pima Indian women with diabetes during pregnancy. *N Engl J Med* 1983;308:242-5.
11 Bennett PH, Burch TA, Miller M. Diabetes mellitus in American (Pima) Indians. *Lancet* 1971;ii:125-8.
12 Comess LJ, Bennett PH, Burch TA, Miller M. Congenital anomalies and diabetes in Pima Indians of Arizona. *Diabetes* 1969;18:471-7.
13 WHO Study Group on Diabetes Mellitus. Second report. *World Health Organ Tech Rep Ser* 1985;727:9-17.
14 Pettitt DJ, Knowler WC, Baird HR, Bennett PH. Gestational diabetes: infant and maternal complications of pregnancy in relation to third-trimester glucose tolerance in the Pima Indians. *Diabetes Care* 1980;3:458-64.
15 Frienkel N. Of pregnancy and progeny. *Diabetes* 1980;29:1023-9.
16 Neel JV. Diabetes mellitus; a thrifty genotype rendered detrimental by 'progress'? *Am J Hum Genet* 1962;14:353-62.
17 Neel JV. The thrifty genotype revisited. In: Köbberling J, Tattersall R, eds. *The genetics of diabetes mellitus. Proceedings of the Serono symposium.* London: Academic Press, 1982:283-93.
18 Thrifty genotype rendered detrimental by progress? [editorial]. *Lancet* 1989;ii: 839-40.
19 McCance RA, Widdowson EM. The determinants of fetal growth and form. *Proc R Soc Lond* 1974;185:1-17.
20 Sweene I, Bone AJ, Howell SL, Hellerström C. Effects of glucose and amino acids on the biosynthesis of DNA and insulin in fetal rats maintained in tissue culture. *Diabetes* 1981;29:686-92.
21 Sweene I, Crace CJ, Milner RDG. Persistent impairment of insulin secretory response to glucose in adult rats after limited period of protein-calorie malnutrition early in life. *Diabetes* 1987;36:454-8.
22 Van Assche FA, Aerts L. The fetal endocrine pancreas. *Contrib Gynecol Obstet* 1979;5:44-57.
23 Wilkin TJ. Early nutrition and diabetes mellitus (editorial). *BMJ* 1993;306: 283-4.
24 Lillioja S, Mott DM, Howard BV, Bennett PH, Yki-Jarvinen H, Freymond D, *et al.* Impaired glucose tolerance as a disorder of insulin action: longitudinal and cross-sectional studies in Pima Indians. *N Engl J Med* 1988;318:1217-25.
25 Lillioja S, Mott DM, Spraul M, Ferraro R, Foley JE, Ravussin E, *et al.* Insulin resistance and insulin secretory dysfunction as precursors of non-insulin-dependent diabetes mellitus: prospective studies in Pima Indians. *N Engl J Med* 1993;329:1988-92..
26 Martin BC, Warram JH, Krolewski AS, Bergman RN, Soeldner JS, Kahn CR. Role of glucose and insulin resistance in development of Type 2 diabetes mellitus: results of a 25-year follow-up study. *Lancet* 1992;340:925-9.
27 Barker DJP, Hales CN, Fall CHD, Osmond C, Phipps K, Clark PMS. Type 2 (non-insulin-dependent) diabetes mellitus, hypertension and hyperlipidaemia (syndrome X): relation to fetal growth. *Diabetologia* 1993;36:62-7.
28 Athens M, Valdez R, Stern M. Effect of birth weight on future development of "Syndrome X" in adult life. *Diabetes* 1993;42:(suppl 1):61A.

(Accepted 11 January 1994)

Incidence and recognition of malnutrition in hospital

Janet P McWhirter, Christopher R Pennington

Abstract

***Objectives*—To determine incidence of malnutrition among patients on admission to hospital, to monitor their changes in nutritional status during stay, and to determine awareness of nutrition in different clinical units.**

***Design*—Prospective study of consecutive admissions.**

***Setting*—Acute teaching hospital.**

***Subjects*—500 patients admitted to hospital: 100 each from general surgery, general medicine, respiratory medicine, orthopaedic surgery, and medicine for the elderly.**

***Main outcome measures*—Nutritional status of patients on admission and reassessment on discharge, review of case notes for information about nutritional status.**

***Results*—On admission, 200 of the 500 patients were undernourished (body mass index less than 20) and 34% were overweight (body mass index >25). The 112 patients reassessed on discharge had mean weight loss of 5·4%, with greatest weight loss in those initially most undernourished. But the 10 patients referred for nutritional support showed mean weight gain of 7·9%. Review of case notes revealed that, of the 200 undernourished patients, only 96 had any nutritional information documented.**

***Conclusion*—Malnutrition remains a largely unrecognised problem in hospital and highlights the need for education on clinical nutrition.**

Department of Clinical Pharmacology, Ninewells Hospital and Medical School, Dundee DD1 9SY
Janet P McWhirter, *research dietitian*
Christopher R Pennington, *consultant physician*

Correspondence to:
Mrs McWhirter.

BMJ 1994;308:945-8

Introduction

More than 15 years have passed since the high incidence of protein energy malnutrition in medical and surgical patients was first shown,[1-3] and adverse changes in nutritional status during hospitalisation have also been reported.[4] Nutritional status has been shown to have important effects on health in recovery from illness or injury. Experimental semistarvation of normal volunteers that caused a 25% loss of body weight (to a body mass index of 17·5) was associated with apathy, depression, fatigue, and loss of will to recover.[5] Consequent loss of muscle power affects respiratory function, increasing susceptibility to chest infection,[6] and reduces cardiac function.[7] Impaired immune function increases the risk of infection.[8] Inevitably, such complications result in increased morbidity and mortality and lengthened stay in hospital,[9] at substantial extra cost in health care.[10]

Despite such findings, we believe that the problem of malnutrition in hospital remains largely unrecognised. The nutritional screening of patients at risk of depletion is not routine in many areas. In this study we sought to determine the incidence of nutritional depletion in patients admitted to hospital, to monitor changes in nutritional status during hospitalisation, to determine awareness of nutrition in different clinical units, and to assess the effect of nutritional intervention in relation to nutritional parameters.

Patients and methods

One of us (JPM) assessed the nutritional status of 500 patients on their admission to Dundee Teaching Hospitals Unit: 100 consecutive admissions from each of five disciplines—general surgery, general medicine, respiratory medicine, orthopaedic surgery, and medicine for the elderly. Patients admitted as day cases were not included in the study. Table I shows details of the