The extent of the reduction in liver content of copper and zinc depends on the type of protein energy malnutrition considered.

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PLATELET ASCORBIC ACID CONCENTRATIONS IN NUTRITIONALLY-DEPRIVED GROUPS IN THE AUSTRALIAN COMMUNITY

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Ascorbic acid (AA) is carried in several compartments of the blood. Plasma AA concentration is poorly correlated with the progression of scorbutic symptoms in volunteers placed on an AA-depleted diet[1]. AA body-pool size correlates better with buffy-coat AA than with plasma AA. Buffy-coat studies of ascorbic acid have shown that the upper fraction, which is platelet-rich, has higher concentrations of AA, indicating that the platelet is an important site of AA transport[2]. The metabolic role of AA within the platelets, and its effect on platelet function, remains unclear.

Ascorbic-acid hypovitaminosis may be due to inappropriate food habits, disease, extra metabolic demands, or drug interactions. Groups which may be at risk include (1) institutionalized elderly, (2) alcohol-abusers, (3) pregnant women, (4) newborn babies, particularly if bottle-fed, (5) surgical patients, (6) patients with hepatic or gastro-intestinal disease, (7) those with severe infection or stress, (8) smokers, (9) women on combined oral contraceptive pill, (10) salicylate users[3].

The institutionalized elderly have particular problems with food intake. Hospital foods can be deficient in nutrient intake as a result of prolonged heating times, or because they are pre-prepared. There may be feeding problems, as with cerebrovascular disease.

Alcohol-abusers are at risk of AA-deficiency. Folic-acid deficiency is commonly recognized, and the folic-acid-rich foods, such as fresh fruit and vegetables, are those which are the principal sources of AA; alcohol-abusers tend to neglect these types of foods. Alcohol may contribute to inadequate absorption of ascorbic acid[4], and the presence of liver disease may contribute.

Elderly Australians (community-based and institutionalized) and patients with upper gastro-intestinal haemorrhage (alcohol-abusers, and non-alcohol-users) were studied. Alcohol abuse was defined as a regular consumption of more than 80 g per day.

Platelets were separated from citrated plasma, the membranes disrupted in liquid nitrogen, and ascorbic acid assayed with the diphenylhydrazine reduction method[5].

The platelet AA-concentration of the institutionalized elderly was significantly less than that of the community-based elderly (Table 1).

Table 1. Platelet AA-concentration in nutritionally-deprived groups

	Elderly		Upper G/I haemorrhage	
	Community	Institutionalised	Non-alcohol abuse	Alcohol abuse
Platelet AA (nmol/10^{10} platelets)	234 ± 17	173 ± 16*	220 ± 18	150 ± 24**
n	27	23	18	12

Level of significance of difference: * $P<0.01$, ** $P<0.05$

The platelet AA-concentration of the alcohol-abusing patients with upper gastrointestinal haemorrhage was significantly less than that of the non-alcohol-abusing group (Table 1).

There was good correlation between plasma and platelet AA-concentration in the institutionalized elderly (n=23, r=0.86, P<0.001) and in the community-based elderly (n=27, r=0.77, P<0.001).

There was poor correlation between plasma and platelet AA-concentrations in patients with upper-gastrointestinal haemorrhage, both alcohol-abusers (n=12, r=0.21, P>0.5) and non-alcohol-abusers (n=18, r=0.35, P>0.5).

Haemostatic dysfunction is prominent in the symptom complex of scurvy. Abnormal collagen formation contributes to this, but platelet dysfunction has been described in isolated instances[6]. Our results show that groups at risk of deficient intake of AA have significantly lower platelet AA-concentration than equivalent control groups, although none suffered from frank scurvy.

In the fasting state, plasma AA concentrations show a good correlation with platelet AA-concentration. However, in the non-fasting, acute state, platelet AA-concentration may give a more reliable index of body stores of AA, if platelet AA turnover is slower than plasma AA. In those with haemostatic dysfunction, as in upper gastrointestinal haemorrhage, the effect of co-existing abnormalities of haemostasis, such as clotting-factor deficiency in liver disease, or thrombocytopaenia due to the effects of alcohol on the bone marrow, hypersplenism and folic-acid deficiency, might be exacerbated by abnormalities of platelet function consequent on lower concentrations of AA. The question could be approached by examining the relationship between platelet AA-concentration and indices of platelet function, such as skin bleeding time, *in-vivo* platelet adhesion, and platelet-aggregation studies.

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DIETARY TREATMENT OF KWASHIORKOR CHILDREN WITH LOCALLY AVAILABLE FOOD – AN OUT-PATIENT STUDY

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Visits to out-patients clinics in Benin constantly reveal that many children still suffer from malnutrition and it has been shown that kwashiorkor was most prevalent in this group[1]. In order to prevent this disease and save the lives of these children, an attempt is being made to find out possible causative, curative and preventive measures of kwashiorkor in Benin. This report is part of a longitudinal study of 80 children followed-up from birth to 36 months of age.

The annual deliveries in the two largest hospitals in Benin in 1975 consisted of 2811 singleton babies, on which average birth weights and infant mortality rate were recorded (in press). During the investigation which consisted of periodic home visits, 80 babies were selected using the multi-stage sampling method, on the basis of place of birth, age of the

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