of blood and breast milk samples collected in rural Mexico in which malabsorption was speculated about but was not tested for. In fact, other investigators have suggested that low holotranscobalamin II concentrations are not specific for cobalamin status and may be caused by such things as erythroid hyperplasia (4).

Until adequate evidence emerges to support the sweeping claim that low holotranscobalamin II concentrations are specific for cobalamin malabsorption and can reliably serve as surrogate Schilling tests (or the equally common, but not at all equivalent, assertion that they are specific for cobalamin deficiency), such claims remain interesting and provocative but unproven. Readers must judge the evidence for themselves.

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REFERENCES


Reply to V Herbert

Dear Sir:

We agree with Herbert that vitamin B-12 deficiency is common in the elderly. However, we do not think there is sufficient experimental evidence currently to definitively state what the minimum effective dose of oral vitamin B-12 is for all elderly subjects. Investigations that determine the fall in methylmalonic acid, homocysteine, or both after ingestion of various quantities of oral vitamin B-12 are urgently needed.

Herbert suggests that measurements of holotranscobalamin II are a useful test for early vitamin B-12 deficiency. We detailed the major methodologic problems in measuring holotranscobalamin II in a previous study (1). Because of the imprecision in vitamin B-12 assays, serum holotranscobalamin II is frequently measured as 0 or a negative number. There is little current evidence that holotranscobalamin II concentrations are a useful diagnostic test. A value of 0 was found in 30 of 69 elderly subjects in a recent investigation of vitamin B-12 status (2). In another investigation, the low holotranscobalamin II concentrations did not differentiate between different diagnoses, all resulting in macrocytosis (3). Also, test kits for serum holotranscobalamin II measurement are not widely available commercially and thus are not currently available to clinicians for diagnosis of vitamin B-12 status.

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REFERENCES


 Differences in resting metabolic rate between obese black and white women

Dear Sir:

Albu et al (1) found lower resting metabolic rates in obese black women than in obese white women. Albu et al noted that Kushner et al (2) had reported similar findings and that Spurr et al (3) had reported contrasting findings. Reasons for the differences were unclear. Perhaps the findings of Barker et al (reviewed by Goldberg and Prentice (4)) provide an explanation. Birth weight (and by implication prenatal care and maternal nutrition) is apparently related to subsequent waist-to-hip ratio in adulthood. Small babies are more likely to be obese adults. Prenatal care, maternal nutrition, and birth weight are related to economic status and the availability of prenatal care. One wonders about the influence of these factors on the findings described by Albu et al and others.

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REFERENCES


Reply to HH Sandstead

Dear Sir:

Sandstead brings forth an interesting issue, that is, the concept of nutritional programming or the effect of early nutritional experiences and in utero fetal growth on disease later in adult life as a possible explanation for the finding of lower resting metabolic rate (RMR) in some black as compared with white populations. This concept was reviewed recently by Goldberg and Prentice (1) and has been studied extensively in humans by Barker et al (1). Studies in animals have shown that undernutrition during certain gestational periods affects growth and health later in life (1). In humans, Barker et al found links between maternal nutrition, birth weight, and the development of risk factors for diabetes and coronary artery disease in adulthood in several population samples (1). The hypothesized explanation for this is that the consequent physiologic adaptation to the in utero environment is accompanied by permanent changes, such as differences in cell number, organ structure, and the resetting of hormonal axes that are perpetuated into adulthood (1).

Could differences in RMR between black and white population samples be attributed by extrapolation to different early nutritional programming or different perinatal or environment? We have not found any evidence in the literature linking maternal and fetal nutrition or birth weight with RMR but it is not inconceivable that such a link exists. Differences in autonomic nervous system activity as well as in the sensitivity to autonomic regulation may be traits that can be influenced in utero (2) and perhaps persist into adulthood. There is precedent for this (1): fetal and placental size and growth in utero as well as birth weight have been related to later hypertension, and fetal growth and infant feeding have been related to later thyroid function.

Although at present we do not know the mechanism underlying the differences in RMR between samples of black and white children and adults, it is conceivable that hormonal concentrations or responsiveness may play a role. Although several studies have shown differences in RMR between black and white population samples (3–7), not all studies have done so (8, 9). This could support the possibility that other factors early in life, perhaps in utero, may be determinants of basal energy expenditure. Studies of RMR in samples of black and white populations from similar or different environments and correlations between maternal weight and RMR of infants and children may help clarify this issue.

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REFERENCES


Erratum

Kotler DP, Burastero S, Wang J, Pierson RN Jr. Prediction of body cell mass, fat-free mass, and total body water with bioelectrical impedance analysis: effects of race, sex, and disease. Am J Clin Nutr 1996;64(suppl):489S–97S. On page 492S in Table 5 and on page 493S above Figure 2, in the equation for the prediction of fat-free mass in women the denominator should be 25.22 and not 22.22. The mistake was typographical and not a result of a miscalculation. It does not affect the interpretation of the results.