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Competing interests: None declared.

Low-calcium diet

Elizabeth Sellers and associates[1] write about the adaptation of Inuit children to a low-calcium diet. Contemporary humans evolved in an equatorial environment, and there can be little doubt that populations living under radically different conditions have had to adapt in substantial ways. Nevertheless, 3 important errors in this article need clarification if we are to gain any insight into the character of the adaptation, at least with respect to calcium.

First, the magnitude of urinary calcium excretion, expressed in this paper as fractional micromoles per mole creatinine, is incorrect by 6 orders of magnitude. As reported by Sellers and associates,[1] the urine of these children would have contained less calcium than distilled water. This might be taken as an indication of the adaptation the authors are seeking to define, except that the values reported are considered either at or above age-specific normal values in all of the 10 children studied. Therefore, the units for this test result are incorrect.

Second, the authors seem to have misinterpreted the data from the reference by Kuhnlein and colleagues[2] when they state "With a traditional diet, Inuit children in northern Canada ingest only 20 mg of elemental calcium per day." In the article concerned, traditional foods, providing 21 mg calcium daily (not the 20 mg cited), constituted only 17% of the total energy intake of the Inuit children studied. Had total energy intake come from traditional foods, total calcium intake would have been at least 120 mg/day. That is still not very much, but it is not safe to extrapolate from such a small proportion of the diet, since deriving total energy from traditional foods might well have involved a change in food types. This is strongly suggested by the standard deviation around the 21-mg average reported by Kuhnlein and colleagues,[2] which was 400 mg. Thus, the intake data were severely skewed to the right, indicating that some of the children must have been getting 1000 mg calcium or more from traditional foods. Given these uncertainties, the article by Kuhnlein and colleagues[2] provides no useful information about the calcium content of diets based completely on traditional foods.

The third error relates to the uncritical assumption that any adaptation at all would suffice to build an adult skeleton with a daily intake as low as the 20-mg figure mentioned by Sellers and associates.[1] If all 20 mg could be absorbed and retained, and if dermal and excretory losses could be reduced to zero (both impossible conditions), total skeletal accumulation from birth to age 16 would produce a skeleton containing less than 120 g calcium. Thus, the premise that adaptation must be possible for such an intake is untenable. Whatever the basis for the error, the authors should have realized than any intake estimate as low as the one cited had to be incorrect.

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[The authors respond:]

Our hospital laboratory customarily reports all concentration ratios with the same units for both numerator and denominator (i.e., moles per mole [mol/mol] or micromoles per micromole [µmol/µmol]), and this was the case for both the results and the normative data for our study.[1] However, as Robert Heaney rightly points out, these values were inadvertently mislabelled and reported with units of micromoles per mole. Nonetheless, because the numbers for both the reported results and the reference values are correct (with units of moles per mole), neither the results, their interpretation nor our conclusions are affected by this error.

The study by Kuhnlein and colleagues[2] does indeed report 21 (standard deviation 400) mg as the calcium intake derived from the traditional portion of a mixed diet. During manuscript revision, this figure was accidentally substituted for the estimated total daily calcium intake, which by extrapolation to a fully traditional diet is on the order of 123 mg/day; this remains profoundly low compared with the recommended daily intake of 900 mg. In any case, as Heaney notes, the reported standard deviation precludes placing too great an emphasis on the precise numeric value. Hence, neither 20 mg nor 120 mg should be regarded as more than a round number illustrating the magnitude of the discrepancy, and neither the results nor the conclusions inferred from them are materially affected by reference to the extrapolated value. Moreover, given this uncertainty and the absence of any reports of bone mineral density for a population using a traditional diet alone, it may be premature to speculate as to the sufficiency of bone mineralization under these circumstances. Further studies in this area are clearly warranted.

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Serotonin syndrome: not a benign toxidrome

Philippe Birmes and associates[1] suggest that serotonin syndrome is a less serious condition than neuroleptic malignant syndrome (NMS), but this has not been our experience.[2-5] In our prospective study of serotonin syndrome,[4,5] 6 of the 16 patients experienced disseminated intravascular coagulation (DIC), rhabdomyolysis and hypotension necessitating admission to the intensive care unit. Acute renal failure developed in 2 patients, and 1 patient died.

Table 2 in the article by Birmes and associates[1] does not capture the key differences between NMS and serotonin syndrome. Both conditions can be fulminant, and patients may present with delirium, hyperthermia, rhabdomyolysis, dilated pupils, tachycardia, daphoresis, rigidity and blood pressure changes[2-5] (see Table 1 with this letter). The main difference lies in the clinical gestalt: typically a patient with serotonin syndrome is agitated, speaks incoherently and has prominent myoclonus, whereas a patient with NMS is immobile, mute and staring. Although rhabdomyolysis is a complication of both toxidromes, DIC, seizures, ventricular tachycardia and severe hypotension are extremely rare in NMS.[2]

We agree with the mainstays of treatment suggested by Birmes and associates,[1] but we also advise monitoring of vital signs, platelet count, muscle enzymes and myoglobin twice daily for at least 72 hours. We have serious concerns about the use of chlorpromazine and propranolol for serotonin syndrome. Both drugs decrease blood pressure, which will exacerbate the hard-to-treat hypotension that can occur in serotonin syndrome; in addition, chlorpromazine may precipitate NMS. An absolute contraindication for the use of propranolol is a history of asthma, which is difficult to elicit if the patient is delirious. Finally, it is important to advise patients taking serotonergic agents about the risks of this potentially serious and fulminant syndrome.

Table 1: Clinical characteristics, laboratory abnormalities, complications, and risk factors for neuroleptic malignant syndrome and serotonin syndrome[2-5]

Characteristic	Neuroleptic malignant syndrome	Serotonin syndrome
Typical clinical presentation	Rigid, mute, staring, immobile	Agitated, incoherent speech, myoclonic twitching, bruising
Cognitive	Mild confusion to delirium; difficult to assess because of mutism	Mild confusion to delirium
Autonomic	Fever, tachycardia, diaphoresis, dilatation of pupils, blood pressure instability	Fever, tachycardia, diaphoresis, dilatation of pupils, blood pressure instability (hypertension in moderate cases, hypotension in severe cases)
Gastrointestinal	Constipation, ileus	Nausea, vomiting, diarrhea
Neurologic	Severe muscular rigidity (cogwheel), rigours, tremulousness	Hyperreflexia, myoclonus, tremulousness, clonus, fasciculations, ataxia, with or without rigidity
Psychiatric	Facial expression "fearful," underlying psychosis, premorbid mood disorder	Underlying mood or anxiety disorder; delirium may be misinterpreted as psychosis
Common laboratory abnormalities	Leukocytosis, elevation of muscle enzymes (CPK, ALT, AST, LDH), low serum iron	Leukocytosis, elevation of muscle enzymes (CPK, ALT, AST, LDH), thrombocytopenia
Complications	Aspiration pneumonia, renal failure, pulmonary embolus, contractures, postepisode muscle weakness	Falls, seizures, severe hypotension, ventricular tachycardia, disseminated intravascular coagulation, renal failure, coma (mortality rate unknown)
Risk factors	Antipsychotic drug use (all types), polypharmacy, rapid increase in neuroleptic dosage, concurrent use of lithium, dehydration, catatonia, agitation, benzodiazepine withdrawal during neuroleptic treatment	Use of serotonergic agents (all types), polypharmacy, concurrent use of lithium, MAOIs plus demerol (other risk factors unknown)

Note: CPK = creatine phosphokinase, ALT = alanine aminotransferase, AST = aspartate aminotransferase, LDH = lactic dehydrogenase, MAOIs = monoamine oxidase inhibitors.

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Smith-Magenis syndrome

Waleed Al Busairi and Fawzi Ali[1] describe a 15-year-old boy with mental retardation and a history of putting inedible objects into his mouth. The authors might want to investigate