

Stanley Boyd Eaton  
Stanley Boyd Eaton III

## Paleolithic vs. modern diets – selected pathophysiological implications

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**Summary** The nutritional patterns of Paleolithic humans influenced genetic evolution during the time segment within which defining characteristics of contemporary humans were selected. Our genome can have changed little since the beginnings of agriculture, so, genetically, humans remain Stone Agers – adapted for a Paleolithic dietary regimen.

Such diets were based chiefly on wild game, fish and uncultivated plant foods. They provided abundant protein; a fat profile much different from that of affluent Western nations; high fibre; carbohydrate from fruits and vegetables (and some honey) but not from cereals, refined sugars and dairy products; high levels of micro-nutrients and probably of phytochemicals as well.

Differences between contempo-

rary and ancestral diets have many pathophysiological implications. This review addresses phytochemicals and cancer; calcium, physical exertion, bone mineral density and bone structural geometry; dietary protein, potassium, renal acid secretion and urinary calcium loss; and finally sarcopenia, adiposity, insulin receptors and insulin resistance.

While not, yet, a basis for formal recommendations, awareness of Paleolithic nutritional patterns should generate novel, testable hypotheses grounded in evolutionary theory and it should dispel complacency regarding currently accepted nutritional tenets.

**Key words** Paleolithic diet – insulin resistance – skeletal health – phytochemicals – type 2 diabetes

S. Boyd Eaton, MD (✉) · S. Boyd Eaton III  
Dept Anthropology  
Emory University  
2887 Howell Mill Road  
Atlanta GA 30327, USA  
e-mail: sboydeaton@aol.com

### Introduction

The Paleolithic or Old Stone Age occupied nearly all the last two million years of human evolutionary experience, an especially critical time segment during which the adaptations that define our genus were selected: body mass and shape, locomotive capability, masticatory apparatus, growth/developmental schedule, relative (and absolute) brain size, resting metabolic rate, and daily foraging range [1]. Experiential changes associated with agriculture and industry have been dramatic and might readily have influenced the human genome given sufficient time; however, the very rapidity of these innovations has almost totally overreached the capacity of genetic evolution to keep pace.

While there has clearly been some Holocene genetic adaptation, primarily to infectious diseases (e.g. malaria), increasing population size and greater mobility have actually reduced the likelihood of genetic innovation in recent millennia [2]. It is, therefore, not mere rhetorical hyperbole to assert that contemporary humans are, in the genetic sense, still Stone Agers and that, consequently, we remain adapted for a preagricultural nutritional pattern.

### Paleolithic diets

There was no universal nutritional regimen for Stone Agers, but in terms of mathematical set theory, the super-

**Table 1** General paleolithic nutrition

Nutrient	Typical	Variation with Latitude
Protein		
Animal	Very High	Positive
Vegetable	Moderate	Negative
Fat		
Total	Moderate to High (~ Mediterranean vs. E. Asian)	Positive
C20 & C22 LCPUFA <sup>1</sup>	Very High	Positive
n-6:n-3 Ratio	~1	Positive
Serum Cholesterol		
Raising FA	Low	Positive
Cholesterol	High (~ US levels)	Positive
Carbohydrate		
Cereals	None to Minimal	Negative
Vegetables & Fruits	Very High	Negative
Dairy Foods	None After Infancy	–
Refined Sugars	None (Honey)	–
Fibre	Very High	Negative
Micronutrients	Very High	Negative
Phytochemicals	(Probably High)	(Probably Negative)

<sup>1</sup>long-chain polyunsaturated fatty acids

set believed to encompass Paleolithic dietary patterns (e. g. high latitude, arid lands, equatorial, coastal) and the super-set including contemporary ones (e. g. traditional East Asian, Mediterranean, typical American, vegetarians) are largely disjoint; they overlap surprisingly little. Preagricultural humans ate wild game, fish, uncultivated plant foods and, when available, honey. Grains were for emergencies and there were no dairy products, oils, salt, processed foods, nor empty calories.

In almost all cases, Stone Agers consumed more animal protein than do current Westerners (Tab. 1). Total fat consumption varied, chiefly with latitude, but intake of serum-cholesterol-raising fat was nearly always less than for Americans and Europeans [3] while there was more dietary long-chain (C20 and above) polyunsaturated fatty acid (LCPUFA) [4]. The preagricultural essential fatty acid ratio ( $\omega 6:\omega 3$ ) approached equality; for average Americans this ratio is 10:1 or higher. Dietary cholesterol roughly equated U. S. levels. Carbohydrate consumption also varied with latitude, but, in all cases, came chiefly from fruits and vegetables, not from cereals, refined sugars, and dairy products. Compared with the Western European dietary pattern, Paleolithic foods provided much more fibre (both soluble and insoluble) and from two to ten times more micronutrients (Table 2); there was probably a similar phytochemical discrepancy.

**Table 2** Paleolithic nutrition

	Paleolithic <sup>1</sup> (mg/d)	Current US <sup>2</sup> (mg/d)	Ratio
MINERALS			
Calcium	1622	920	1.8
Copper	12.2	1.2	10.2
Iron	87.4	10.5	8.3
Magnesium	1223	320	3.8
Manganese	13.3	3.0	4.4
Phosphorus	3223	1510	2.1
Potassium	10500	2500	4.2
Sodium	768	4000	0.2
Zinc	43.4	12.5	3.5
VITAMINS			
Ascorbate	604	93	6.5
Folate	0.36	0.18	2.0
Riboflavin	6.49	1.71	3.8
Thiamin	3.91	1.42	2.8
Vitamin A	17.2	7.8	2.2
Vitamin E	32.8	8.5	3.9

<sup>1</sup> based on 3000 kcal/d, 35 % animal; 65 % plant subsistence<sup>2</sup> average of US men and women; Food and Nutrition Board, 1989

## Evolution, nutrition, and pathophysiology

An appreciation of Paleolithic nutrient-nutrient interactions and of the relationships between diet and other aspects of ancestral experience can enlighten topics of current interest and/or controversy. Three (of many possible) examples are phytochemicals, skeletal health and insulin resistance.

## Phytochemicals

No free-living primates except humans consume cereal grains, but from the emergence of agriculture, wheat, rice, corn, and their like have provided from 40 to 90 % of our energy requirements. In so doing they have reduced the contribution of fruits and vegetables, the major energy source for Stone Agers and their simian predecessors over the preceding fifty million year evolutionary span. Recent epidemiological metaanalyses strongly suggest that fruits and vegetables have far more cancer-preventive potential than do cereals [5], perhaps reflecting the phytochemical content of non-cereal plant foods, phytochemicals to which current human biology became adapted through many million years of interrelationships. In contrast, the phytochemicals of grains have interacted with the human genome for only 10,000 years. While it required epidemiology to demonstrate that fruits and vegetables have cancer preventive-potential (and more such potential than do cereals), evolutionary understanding suggests why this

should be so and also implies that the partial replacement of fruits and vegetables by cereals following the emergence of agriculture probably increased our susceptibility to neoplastic diseases.

### Skeletal health

Despite their lack of dairy products, Paleolithic diets generally provided more calcium than do the diets of Americans and Europeans. Wild plant foods often contain considerable calcium, the average for 119 such items being 132.6 mg/100 g (vs. whole milk ~120 mg/100 mg) [6]. However, in far northern latitudes, plant foods sometimes made up only 5 % of annual energy intake, so that low bone mineral density (osteoporosis) was common among Stone Agers living in such regions. Nevertheless, fractures were infrequent because a bone's strength is determined not only by its mineral density, but also by its structural geometry, especially its diameter and cross-sectional configuration [7]. Structural geometry is affected by habitual physical activity which tends to increase bone diameter and create cross-sections more oval than round. Such bones resist mechanical stresses effectively, even when osteoporotic. This effect helps explain why Melanesians and black South Africans, whose calcium intake is low, have far fewer age-related fractures than do black and white Americans whose calcium intake is greater.

Diets high in protein have been positively correlated with hip fracture rates, presumably because protein increases urinary calcium loss. The mechanism involves endogenous acid production which is increased by metabolism of protein; renal acid secretion and urinary calcium loss are highly correlated. Conversely, dietary potassium produces a net alkalinising effect, opposite to that of protein, and it appears that net endogenous acid production, which largely determines net renal acid excretion, can be predicted by assessing the dietary protein (g/d) / potassium (mg/d) ratio, with lower values being protective [8]. The extremely high potassium content of Paleolithic diets (e.g. 10500 mg/d for a 3000 kcal, 35:65 animal:plant subsistence pattern vs. ~2500 mg/d in the average American diet) thus may have exerted a critically beneficial influence: a protein / potassium ratio of 0.84 for typical Stone Agers compared with an average value of 1.24 for 141 American subjects consuming 20 different contemporary diets.

Dietary sodium also increases urinary calcium loss [9]; its effect is roughly similar in magnitude to that of protein. The typically low sodium intake of Stone Agers (~768 mg/d vs. Americans ~4000 mg/d) would have exerted additional protective influence on Paleolithic skeletal health.

### Insulin resistance

Insulin resistance and hyperinsulinaemia are increasingly thought of as central factors in the pathophysiology, not only of type 2 diabetes, but also of hypertension and coronary heart disease. While it is clear that genetics, diet and exercise are all involved in the development of insulin resistance, the exact mechanism remains unclear. Since the field remains open, perhaps an evolution-based hypothesis merits consideration.

The relationship between excess adiposity and insulin resistance is well recognised, but evolutionary considerations suggest a parallel linkage involving relative skeletal muscle deficiency. Contemporary humans are distinguished from their predecessors not just by hyperadiposity, but also by sarcopenia [10]. In addition, current physical fitness levels are much below those typical in the past. These altered parameters distort the physiological milieu for insulin action from what it was when the genetic bases for carbohydrate metabolic regulation was selected.

Gramme for gramme, insulin-stimulated muscle can extract far more glucose from the blood than can insulin-stimulated adipose tissue and exercise-conditioned muscle extracts more than does non-conditioned muscle [11]. Hence, a logical hypothesis is that functional insulin resistance, especially in its earliest stages, is directly proportional to body fat mass and inversely proportional to the mass and metabolic activity of skeletal muscle. This relationship might reflect competition between the insulin receptors of myocytes and those of adipocytes for available insulin molecules. The initial effect would be repetitive episodes of transient hyperglycaemia and hyperinsulinaemia. In genetically susceptible individuals, further metabolic deterioration could result from secondary down-regulation of insulin receptors, glucose transporters, and intracellular enzymatic sequences, leading ultimately to glucose intolerance and type 2 diabetes.

### Conclusion

At one level the insights arising from these examples – eat more fruits and vegetables, reduce sodium intake, and increase exercise – are banal. However, there are more profound implications. Basing the nutritional pyramid on grains is wholly out of line with primate and hominid evolutionary experience. A Paleolithic model experimental diet – high protein, high fibre, minimal serum-cholesterol raising potential, balanced n-3:n-6 fatty acid ratios, high LCPUFA content, etc. – deserves investigative support. Micronutrient intake throughout evolutionary experience exceeded current RDAs. The importance of bone structural geometry and possible myocyte-adipocyte insulin receptor competition both deserve study.

Paleolithic nutritional awareness is not, yet, a basis for formal recommendations, but it can generate testable hy-

potheses grounded in evolutionary theory. And it should dispel complacency regarding currently accepted nutritional tenets.

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## References

1. Wood B, Collard M (1999) The human genus. *Science* 284: 65–71
2. Tattersall I (1998) *Becoming Human. Evolution and Human Uniqueness*. Harcourt Brace, New York; pp 239.
3. Eaton SB, Eaton SB III, Konner MJ (1997) Paleolithic nutrition revisited: a twelve-year retrospective on its nature and implications. *Eur J Clin Nutr* 51: 207–16
4. Eaton SB, Eaton SV III, Sinclair AJ, Cordain L, Mann NJ (1998) Dietary intake of long-chain polyunsaturated fatty acids during the paleolithic. *World Rev Nutr Diet* 83: 12–23
5. World Cancer Research Fund, American Institute for Cancer Research (1997) Food, Nutrition and the Prevention of Cancer: a Global Perspective. American Institute for Cancer Research, Washington D.C, pp 506–507
6. Eaton SB, Nelson DA (1997) Calcium in evolutionary perspective. *Am J Clin Nutr* 54: 281S–2817S
7. Ruff CB (1992) Biomedical analyses of archaeological human material. In: Saunders SR, Katzenburg A (eds). *The Skeletal Biology of Past Peoples*. Alan R Liss, New York, pp 41–62
8. Frassetto LA, Todd KM, Morris Jr RC, Sebastian A (1998) Estimation of net endogenous noncarbonic acid production in humans from diet potassium and protein contents. *Am J Clin Nutr* 68: 576–583
9. Devine A, Criddle RA, Dick IM, Kerr DA, Prince RL (1995) A longitudinal study of the effect of sodium and calcium intakes on regional bone density in postmenopausal women. *Am J Clin Nutr* 62: 740–745
10. Rode A, Shephard RJ (1994) The physiological consequences of acculturation: a 20-year study in an Inuit community. *Eur J Appl Physiol* 69: 16–24
11. DeFronzo R (1997) Pathogenesis of type 2 diabetes: metabolic & molecular implications for identifying diabetes genes. *Diabetes Rev* 5: 177–269