The gourmet ape: evolution and human food preferences

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ABSTRACT

This review explores the relation between evolution, ecology, and culture in determining human food preferences. The basic physiology and morphology of Homo sapiens sets boundaries to our eating habits, but within these boundaries human food preferences are remarkably varied, both within and between populations. This does not mean that variation is entirely cultural or learned, because genes and culture may coevolve to determine variation in dietary habits. This coevolution has been well elucidated in some cases, such as lactose tolerance (lactase persistence) in adults, but is less well understood in others, such as in favism in the Mediterranean and other regions. Genetic variation in bitter taste sensitivity has been well documented, and it affects food preferences (eg, avoidance of cruciferous vegetables). The selective advantage of this variation is not clear. In African populations, there is an association between insensitivity to bitter taste and the prevalence of malaria, which suggests that insensitivity may have been selected for in regions in which eating bitter plants would confer some protection against malaria. Another, more general, hypothesis is that variation in bitter taste sensitivity has coevolved with the use of spices in cooking, which, in turn, is thought to be a cultural tradition that reduces the dangers of microbial contamination of food. Our evolutionary heritage of food preferences and eating habits leaves us mismatched with the food environments we have created, which leads to problems such as obesity and type 2 diabetes. Am J Clin Nutr 2009;90(suppl):707S–11S.

FOOD PREFERENCES: EVOLUTION, ECOLOGY AND CULTURE

A scientist from another planet, observing human feeding habits, would be struck by 4 things: the remarkable variation in food habits within and between populations, the fact that in many populations food is farmed rather than hunted or gathered, the importance of cultural traditions and ritual in relation to food, and the fact that food is often processed—eg, by cooking or other means—before it is eaten. It is the last of these that is uniquely human. There are other animals that farm food (eg, certain ants), food traditions may be culturally transmitted (eg, the pecking open of milk-bottle tops by birds, which was a phenomenon that spread through Britain in the middle of the 20th century), and other generalist feeders, such as humans, show incredible variation in diet from one place to another. But no other animals cook or manufacture their food.

Given the unusual combination of features of our food lives, it might be tempting conclude that human food preferences are shaped entirely by culture and individual experience. But in this review I discuss how these factors interact with genetics, ecology, and evolution, and in particular how this might give rise to variation within and between populations.

The importance of evolutionary heritage is underlined by our anatomy and physiology. We have inherited from our proto-hominid ancestors the teeth and digestive system of an omnivore. Carbon isotope analysis of the fossil remains of our early ancestors confirms that they were indeed omnivores. Australopithecus africanus, for example, probably had a diet that was ≈75% fruit and leaves but also 25% meat. This conclusion is drawn from the fact that grasses, and the herbivores that feed on them, have more carbon-13 than do leaves or fruit. We know from their teeth that Australopithecines were not adapted to eat grass, so they must have acquired the carbon-13 from meat (1, 2).

Some anthropologists have also suggested that our ancestors’ feeding habits were not shown only by their teeth and digestive systems but had much wider ramifications. Richard Wrangham controversially proposed, for example, that the discovery of cooking was linked to the extraordinarily rapid evolutionary enlargement of the human brain (3, 4).

Likewise, our 5 senses of taste—sweet, salt, umami, bitter, and sour—equipped us for consumption of the essentials for survival—energy, salt, and protein—as well for the avoidance of the dangers of poisonous or rotten food. Of course, food preference is determined by much more than our senses of taste. The perceived flavor of food often depends as much on scent as on taste, and our expectations can affect the perception of food flavors (5); however, I explore how genetic variation in taste may interact with food traditions and ecology.

Genes, culture, and ecology: lactase persistence

The phenomenon of lactose intolerance, or lactase persistence, caused by one or more dominant mutations that arose at the time of the dawn of agriculture, ∼10,000 y ago, and which affects the production of lactase, the enzyme responsible for breaking down milk sugar, is a well-studied instance of how genes, ecology, and culture interact to determine food preferences (6).

Prior to that time, it is generally thought that humans stopped lactase production after weaning, and therefore lost the ability to digest milk at this point in life. In other words, this was the...
primitive, or ancestral, state. Lactose is an important component of breast milk, and therefore infants need to be able to digest it, but why should this be lost after weaning? Two arguments have been put forward. One pertains to the economics of metabolism: there is a selective disadvantage in continuing to produce an enzyme, with its associated energy costs, once it is no longer needed. The other argument is that the loss of lactase activity postweaning is an outcome of parent-offspring conflict. Evolutionary theorists have pointed out (7) that parents and offspring may often have divergent interests concerning the amount of investment in an individual offspring. Each offspring values his or her own survival more than that of his or her siblings, whereas parents value each offspring more or less equally. In the simplest case, genes expressed in a mother producing offspring sequentially should favor continued investment in the current one as long as $\partial B > \partial C$, where $\partial B$ is the marginal benefit from additional investment and $\partial C$ the marginal cost to future offspring. However, genes expressed in an offspring would favor additional investment provided that $\partial B > r \partial C$, where $r$ is the degree of relatedness between offspring (typically 0.5 in diploid species with sexual reproduction) (8). One possible evolutionary outcome of this conflict over time of weaning would be for selection to favor parents who produce offspring that lose the ability to digest milk.

Derived from this ancestral state of lactase intolerance after weaning, today ≈25% of the human population is unable to digest lactose into adulthood. Models of selection suggest a selective advantage of 3–5% could account for the change in gene frequency over 10,000 y. The selective benefit of lactose tolerance in adulthood probably arose with the birth of livestock farming. Milk was a new, nutritious, and energy-rich source of food, and individuals able to capitalize on this would have been at an advantage. This is indeed reflected in present-day gene frequencies. Lactose tolerance is much commoner in populations with a long history of livestock agriculture than in those with little or only recent history. For example, in northern Europe, ≈95% of adults are able to digest lactose, whereas in some parts of Asia, <10% are able to do so (9). Bloom and Sherman (10) extend this analysis further by showing that the distribution of lactase intolerances is also correlated with the historical distribution of communicable diseases of cattle. Hence, the ecology of disease influenced the likelihood of herding being adopted by our ancestors, and this in turn generated a selective force for a change in our digestive system.

Recent genetic evidence indicates that lactase persistence has evolved independently in African and European populations (10, 11), perhaps associated with the independent development of dairying.

In summary, variation among populations in consumption of milk as adults is a result of an interaction between cultural traditions, genes, and ecology.

A dynamic polymorphism, driven by disease: favism

Lactose tolerance is one of many examples of the way in which genes, culture, and food preferences coevolve. About 400 million people, especially in Africa, the Mediterranean, and Middle East have a mutation affecting the activity of the red blood cell enzyme glucose 6 phosphate dehydrogenase (G6DP), which is involved in the oxidative phase of the pentose phosphate pathway.

This mutation, analogous to sickle cell anemia, confers an advantage in resistance to malaria because it deprives the Plasmodium falciparum parasite of oxygen in the red blood cells. The mutation is sex-linked and probably maintained by heterozygote advantage in females. But there is a cost to males bearing the mutation (as well as homozygous females) because they are unable to digest one of the staple diet items of the region, broad beans (a condition known as favism).

The frequency of the mutation that causes favism is correlated with the geographical distribution of malaria in the Mediterranean and Middle East. This suggests that there is a selective balance between disease resistance and food consumption. Intriguingly, some of the traditional herbs and spices used in preparing broad beans in areas of high frequency of favism may actually make the beans easier to digest for those with the mutation (12, 13).

Taste sensitivity

We are all familiar with the observation that the same kind of food might taste very different to 2 individuals. In part, this difference is cultural. We are attuned by our individual experiences and our social environment to react to some foods as delicious and others as disgustng. But there is also a genetic component to taste preference.

The first discovery of a genetic basis was the accidental finding of Arthur L Fox while working for the Dupont Chemical Company: Fox found that people varied in their response to the bitter chemical phenylthiocarbamide (PTC; subsequently, the same variation in response was found to occur with 6-n-propylthiouracil) (14). At the next American Association for the Advancement of Science meeting (in 1931), he and a colleague tested the audience and found that 28% of the audience could not taste PTC, 65% could, and the remainder could not be classified. Subsequently, the tasters were divided into those who are very sensitive, known as super-tasters (25% of the population), and those who are sensitive, known as tasters (50% of the population) (14).

This variation is associated with 25 genes and 8 pseudogenes on 3 chromosomes (15). The receptors involved largely respond to plant secondary compounds, ie, compounds manufactured by plants that are poisonous or unpalatable to predators. In other words, the receptors serve to enable us to avoid toxins in food. Not surprisingly, super-tasters tend not to like vegetables, such as crucifers, that contain bitter secondary compounds. As with any toxin, the risk associated with ingesting plant toxins depends on the dose, and at low doses, plant secondary compounds may have beneficial effects as antioxidants. So there is likely to be a balance of risk and benefit in avoiding toxins. But this leaves open the question of why there is genetic variation between populations in sensitivity to bitter taste. The frequency of non-tasters varies among populations, from as low as 7% to >40% in populations that have been studied (13). Why should this be?

One intriguing explanation for the variation among populations within Africa (16) is that it is a product of selection for malaria resistance. A low sensitivity to bitter taste was observed in areas with endemic malaria, and it was hypothesized that this is because certain bitter secondary compounds in plants are protective against malaria. In areas with endemic malaria, the
protective benefit of consuming potentially dangerous bitter plants could outweigh the risks of poisoning.

Spices

Another possibility, as yet to be explored, is genetic variation in taste sensitivity between populations linked to the cultural tradition of adding spices to food. Spices are made from plant secondary compounds, and therefore nontasters are less sensitive to strongly spiced food.

But why are spices, which are potentially toxic as well as costly, added to food? Many ideas have been suggested, and it is important to distinguish between those that concern ultimate survival value, and those that refer to the causal, or proximate, mechanisms. For example, the suggestions that “spices make bland food taste better,” “cover up the taste of bad meat,” or “are cultural traditions” refer to proximate mechanisms but beg the question of the survival value of using spices.

Three often-quoted ideas about the evolutionary advantage of using spices are that they contain important micronutrients, that they are a sign of wealth of the spice owner, or that they have antimicrobial properties. The importance of spices as a sign of wealth is underlined by that fact that Alaric the Goth, who laid siege to Rome in 408 AD, demanded as a ransom various precious metals and 3000 pounds of pepper (17). In medieval England, members of the Peppers’ Guild, founded in 1180, were the custodians of the King’s weights, which reflected the importance of pepper as a barometer of the state of business. In 1373 the pepperers, having joined forces with the spicers, renamed themselves the company of Grossers (or Grocers) (because they were responsible for the heavy weights or peso grosso). The Grossers soon took on responsibility for ensuring the purity of spices sold, a process called garbling (from the Arabic gharaiba, meaning to sift or select) (18). In many animal species, differences among individual males in mating success are related to differences in the amount of resources they are able to provide for females (19), and spices could be an analog of this.

Spices could have nutritional benefits: for example, they tend to be antioxidants, which are thought to reduce oxidative damage to cells. It has even been suggested that consumption of turmeric (cucurmin) might contribute to the low prevalence of Alzheimer’s disease in India (20).

The analysis of spice use in relation to ecology, however, supports the antimicrobial hypothesis. Paul Sherman and his students analyzed the use of spices in the traditional cuisines of 37 countries. The patterns are striking, and fit with our intuition. More spices are used in hotter climates. But this pattern applies only to the spices that inhibit bacterial growth and only in meat dishes (17, 21). There is no correlation between temperature and the number of spices grown in a country, so the relation between cuisine and temperature is not simply a matter of using what is available. Furthermore, in the United States and China, where there are large variations in temperature, a similar pattern holds.

Sherman concludes that cultural predilections for spicy food have arisen because, through cultural evolution, we have harnessed the natural antimicrobial compounds of plants and incorporated them into our diets. This may go hand in hand with selection for different degrees of taste sensitivity: ecology, genes, and culture woven together to influence our food preferences.

MISMATCH: OUR EVOLUTIONARY PAST COMING HOME TO ROOST

In the second part of this review, I look briefly at another facet of our evolutionary heritage and food preferences, namely the mismatch between evolved preferences and modern environments.

As many experts have pointed out, some of the major global health epidemics that have emerged in the past few decades are a result of this mismatch. I am going to refer briefly to 2 interrelated “diseases”: obesity and type 2 diabetes (I put disease in quotes, because not all agree that obesity is a disease, although all agree that there is an obesity epidemic).

Lest we get too pessimistic about these new health risks, let us remind ourselves that in many countries of the world life expectancy has increased dramatically over the past century, as major infectious diseases have been conquered and nutrition, sanitation, and other provisions of essentials for health have improved. In the United Kingdom, for example, life expectancy has increased by 60% in the past 100 y. But in the future this trend might well reverse, as a result of so-called lifestyle diseases.

Obesity

Obesity in the United Kingdom is rising rapidly, as it is in most parts of the world. In fact, the World Health Organization estimates that, globally, ≈1.2 billion people are overweight, of which ≥300 million are obese, a sobering contrast with the estimated 800 million who are undernourished.

Obesity carries with it significantly increased health risks—for example, of cardiovascular disease, some cancers, and type 2 diabetes—and a projected reduction in life expectancy of ≈9 y (22). As a result, most developed countries are wrestling with the problem of how to tackle the obesity epidemic. This is a particularly difficult challenge, both because of the multifactorial causation and because almost any action by government is likely to be seen as interfering with people’s individual lifestyles and therefore labeled as “nanny state” intervention.

From a biological perspective, the puzzle of obesity is to understand why the normal homeostatic control of body weight has failed in so many people. A wide range of potential contributing factors, both in terms of energy intake and energy expenditure, have been suggested, although their relative importance is not known. The features of modern living that work against the body’s normal mechanisms for balancing energy intake and expenditure are often summarized by saying that we live in an obesogenic environment, in which our normally adaptive physiology misfires.

One such misfiring relates to protein intake, and hence to the theme of this conference, the umami receptor. The body regulates protein intake, to ≈15% of intake, more tightly than either carbohydrate or fat, so when our diet contains too little protein, we compensate by overconsuming the other 2 macronutrients to keep protein intake constant (23). In recent decades, at least in the United States, where data are available, the average protein content of the diet has declined (23); in association with this, carbohydrate and fat intake has increased. No one factor accounts for the obesity epidemic, but protein regulation may play a part, and one policy response might be to increase the protein content of processed foods.
Type 2 diabetes

Just as the prevalence of obesity is rising rapidly, so is the prevalence of type 2 diabetes: it is projected to affect ~0.5 billion people within a few decades, at great cost both to the individual sufferers and to the health care systems that support them. But just as striking as the overall increase is that type 2 diabetes affects different groups to very different extents, from ~2% of the population in Europe to ~50% among Pima Indians of North America (24). Even within groups there are striking contrasts: for example, between rural and urban native peoples in Australia, or within the Pacific Island of Nauru over the past 50 y.

Is this due to differences in genetic makeup or in the environment? The answer is “both.” We know from twin studies, as well as from genome-wide associations, that there is genetic variation in susceptibility to the disease. We also know that lifestyle factors such as obesity, lack of exercise, and high caloric intake contribute to the risk (24). Furthermore, in utero effects contribute to risk, including fetal hyperglycemia (25) and low birth weight (26). Barker suggested that poor nutrition during fetal development and in early childhood could affect the development of the pancreas and predispose individuals to type 2 diabetes in later life. There are also epigenetic effects that transmit across generations: in a Swedish study, risk increased if paternal grandparents grew up in times of food abundance (27).

But if there is genetic variation in susceptibility to such a debilitating disease, why has selection not eliminated the genes that make us susceptible, and why is there so much apparent variation among populations?

Diamond (24) has suggested that the old idea of “thrifty genes” (28) might be the explanation. The idea is that, in the past, humans were selected to cope with periods of feast and famine. Hence, adaptations that facilitate large appetite and rapid energy uptake would have been an advantage. But these same adaptations misfire, and are therefore selected against, when we are exposed to continuous, plentiful food. The kinds of genetic variation that might be encompassed within the idea of thrifty genes could, for example, include genes affecting sensitivity to insulin or insulin release, as well as genes affecting the mechanisms of appetite control such as leptin release.

Diamond argues that, in Europe, with the advent of more stable food production, these thrifty genes were selected out of the population in a cryptic epidemic (30). Adaptations that have been exposed to famine conditions in more recent times, and now have plentiful food, are undergoing an epidemic of type 2 diabetes with selection against thrifty genes. Recent single nucleotide polymorphism analysis indicates that genes affecting obesity and diabetes are under strong selection (29) Even if Diamond is correct, this does not rule out other, social, factors, such as stress and low self-esteem, which have been suggested as contributors.

CONCLUSIONS

In this brief review, I have touched on a few examples to illustrate the ways in which our genetic heritage might affect our food preferences and eating habits. In conclusion, I want to make just 2 points. First, although our genetic predispositions may help to explain both our food preferences and our responses to food, as well as variation in these responses, this does not downplay the crucial importance of environmental, developmental, and cultural influences. In most of the examples I have discussed, the outcome depends on a complex interaction between genes and environment. Second, in tackling the major challenge of the links between diet and disease, policy makers should aim to work with, rather than against, our predispositions. I can do no better than quote from Gluckman and Hansen’s (30) book on the concept of mismatch: “the mismatch paradigm, as part of a life course approach to understanding disease causation, can explain and even predict the patterns of disease that are developing rapidly . . . . It may hold the key to interventions which could pay off.” (Other articles in this supplement to the Journal include references 31–59.)

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