Published in final edited form as:

Nutr Rev. 2013 August ; 71(8): 501–510. doi:10.1111/nure.12039.

BEYOND THE PALEOLITHIC PRESCRIPTION: INCORPORATING DIVERSITY AND FLEXIBILITY IN THE STUDY OF HUMAN DIET EVOLUTION

Bethany L. Turner[a] and **Amanda L. Thompson**[b]

[a]Department of Anthropology, Georgia State University, PO Box 3998, Atlanta, GA 30302, USA, bturnerlivermore@gsu.edu; phone (404) 413-5162; fax (404) 413-5159.

[b]Department of Anthropology, University of North Carolina, Chapel Hill, NC, USA

Abstract

Evolutionary paradigms of human health and nutrition center on the evolutionary discordance or "mismatch" model whereby human bodies, reflecting adaptations established in the Paleolithic era, are ill-suited to modern industrialized diets resulting in rapidly increasing rates of chronic metabolic disease. Whereas this model remains useful, we argue that its utility in explaining the evolution of human dietary tendencies is limited. The assumption that human diets are mismatched to our evolved biology implies that they are instinctual or genetically determined and rooted in the Paleolithic. We review current research indicating that human eating habits are primarily learned through behavioral, social and physiological mechanisms starting *in utero* and extending throughout the life course. Those adaptations that appear to be strongly genetic likely reflect Neolithic, rather than Paleolithic, adaptations and are significantly influenced by human niche-constructing behavior. Incorporating a broader understanding of the evolved mechanisms by which humans learn and imprint eating habits and the reciprocal effects of those habits on physiology would provide useful tools for structuring more lasting nutrition interventions.

Keywords

Human Evolution; Diet; Nutrition; Health; Mismatch; Food Choice; Gut Microflora; Niche Construction

Introduction

Concerns about the alarming number of American adults consuming poor diets associated with the development of obesity, cardiovascular disease, and type 2 diabetes mellitus (T2DM)[1-3] have prompted a great deal of research into the "true" or evolved ways that humans are meant to eat for optimal health.[4-8] In their seminal 1985 paper, Eaton and Konner[9] first posited that many of the chronic metabolic disorders now rampant in the industrialized world result from a way of life that is mismatched to human evolutionary history. They suggested that the "Paleolithic diet," an ancestral diet characterized by higher protein, less total fat, more essential fatty acids, lower sodium, and higher fiber, should serve as a reference standard for modern human nutrition. This argument has been widely incorporated into popular scientific and diet research[10], leading to the assumption that

modern human bodies, largely representing traits evolved during the Paleolithic period (2.6 million to 12,000 years ago) have been outpaced by culture change resulting in a gene-culture mismatch and the epidemic "diseases of civilization."[11]

However, this lingering perception that we are "Stone-Agers in the Fast Lane"[11] limits both our understanding of the long-standing flexibility that characterizes human dietary evolution and the utility of evolutionary models in shaping dietary interventions, particularly in a modern world in which many people are structurally limited in their lifestyle choices.[12-15] In this paper, we: 1) discuss the limitations of the reliance on the mismatch, or evolutionary discordance, model as the sole model of human diet evolution and fundamental cause of cardiometabolic disease; 2) review recent research in nutrition, evolutionary medicine, paleoanthropology, and physiology that documents considerable ecological, genetic, cultural and behavioral variation in human diet and metabolism; and 3) propose strategies for nutrition intervention that focus on the flexibility and diversity that have characterized much of human diet evolution, and the mechanisms through which human feeding behaviors are shaped within individual lifetimes, rather than on a return to an idealized hunter-gatherer subsistence pattern.

The Mismatch Hypothesis: Applications and Limitations for Understanding Human Dietary Evolution

The mismatch hypothesis has its origins in the "thrifty genotype" hypothesis[16] that populations who had remained hunter-gatherers into modern historical periods maintained traits that would favor insulin resistance and energy storage in times of famine, an adaptive complex-turned-liability in modern sedentary agricultural life. "Paleolithic Nutrition" expanded on this by compiling data from populations still engaged in more "traditional" foraging subsistence to generate testable hypotheses centered on the transition to agricultural, then industrialized, diets and its effects on rates of cancers,[17] T2DM,[18,19] heart disease,[20] and hypertension.[21] More recent mismatch studies have modeled differential nutritional outcomes of modern versus non-agricultural populations related to specific macro-[22-24] and micronutrients[25] and factors such as net dietary acidity,[26] diet breadth,[27,28] seasonality, physical activity,[29,30] and the production of toxic by-products by cell mitochondria.[31] These studies have ushered in a major paradigm shift by incorporating an explicitly evolutionary interpretation of human diet and its relationships to modern health crises,[32] framing explanations for the dramatic increases in obesity and diabetes incidence and prevalence as populations adopt Westernized diets and lifestyles[18,33,34] and highlighting the importance of recent changes in food availability and dietary breadth in generating disease risk.[18] This approach has also informed clinical interventions in at-risk populations: emerging research demonstrates that restricting refined carbohydrates and dairy products and emphasizing vegetables and lean proteins leads to encouraging reductions in fat mass, serum cholesterol and circulating glucose levels,[35,36] particularly in aboriginal populations.[37]

However, the evolutionary discordance model centers on assumptions that "our current gene pool is hardly changed from that of Stone Age humans,"[38: 26] and that "genetically, man remains adapted for the foods consumed [during the Paleolithic],"[39: 1] which has led to a

"dissociation between our genes and our lives."[40: 109] We argue that this perspective fuels perceptions that: 1) the diet of the evolutionary past, or EEA, was more or less universal, (2) the microevolutionary changes of the past twelve millennia were not sufficiently significant or adaptive to outweigh traits originating in the Paleolithic, and (3) human dietary behavior is determined primarily through instinctual and/or genetic mechanisms. All three of these assumptions are problematic, as the following discussion demonstrates.

Environments of Evolutionary Adaptation (EEA)

Though it may be reasonable to assume that our nutritional requirements, which are relatively universal across human populations, were established in the prehistoric past, the question of *which* prehistoric past is the relevant one remains.[41,42] Despite decades of critique attacking the notion of a single EEA as an overly simplistic view of dynamic, variable prehistoric environments and lifestyles,[12,43,44] proponents of the mismatch hypothesis rely on the idea of a single type of ancestral diet and place the mismatch between our evolved physiology and modern environments at the boundary between the Paleolithic and Neolithic eras with the introduction of agriculture. Yet, variation in ancestral diets has been thought for some time to be more than just a function of hunting-gathering vs. agriculture, but also a function of geography, food availability, seasonality and climatic conditions.[41] Paleoanthropological research into human diet evolution, drawing on analyses of preserved remains and materials, geochemical analyses, and modern human and non-human analogs, highlights the long-standing plurality and flexibility in human subsistence behavior.[45-49] Studies of modern hunter-gatherer populations further highlight the importance of social factors, such as reciprocal food sharing[50,51] and gendered divisions of subsistence labor and risk,[52] in shaping what was consumed and by whom.

Growing evidence further indicates that agricultural diets are not as easily associated with "diseases of civilization" as first thought. For example, the mismatch hypothesis assumes that prior to cultivation, hunter-gatherers obtained very few of their carbohydrates from cereal grains[9,32] and, because the carbohydrates from fruits and vegetables are somehow better than those from grains, were less likely to suffer from cardiometabolic diseases such as obesity, T2DM, and cardiovascular disease.[43] Modern hunter-gatherers and horticulturalists, however, have a wide range of carbohydrate intakes,[53,54] and even those relying on single cultivars high in carbohydrates remain free from many of the "diseases of civilization." Moreover, the boundaries between hunting-gathering and agriculture were likely quite fluid over much of the past 14,000 years,[55] indicating that mismatch was not an automatic, inevitable response to the move from predominant foraging to agriculture.[43]

On-going genetic variation

Emerging genomic evidence has called into question the assumption that human populations are essentially unchanged since the Paleolithic.[10] Recent studies reveal that humans have continued to evolve well into the Neolithic period, perhaps at accelerated rates relative to those of the Paleolithic.[56] Importantly, the most significant of these evolutionary changes are directly tied to changes in diet and subsistence,[57] including variation in the number of genes that code for amylase production depending on starch consumption[58] and the parallel evolution of lactase persistence in ancestral pastoralist populations.[59-62] In fact, current data

likely underestimate the number of independent alleles coding for lactase persistence,[62] meaning such mutations could have occurred at different points during human evolution and only became adaptive (rather than selectively neutral) *after* human cultures developed dairying practices. These examples represent only a few along a diverse spectrum of plausible evolutionary models linking specific environments to complex traits such as metabolism and cardiometabolic disease.[63]

Developmental and social flexibility in diet

Paradoxically, another limitation of the evolutionary discordance hypothesis is that it relies too heavily on human diets as a set of genetic adaptations. Numerous studies suggest that no "wisdom of the body" exists to drive food selection in direct response to physiological needs.[64-67] Instead, the majority of dietary behavior in omnivores is socially learned rather than instinctual, including the development of taste preferences and aversions,[68,69] definitions of what is "food" versus "not-food"[70,71] and patterns for combining different food resources.[72-74]

Research from the fields of nutrition and human biology within the last few decades has increasingly focused on developmental environments, rather than ancestral ones, as salient predictors of later metabolic disease. [75-79] Pre- and early post-natal nutritional cues may transmit important information about the expected energetic environment to the developing human, providing an important non-genomic form of inheritance that could enhance fitness during short-term environmental shifts. Fetal nutrition may also alter DNA methylation and chromatin modification, two key epigenetic processes contributing to gene expression,[80] creating differential risks for obesity[81] and possibly energy extraction and utilization.[82,83] Fetal imprinting and other epigenetic processes during development underscore the importance of fetal environments in shaping long-term body composition and metabolic health in ways that are not genetically determined.

Mechanisms Generating Flexibility in Human Diets

The genetic, physiological and behavioral mechanisms underlying human dietary and nutritional adaptations are far more complex and dynamic than a mismatch among Paleolithic bodies and post-Neolithic cultural environments. We argue through the three examples below that understanding the mechanisms that generate flexibility and diversity in human feeding behaviors and metabolic responses to new dietary niches is critical for fully interpreting modern human subsistence and dietary behavior in evolutionary context.

Neuroanatomical and Cognitive Mechanisms of Food Preferences and Diet

Human dietary behaviors are mediated in part by evolved processes in the brain that are shared with other primates.[34,57,84-92] Interestingly, the processing of food-related stimuli appears to occur independently of the hedonic value (liking versus disliking) of the foods consumed,[33,91] reinforcing the distinction between homeostatic (i.e., concerning energy balance) and hedonic (i.e., reward-seeking) mechanisms of feeding behavior, summarized in Figure 1.

Among humans, responses to food and eating situations are strongly associated with hedonic experiences[93] and emotional states considered critical to "ground[ing] social intelligence."[94] Therefore, much of human subsistence and eating behavior is produced by neural mechanisms linking emotional state, cultural context, and memory to the sensory properties of foods[94-96] and the anatomical structures involved in feeding. Merely thinking about one's favorite foods has been shown to activate brain centers for long-term and associative memory[97] and possibly the primary olfactory cortex.[98] Humans also appear to change their subjective ratings of the pleasantness of a food (i.e., to reach sensory-specific satiety) by merely chewing a food for a period of time, without swallowing it and thus without it reaching the stomach.[99] This cognitive feedback network generates the salient and often-intense associations that individuals experience between smells or tastes and memory or emotional state, independent of energy balance.[91] In addition, recent evidence from rats[100] suggests that fatty mouthfeel sensations detected in oral taste receptors trigger the release of endocannabinoids in the upper intestine via signaling by the vagus nerve; moreover, rat models demonstrate that sugar can act to change dopamine and opioid receptor binding in the nucleus accumbens of the brain, prompting a reward response that is similar to that observed in (and thus likely co-opted by) narcotics.[101] If identified in humans, such a relationship would further support the notion that taste, sensation, and associative memory form a complex feedback network that significantly shapes human taste preferences and feeding behavior in non-genomic ways.

Taste Perception, Food Choice and Social Learning

Adaptive models that consider the interaction of biological and cultural factors[73,102-105] have highlighted their dual importance in shaping human diets. For example, people tend to exhibit a liking of sweet tastes and a dislike of bitter tastes and irritants starting at birth,[106,107] which some suggest represents an adaptive proclivity for nutrient-dense, safe foods and an avoidance of poisonous plants and other harmful compounds in nature.[107-110] Accordingly, multiple alleles have been identified that mediate differences in human taste receptors, resulting in individual variation in the perception of sugars, fats, and bitter compounds.[89,108,111-114] However, circulating levels of leptin, a hormone involved in energy regulation, and sex steroids[115] also appear to influence sweet taste perception and preference in humans. The fact that sugars also activate a dopaminergic or reward response, noted above, has been implicated in possible links between chronic psychosocial stress and increased sensitivity to sweet tasting foods.[116] Studies highlighting the link between chronic social stress and elevations in circulating ghrelin, a hormone associated with energy depletion and food intake, further implicate psychosocial stress and increased consumption.[117] The combined effects of a stress-induced dopaminergic response and ghrelin elevation could well induce excessive consumption of sweets and other unhealthy foods. Results of these studies point to other physiological and environmental factors that may attenuate or even override genetic tendencies.

Importantly, the degree to which genetically-based preferences predict actual consumption behavior is unclear.[113,118] For example, bitter taste perception is indeed genetically-based,[111] but a demonstrated dislike for bitter tastes is not only dose-dependent,[119] its role in determining feeding behavior is belied by the worldwide popularity of coffee, tea,

chocolate, and hops. Even if initially disliked, repeated exposure has been shown to engender preferences for bitter tastes and irritant compounds (such as capsaicin in chili peppers) in humans and nonhuman primates.[120,121]

Food habits socially learned during critical periods of development can have lasting effects on aversions, preferences and choices throughout life.[67,122] Many highly salient taste associations and resultant eating behaviors are “learned” pre- and post-natally through exposure to flavor volatiles from garlic, mint, vanilla, carrot, anise and alcohol in amniotic fluid and breastmilk.[119,123] These exposures subsequently influence flavor acceptance, variety, and willingness to try new foods.[65,106,124] Conversely, food aversions appear universally to be learned in association with the negative physical consequences of eating a particular food, even altering the hedonic value of a previously-liked food to one of disgust.[69] Eating behaviors and preferences—both individual and collective—are further manipulated through the social environments in which individuals are immersed,[67,125,126] including ethnic tradition,[65,127] family experience,[125,126] and cultural practices.[128-131]

A classic body of anthropological research has revealed highly adaptive systems of behavior in cuisines.[73] The rules, combinations, processing techniques, and flavor themes that characterize different cuisines often reflect local ecological constraints[132] and provide the context in which food preferences are learned.[133,134] More recently, proponents of Niche Construction Theory[135]—an evolutionary framework that proposes that organisms cause evolutionary change through their creation of new environments due to their metabolism and behavior—have argued that cuisines also allow human groups to carve out wider niches than would exist without their manipulation,[136] making poisonous foods edible[137] and indigestible resources digestible,[138] staving off microbial contamination,[72] and achieving more complete nutrition.[74,104] Cuisines are thus components of larger socially-learned behavioral repertoires that have created significant selective processes on human populations through the creation of novel and widely varying dietary niches.[136,139,140]

Human Dietary Niches and Metabolism

Whereas prenatal diets appear critically important to shaping long-term physiology, metabolism, and feeding behavior, postnatal diets may also be distinctly important through the establishment and maintenance of the human gut microbiome. Composed of an estimated one hundred trillion microbes, this microniche plays important roles in digestion, immune function and nutrient production.[141] The human infant is born with a sterile intestine and experiences rapid bacterial colonization during birth, breastfeeding, and solid food supplementation; these early exposures shape lifelong patterns of gut colonization.[141-143] Humans, like most mammals, are colonized by relatively few bacterial phyla, reflecting a long history of microbial niche stability likely associated with broad dietary patterns.[144,145] However, human populations tend to show excess phylotypic diversity at the species and strain level[145] and patterned diversity among families and local communities.[146] These patterns suggest that local environments and diets may result in the development of distinct intestinal microbiomes in different human populations.

Since one important function of gut bacteria is the metabolism of indigestible polysaccharides into simple sugars and short-chain fatty acids,[141] the disruption of co-

evolved mutualism between human populations and gut microbiota may be a contributing factor to the increasing prevalence of chronic and degenerative diseases[145] and risk of obesity.[147,148] Moreover, recent work suggests that high-fat diets may promote obeseogenic bacterial communities in the gut, promoting inflammation, insulin resistance and diabetes.[149] These examples may point to a mismatch between humans and their recent dietary environment, but they also highlight the dynamic and varied nature of gut microbiomes, and the critical link between created dietary niches and human metabolic physiology.

New evolutionary frameworks for understanding diet

The above sections detail the various ways in which human dietary habits and resulting metabolic states are deeply ingrained through various forms of environmental exposure and social learning, well before birth and continually throughout early life. The result is that one must differentiate when describing behavior that is inborn versus instinctive, imprinted versus genomic, unconscious versus hard-wired. Whilst an enormous amount of insight is gained from reconstructing hunter-gatherer subsistence and nutrition in deep antiquity, we argue that using these data as foundations of an evolutionary paradigm aiming to inform modern nutrition interventions is insufficient for generating meaningful and sustainable policies. Moreover, the use of Paleolithic subsistence patterns as a *de facto* standard up to which all subsequent human subsistence should be measured is impractical for a number of reasons.

Arguments that the evolutionary discordance hypothesis provides the “unifying hypothesis on which to build a dietary strategy for prevention”[39: 1] needed to fix modern nutrition crises advocate for one overarching strategy based on one, or a relative few,[32] set(s) of broad genetic and behavioral foundations. These arguments assume that human diets are primarily influenced by genetic traits selected for Paleolithic living, when, instead, much of human subsistence is guided by behavioral flexibility contingent on local ecologies and social learning, and therefore detached from a particular evolutionary environment. Adding to this counterpoint are the valid critiques that its focus on meat and fish as the desirable proteins[32] is ecologically unsustainable given the current and projected global human population,[150,151] and that an emphasis on lean meat, preferably from free-ranged or wild animals, and vegetables is economically unviable for many of the populations that experience the highest rates of obesity and metabolic disease.[15] Therefore, the Paleolithic diet, when taken as the best option for optimal metabolic health, results in a limited view of both human dietary behavior and the modern structural barriers limiting subsistence choice in the most high-risk populations.

Potential Nutrition Interventions

Basing policies and interventions on an evolutionary framing requires expanding the list of evolved traits and mechanisms that shape the bulk of human eating behavior. Here we provide several illustrative examples of ways in which a more developmental, mechanistic, and behavioral perspective could provide effective strategies for intervention that do not rely on an assumption of evolutionary discordance.

1. Rethinking the Human Sweet Tooth

The assumption that humans evolved an affinity for sweet and fatty tastes that is highly adaptive but mismatched to modern contexts might reasonably lead one to conclude that unchecked consumption of sugary and high-fat foods is something of an inevitability. However, a wider perspective focuses on the mechanisms of an affinity for sweet and fatty tastes rather than ending the explanation with a discordant adaptation. Humans learn to like sugar along with a host of other flavors *in utero*; moreover, sugars are associated with the secretion of endogenous opiates that confer pleasurable sensations and activate reward pathways in the brain. Similarly, the consumption of fatty foods stimulates the production of endogenous cannabinoids that create comparable reward effects.[100] In modern environments characterized by cheap, readily available sugary and fatty foods,[12,15] and psychosocial stress that is both uniquely human[152] and differentially endured,[153] an unchecked consumption of sugars and high-fat foods could more reasonably reflect socially learned and socially reinforced behaviors than an adaptation gone awry.[117,154]

Intervention strategies based on this broader perspective would not assume that removal of sugars, other simple carbohydrates, and excessive saturated fats from the diet is necessary because they trigger a mismatch born of adaptation. Instead, interventions could focus on manipulating the intrauterine flavor-scape or early life diets to impart an affinity to a broader range of taste stimuli unrelated to sweet tastes. Plant-based spices and aromatics can play a significant role in positive associations with foods based on flavor & olfactory properties; these associations would be unrelated to fat or caloric content, and could therefore make them useful tools in shaping children's preferences for plant-rich diets. Importantly, interventions aimed at preventing metabolic diseases could also benefit from focusing as strongly on reducing sources of psychosocial stress as on controlling food intake.

2. Broadening the Genetic Scope

Moving away from a paradigm of Paleolithic dietary profiles might open up more options for healthy diet recommendations based on understandings of Neolithic adaptations as well. For example, decades of research have failed to identify the hypothetical "thrifty gene" first suggested by Neel[16] as a pre-agricultural adaptation responsible for high rates of diabetes among Native American and aboriginal Australian populations. This lack of evidence has also prompted criticism that the emphasis on genetic mechanisms also ignores social and economic barriers to improved nutrition as the likelier culprits.[155] However, recognizing that aboriginal populations may not have a "thrifty gene," but instead have fewer amylase copy number variants, might permit focused dietary interventions based on genetic screening for the *absence* of a Neolithic adaptation rather than the *presence* of a Paleolithic one. Differences in salivary amylase concentrations might also influence the composition of digested food that enters the intestinal tract in ways that could promote the growth of obeseogenic bacterial communities in contexts of starch-heavy diets.

3. Harnessing the Human Microbiomes

Attending to the central role of constructed microniches within the human body, and their interaction with aspects of human metabolism such as the amylase concentration example above, may prove more feasible than only attending to high-fat diets alone. Shifting the

focus of inquiry in this manner could lead to the development of prebiotic and probiotic supplements or diets that are specifically tailored to promote particular species of beneficial microflora[156] as part of long-term diet modification. These broader perspectives, still rooted in evolutionary medicine but equally focused on physiological mechanisms and behavioral flexibility, provide loci for intervention that do not focus on hunter-gatherer diets heavy in lean meats or seafood, the latter of which is becoming a particularly unsustainable form of subsistence.[157] The rising interest in therapeutic use of probiotics to address obesity[158] and related metabolic diseases would thus benefit from this broader evolutionary framework.

Conclusions

Almost three decades after the evolutionary discordance hypothesis was put forth, worldwide obesity, heart disease, and T2DM statistics continue to skyrocket; these trends do not suggest that this hypothesis is incorrect, but rather incomplete in relying primarily on genetic understandings of human diet and the assumption of Paleolithic life as the human evolutionary standard. The ability to use evolutionary medicine in multiple applications for diet in addition to the evolutionary discordance hypothesis by itself would therefore open up new avenues for intervention in populations whose dietary options are already constrained by structural and economic barriers to resources like fresh produce and lean protein.[10] Certainly an acknowledgement of both the long-standing diversity and socially learned mechanisms in human diets shifts the focus away from the “lifestyle factors” of individuals living at odds from their evolutionary past. It does not challenge research showing that a diet rich in plant materials and lean proteins is beneficial to health; it simply questions the extent to which this diet is unequivocally Paleolithic in nature[159], and the extent to which consuming this diet is somehow hard-wired in human genes. It also underscores the importance of increasing nutrition interventions for pregnant women and children, and broadening the variables that are manipulated in these interventions, as a long-term investment in reducing the heavy burden of diet-related health care costs.[99] Such emphasis on flexibility and social context would serve as an important counter-point to the blanket prescription of an “evolutionarily appropriate diet.”

The evolutionary discordance hypothesis has provided a valuable theoretical framework for studying human diet in an evolutionary context, but its focus on a single model of human ancestral diets, and its assumption that cultural evolution outpacing genetic evolution is a fundamental cause of disease in the modern world, have resulted in an incomplete view of the flexibility and variability in human dietary behavior and health in the past and present. A growing body of scholarly data suggests that no such thing as an evolved human diet exists, and that popular notions of returning to a diet that is more true to human nature are inconsistent with the ways in which humans metabolisms and eating habits develop. Much of the story of human evolution is about hominin populations learning about and manipulating resources in their environment to more effectively meet their nutritional needs and hedonic wants; understanding the versatile and generative nature of human diet evolution provides a more nuanced and productive avenue to promoting optimal nutrition. It also provides new avenues for practical intervention and long-term improvements in nutrition among at-risk populations, a necessary step for not only comprehending this

fundamental aspect of human behavior, but more comprehensively applying it to modern settings.

Acknowledgments

The authors are grateful to George Armelagos, Molly Zuckerman, Sarah Barks, Jennifer Mascaro, Anna Agbe-Davies, Jocelyn Chua, Jean Dennison, Silvia Tomásková and two anonymous reviewers for their helpful comments on earlier versions of this manuscript.

References

1. Mokdad AH, Bowman BA, Ford ES, Vinicor F, Marks JS, Koplan JP. The Continuing Epidemics of Obesity and Diabetes in the United States. Journal of the American Medical Association (JAMA). 2001; 286(10):1195–1200.
2. Fonseca VA, Zinman B, Nauck MA, Goldfine AB, Plutzky J. Confronting the Type 2 Diabetes Epidemic: The Emerging Role of Incretin-Based Therapies. Am J Med. 2010; 123(7):S2–S10. [PubMed: 20609666]
3. World Health Organization. [Accessed December 13, 2011] Obesity and Overweight Factsheet. WHO Global Strategy on Diet, Physical Activity and Health. 2003. http://www.who.int/mediacentre/factsheets/fs311/en/
4. Audette, RV.; Gilchrist, T. NeanderThin: Eat Like a Caveman to Achieve a Lean, Strong, Healthy Body. Paleolithic Press; Dallas: 1995.
5. d'Adamo, PJ.; Whitney, C. Eat Right 4 Your Type: The Individualized Diet Solution to Staying Healthy, Living Longer & Achieving Your Ideal Weight. 1st Edition. Putnam Adult; New York: 1996.
6. Durant, J. Hunter-Gatherer: Health and Human Nature from an Evolutionary Perspective. 2010. http://www.hunter-gatherer.com/about
7. Horne, R. Health & Survival in the 21st Century. HarperCollins Australia; Pymble: 1997.
8. People for the Ethical Treatment of Animals. [Accessed August 5, 2010] Health Issues//The Natural Human Diet: Early Human Evolution. 2010. http://www.goveg.com/naturalhumandiet_evolution.asp
9. Eaton SB, Konner M. Paleolithic Nutrition: A Consideration of Its Nature and Current Implications. New England Journal of Medicine. 1985; 312(5):283–289. [PubMed: 2981409]
10. Knight C. "Most People are Simply Not Designed to Eat Pasta": Evolutionary Explanations for Obesity in the Low-Carbohydrate Diet Movement. Public Understanding of Science. 2011:1–14. in press. DOI: 10.1177/0963662510391733.
11. Eaton SB, Konner MJ, Shostak M. Stone Agers in the Fast Lane: Chronic Degenerative Diseases in Evolutionary Perspective. Am J Med. 1988; 84(4):739–749. [PubMed: 3135745]
12. Drewnowski A. Nutrition Transition and Global Dietary Trends. Nutrition. 2000; 16(7/8):486–487. [PubMed: 10906531]
13. Kuzawa CW, Sweet E. Epigenetics and the Embodiment of Race: Developmental Origins of US Racial Disparities in Cardiovascular Health. American Journal of Human Biology. 2009; 21:2–15. [PubMed: 18925573]
14. Armelagos GJ, Brown PJ, Turner BL. Evolutionary, Historical and Political Economic Perspectives on Health and Disease. Social Science and Medicine. 2004 In Press.
15. Nestle M, Wing R, Birch L, et al. Behavioral and Social Influences on Food Choice. Nutrition Reviews. 1998; 56(5):S50–S74. [PubMed: 9624880]
16. Neel JV. Diabetes mellitus: a "thrifty" genotype rendered detrimental by "progress"? Am J Hum Genet. Dec.1962 14:353–362. [PubMed: 13937884]
17. Davis DL. Paleolithic Diet, Evolution, and Carcinogens. Science. 1987; 238(4834):1633–1634. [PubMed: 3120316]
18. Lieberman LS. Dietary, Evolutionary, and Modernizing Influences on the Prevalence of Type 2 Diabetes. Annual Review of Nutrition. 2003; 23:345–377.

19. Lieberman LS. Evolutionary and Anthropological Perspectives on Optimal Foraging in Obesogenic Environments. Appetite. 2006; 47:3–9. [PubMed: 16806580]
20. O'Keefe JHJ, Cordain L. Cardiovascular Disease Resulting from a Diet and Lifestyle at Odds With Our Paleolithic Genome: How to Become a 21st-Century Hunter-Gatherer. Mayo Clinic Proceedings. 2004; 79(1):101–108. [PubMed: 14708953]
21. Jackson FL. An Evolutionary Perspective on Salt, Hypertension, and Human Genetic Variability. Hypertension. 1991; 17(Suppl.):129–132.
22. Cordain L, Watkins BA, Mann NJ. Fatty acid composition and energy density of foods available to African hominids. Evolutionary implications for human brain development. World Review of Nutrition & Dietetics. 2001; 90:144–161. [PubMed: 11545040]
23. Eaton, SB. Fibre Intake in Prehistoric Times. In: Leads, AR., editor. Dietary Fibre Perspectives: Reviews and Bibliography. Vol. 2.. John Libbey; London: 1990. p. 27-40.
24. Eaton SB. Humans, lipids and evolution. Lipids. 1992; 27(10):814–820. [PubMed: 1435101]
25. Eaton SB, Nelson DA. Calcium in evolutionary perspective. American Journal of Clinical Nutrition. 1991; 54:281S–287S. [PubMed: 2053574]
26. Sebastian A, Frassetto LA, Sellmeyer DE, Merriam RL, Morris RCJ. Estimation of the Net Acid Load of the Diet of Ancestral Preagricultural Homo sapiens and their Hominid Ancestors. American Journal of Clinical Nutrition. 2002; 76(6):1308–1316. [PubMed: 12450898]
27. Bogin, B. The Evolution of Human Nutrition. In: Romanucci-Ross, L.; Moerman, D.; Tancredi, LR., editors. The Anthropology of Medicine. Bergen and Garvey; South Hadley, MA: 1998. p. 96-142.
28. Turner, BL.; Maes, K.; Sweeney, JL.; Armelagos, GJ. Human Evolution, Diet and Nutrition: Where the Body Meets the Buffet. In: Trevathan, W.; Smith, EO.; McKenna, JJ., editors. New Perspectives in Evolutionary Medicine. Oxford University Press; Oxford: 2008. p. 55-71.
29. Chakravarthy MV, Booth FW. Eating, Exercise, and "Thrifty" Genotypes: Connecting the Dots Toward an Evolutionary Understanding of Modern Chronic Diseases. Journal of Applied Physiology. 2003; 96(1):3–10. [PubMed: 14660491]
30. Cordain L, Gotshall RW, Eaton SB, Eaton SB 3rd. Physical activity, energy expenditure and fitness: an evolutionary perspective. Int J Sports Med. 1998; 19(5):328–335. [PubMed: 9721056]
31. Wallace DC. A Mitochondrial Paradigm of Metabolic and Degenerative Diseases, Aging, and Cancer: A Dawn for Evolutionary Medicine. Annual Review of Genetics. 2005; 39:359–410.
32. Konner M, Eaton SB. Paleolithic Nutrition: Twenty-Five Years Later. Nutrition in Clinical Practice. 2010; 25(6):594–602. [PubMed: 21139123]
33. Rolls ET, Sienkiewicz ZJ, Yaxley S. Hunger Modulates the Responses to Gustatory Stimuli of Single Neurons in the Caudolateral Orbitofrontal Cortez of the Macaque Monkey. European Journal of Neuroscience. 1989; 1(1):53–60. [PubMed: 12106174]
34. Rolls ET, Yaxley S, Sienkiewicz ZJ. Gustatory Responses of Single Neurons in the Caudolateral Orbitofrontal Cortex of the Macaque Monkey. Journal of Neurophysiology. 1990; 64(4):1055–1066. [PubMed: 2258734]
35. Frassetto LA, Schloetter M, Mietus-Snyder M, Morris RCJ, Sebastian A. Metabolic and Physiologic Improvements from Consuming a Paleolithic, Hunger-Gatherer Type Diet. Eur J Clin Nutr. 2009; 63:947–955. [PubMed: 19209185]
36. Lindeberg S, Jönsson T, Granfeldt Y, et al. A Paleolithic Diet Improves Glucose Tolerance More than a Mediterranean-like Diet In Individuals With Ischaemic Heart Disease. Diabetologia. 2007; 50:1795–1807. [PubMed: 17583796]
37. O'Dea K. Marked Improvement in Carbohydrate and Lipid Metabolism in Diabetic Australian Aborigines After Temporary Reversion to Traditional Lifestyle. Diabetes. 1984; 33:596–603. [PubMed: 6373464]
38. Eaton SB, Cordain L. Evolutionary Aspects of Diet: Old Genes, New Fuels. World Review of Nutrition & Dietetics. 1997; 81:26–37. [PubMed: 9287501]
39. Eaton SB. The Ancestral Human Diet: What Was It and Should It Be A Paradigm for Contemporary Nutrition? Proceedings of the Nutrition Society. 2006; 65:1–6. [PubMed: 16441938]

40. Eaton, SB.; Eaton, SB., III; Cordain, L. Evolution, Diet, and Health. In: Ungar, PS.; Teaford, MF., editors. Human Diet: Its Origin and Evolution. Bergin & Garvey; Westport, CT: 2002. p. 7-17.
41. Garn SM, Leonard WR. What did our ancestors eat? Nutrition Reviews. 1989; 47(11):337–345. [PubMed: 2689923]
42. Milton K. Nutritional characteristics of wild primate foods: do the diets of our closest living relatives have lessons for us? Nutrition. 1999; 15(6):488–498. [PubMed: 10378206]
43. Elton, S. Environments, Adaptation, And Evolutionary Medicine: Should We Be Eating a Stone Age Diet?. In: O'Higgins, P.; Elton, S., editors. Medicine and Evolution: Current Applications, Future Prospects. CRC Press; 2008. p. 9-33.
44. Potts R. Variability Selection in Hominid Evolution. Evolutionary Anthropology. 1998; 7:81–96.
45. Casimir, MJ. Basic Human Nutritional Needs. In: Casimir, MJ., editor. Flocks and Food: A Biocultural Approach to the the Study of Pastoral Foodways. Verlag, Koln, Weimar & Wien; Bohlau: 1991. p. 47-72.
46. Leonard WR, Stock JT, Velggia CR. Evolutionary Perspectives on Human Diet and Nutrition. Evolutionary Anthropology. 2010; 19:85–86.
47. Ungar, PS., editor. Evolution of the Human Diet: The Known, The Unknown, and the Unknowable. Oxford University Press; New York: 2007.
48. Ungar PS, Grine FE, Teaford MF. Diet in Early Homo: A Review of the Evidence and a New Model of Adaptive Versatility. Annual Review of Anthropology. 2006; 35:209–228.
49. Ungar PS, Sponheimer M. The Diets of Early Hominins. Science. 2011; 334:190–193. [PubMed: 21998380]
50. Hill K, Kaplan H. On Why Male Foragers Hunt and Share Food. Current Anthropology. 1993; 34(5):701–710.
51. Hill K, Kaplan H, Hawkes K, Hurtado AM. Foraging Decisions among Ache Hunter-Gatherers - New Data and Implications for Optimal Foraging Models. Ethology and Sociobiology. 1987; 8(1): 1–36.
52. Hurtado AM, Hawkes K, Hill K, Kaplan H. Female Subsistence Strategies among Ache Hunter Gatherers of Eastern Paraguay. Human Ecology. 1985; 13(1):1–28.
53. Milton K. Back to Basics: Why Foods of Wild Primates Have Relevance for Modern Human Health. Nutrition. 2000; 16(7/8):480–483. [PubMed: 10906529]
54. Strohle A, Hahn A. Diets of modern hunter-gatherers vary substantially in their carbohydrate content depending on ecoenvironments: results from an ethnographic analysis. Nutrition Research. 2011; 31(201):429–435. [PubMed: 21745624]
55. Pringle H. The Slow Birth of Agriculture. Science. 1998; 20(282):5393.
56. Hawks J, Wang ET, Cochran GM, Harpending HC, Moyzis RK. Recent Acceleration of Human Adaptive Evolution. Proceedings of the National Academy of Sciences. 2007; 104(52):20753–20758.
57. Rolls BJ, Rolls ET, Rowe EA, Sweeney K. Sensory Specific Satiety in Man. Physiology & Behavior. 1981; 27(1):137–142. [PubMed: 7267792]
58. Perry GH, Dominy NJ, Claw KG, et al. Diet and the evolution of human amylase gene copy number variation. Nat Genet. Oct; 2007 39(10):1256–1260. [PubMed: 17828263]
59. Holden C, Mace R. Phylogenetic Analysis of the Evolution of Lactose Digestion in Adults. Human Biology. 1997; 69:605–628. [PubMed: 9299882]
60. Aoki K. A Stochastic Model of Gene-Culture Coevolution Suggested by the "Culture Historical Hypothesis" for the Evolution of Adult Lactose Absorption in Humans. Proceedings of the National Academy of Sciences. 1986; 83:2929–2933.
61. Tishkoff SA, Reed FA, Ranciaro A, et al. Convergent Adaptation of Human Lactase Persistence in Africa and Europe. Nature Genetics. 2007; 39:31–40. [PubMed: 17159977]
62. Itan Y, Jones BL, Ingram CJE, Swallow DM, Thomas MG. A Worldwide Correlation of Lactase Persistence Phenotypes and Genotypes. BMC Evolutionary Biology. 2010; 10(36):1–11. [PubMed: 20044934]
63. Di Rienzo A, Hudson RR. An evolutionary framework for common disease: the ancestral-susceptibility model. TRENDS in Genetics. 2005; 21(11):596–601. [PubMed: 16153740]

64. Leathwood, PD.; Ashley, DV. Behavioural strategies in the regulation of food choice. In: Mauron, J., editor. Nutritional Adequacy, Nutrient Availability and Needs. Vol. 44. Birkhauser Verlag; Basel: 1983. p. 171-196.
65. Hudson R, Distel H. The flavor of life: perinatal development of odor and taste preferences. Schweiz Med Wochenschr. 1999; 129:176–181. [PubMed: 10081075]
66. Galef BG Jr. A Contrarian View of the Wisdom of the Body as It Relates to Dietary Self-Selection. Psychological Review. 1991; 98(2):218–223. [PubMed: 2047513]
67. Birch, LL.; Fisher, JA. The Role of Experience in the Development of Children's Eating Behavior. In: Capaldi, ED., editor. Why We Eat What We Eat: The Psychology of Eating. American Psychological Association; Washington, DC: 1996. p. 113-141.
68. Bernstein IL. Taste Aversion Learning: A Contemporary Perspective. Nutrition, Immunology, Neuroscience, and Behavior. 1999; 15(3):229–234.
69. Pelchat ML, Rozin P. The Special Role of Nausea in the Acquisition of Food Dislikes by Humans. Appetite: Journal for Intake Research. 1982; 3:341–351.
70. Galef BG. Social Factors in Diet Selection and Poison Avoidance by Norway Rats: A Brief Review. Appetite. 1989; 12:73–73.
71. Galef, BG. Social Influences on Food Preferences and Feeding Behaviors of Vertebrates. In: Capaldi, ED., editor. Why We Eat What We Eat: The Psychology of Eating. American Psychological Association; Washington, DC: 1996. p. 207-231.
72. Billing J, Sherman PW. Antimicrobial functions of spices: why some like it hot. Q Rev Biol. 1998; 73(1):3–49. [PubMed: 9586227]
73. Farb, P.; Armelagos, GJ. Consuming Passions: The Anthropology of Eating. Houghton Mifflin and Company; Boston: 1980.
74. Katz, SH. Food, Behavior, and Biocultural Evolution. In: Barker, LM., editor. The Psychobiology of Human Food Selection. AVI Publishing Company, Inc.; Westport, CT: 1982. p. 171-188.
75. Hales CN, Barker DJ. Type 2 (non-insulin-dependent) diabetes mellitus: the thrifty phenotype hypothesis. Diabetologia. Jul; 1992 35(7):595–601. [PubMed: 1644236]
76. Crystal SR, Bowen DJ, Bernstein IL. Morning Sickness and Salt Intake, Food Cravings, and Food Aversions. Physiology & Behavior. 1999; 67(2):181–187. [PubMed: 10477048]
77. Bayol SA, Farrington SJ, Stickland NC. A maternal 'junk food' diet in pregnancy and lactation promotes an exacerbated taste for 'junk food' and a greater propensity for obesity in rat offspring. Br J Nutr. Oct; 2007 98(4):843–851. [PubMed: 17697422]
78. Godfrey KM, Barker DJ. Fetal nutrition and adult disease. Am J Clin Nutr. May; 2000 71(5 Suppl):1344S–1352S. [PubMed: 10799412]
79. Gluckman PD, Cutfield W, Hofman P, Hanson MA. The fetal, neonatal, and infant environments —the long-term consequences for disease risk. Early Hum Dev. 2005; 81(1):51–59. [PubMed: 15707715]
80. Reik W, Constancia M, Fowden A, et al. Regulation of supply and demand for maternal nutrients in mammals by imprinted genes. J Physiol. Feb 15; 2003 547(Pt 1):35–44. [PubMed: 12562908]
81. Waterland RA, Jirtle RL. Transposable elements: targets for early nutritional effects on epigenetic gene regulation. Mol Cell Biol. Aug; 2003 23(15):5293–5300. [PubMed: 12861015]
82. Haig D, Graham C. Genomic imprinting and the strange case of the insulin-like growth factor II receptor. Cell. Mar 22; 1991 64(6):1045–1046. [PubMed: 1848481]
83. Haig D, Wharton R. Prader-Willi syndrome and the evolution of human childhood. Am J Human Biol. May-Jun;2003 15(3):320–329. [PubMed: 12704708]
84. Arana FS, Parkinson JA, Hinton E, Holland AJ, Owen AM, Roberts AC. Dissociable Contributions of the Human Amygdala and Orbitofrontal Cortex to Incentive Motivation and Goal Selection. The Journal of Neuroscience. 2003; 23(29):9632–9638. [PubMed: 14573543]
85. Shepherd GM. Smell Images and the Flavour System in the Human Brain. Nature. 2006; 444:316–326. [PubMed: 17108956]
86. de Araujo IET, Rolls ET, Kringelbach ML, McGlone F, Phillips N. Taste-olfactory convergence, and the representation of the pleasantness of flavour, in the human brain. European Journal of Neuroscience. 2003; 18:2059–2068. [PubMed: 14622239]

87. LaBar KS, Gitleman DR, Parrish TB, Kim Y-H, Nobre AC, Mesulam M-M. Hunger Selectively Modulates Corticolimbic Activation to Food Stimuli in Humans. Behavioral Neuroscience. 2001; 115(2):493–500. [PubMed: 11345973]

88. Morris JS, Dolan RJ. Involvement of Human Amygdala and Orbitofrontal Cortex in Hunger-Enhanced Memory for Food Stimuli. The Journal of Neuroscience. 2001; 21(4):5304–5310. [PubMed: 11438606]

89. Reed DR, Tanakab T, McDaniel AH. Diverse Tastes: Genetics of Sweet and Bitter Perception. Physiology & Behavior. 2006; 88(3):215–226. [PubMed: 16782140]

90. Rolls ET. The Functions of the Orbitofrontal Cortex. Brain and Cognition. 2004; 55:11–29. [PubMed: 15134840]

91. Rolls ET. Brain Mechanisms Underlying Flavour and Appetite. Philosophical Transactions of the Royal Society B: Biological Sciences. 2006; 361(1471):1123–1136.

92. Rolls ET, Kringelbach ML, De Araujo IET. Different Representations of Pleasant and Unpleasant Odors in the Human Brain. European Journal of Neuroscience. 2003; 3:695–703. [PubMed: 12911766]

93. Kringelbach ML. The Human Orbitofrontal Cortex: Linking Reward to Hedonic Experience. Nature Reviews Neuroscience. 2005; 6:691–702.

94. Worthman CM. Habits of the Heart: Life History and the Developmental Neuroendocrinology of Emotion. American Journal of Human Biology. 2009; 21:772–781. [PubMed: 19621433]

95. Bechara A, Damasio H, Damasio AR. Emotion, Decision Making and the Orbitofrontal Cortex. Cerebral Cortex. 2000; 10(3):295–307. [PubMed: 10731224]

96. Damasio, AR. Descartes' Error: Emotion, Reason and the Human Brain. G.P. Putnam's Sons; New York: 1994.

97. Bird CM, Burgess N. The Hippocampus and Memory: Insights from Spacial Processing. Nature Reviews Neuroscience. 2008; 9:182–194.

98. de Araujo IE, Rolls ET, Velazco MI, Margot C, Cayeux I. Cognitive Modulation of Olfactory Processing. Neuron. 2005; 46(4):671–679. [PubMed: 15944134]

99. Rolls ET, Rolls JH. Olfactory Sensory-Specific Satiety in Humans. Physiology & Behavior. 1997; 61(3):461–473. [PubMed: 9089767]

100. DiPatrizio NV, Astarita G, Schwartz G, Li X, Piomelli D. Endocannabinoid Signal in the Gut Controls Dietary Fat Intake. Proceedings of the National Academy of Sciences. 2011 in press. doi 10.1073/pnas.1104675108.

101. Avena NM, Rada P, Hoebel BG. Evidence for Sugar Addiction: Behavioral and Neurochemical Effects of Intermittent, Excessive Sugar Intake. Neuroscience & Behavioral Reviews. 2008; 32(1):20–39.

102. Mintz, SW. Tasting Food, Tasting Freedom: Excursions into Eating, Culture and the Past. Beacon Press; Boston, MA: 1996.

103. Harris, M.; Ross, E., editors. Food and Evolution: Toward a Theory of Human Food Habits. Temple University Press; Philadelphia: 1987.

104. Stinson S. Nutritional Adaptation. Annual Review of Anthropology. 1992; 21:143–170.

105. Katz, SH. Food and Biocultural Evolution: A Model for the Investigation of Modern Nutritional Problems. In: Johnston, FE., editor. Nutritional Anthropology. Alan R. Liss, Inc; New York: 1987. p. 41-63.

106. Blass EM, Shide DJ, Weller A. Suckling: Opioid and Non-opioid Processes in Mother-Infant Bonding. Appetite. 1989; 12:75.

107. Anderson GH. Sugars, Sweetness, and Food Intake. The American Journal of Clinical Nutrition. 1995; 62(1):195S–202S. [PubMed: 7598077]

108. Kim, U-k; Wooding, S.; Riaz, N.; Jorde, LB.; Drayna, D. Variation in the Human TAS1R Taste Receptor Genes. Chemical Senses. 2006; 31(7):599–611. [PubMed: 16801379]

109. Wang X, Puram S. The Toughness of Cortical Bone and Its Relationship with Age. Annals of Biomedical Engineering. 2004; 32(1):123–135. [PubMed: 14964728]

110. Yamaguchi S, Ninomiya K. Umami and Food Palatability. Journal of Nutrition. 2000; 130:921S–926S. [PubMed: 10736353]

111. Montmayeur JP, Matsunami H. Receptors for Bitter and Sweet Taste. Current Opinion in Neurobiology. 2002; 12(4):366–371. [PubMed: 12139982]
112. Eny KM, Wolever TMS, Corey PN, El-Sohemy A. Genetic Variation in TAS1R2 (Ile191Val) is Associated with Consumption of Sugars in Overweight and Obese Individuals in 2 Distinct Populations. American Journal of Clinical Nutrition. 2010; 92(6):1501–1510. [PubMed: 20943793]
113. Reed DR, McDaniel AH. The Human Sweet Tooth. BMC Oral Health. 2006; 6(Suppl 1):S17. [PubMed: 16934118]
114. Bartoshuk LM. Comparing Sensory Experiences Across Individuals: Recent Psychophysical Advances Illuminate Genetic Variation in Taste Perception. Chemical Senses. 2000; 25(4):447–460. [PubMed: 10944509]
115. Than TT, Delay ER, Maler ME. Sucrose Threshold Variation During the Menstrual Cycle. Physiology & Behavior. 1994; 56(2):237–239. [PubMed: 7938232]
116. Striegel-Moore RH, Morrison JA, Schrieber G, Schumann BC, Crawford PB, Obarzanek E. Emotion-Induced Eating and Sucrose Intake in Children: The NHLBI Growth and Health Study. International Journal of Eating Disorders. 1999; 25(4):389–398. [PubMed: 10202649]
117. Lutter M, Nestler EJ. Homeostatic and Hedonic Signals Interact in the Regulation of Food Intake. The Journal of Nutrition. 2009; 139(3):629–632. [PubMed: 19176746]
118. Sclafani A. Oral, Post-Oral and Genetic Interactions in Sweet Appetite. Physiology & Behavior. 2006; 89(4):525–530. [PubMed: 16647093]
119. Mennella JA, Beauchamp GK. Early Flavor Experience: Research Update. Nutrition Reviews. 1998; 56(7):205–211. [PubMed: 9697386]
120. Rozin P, Kennel K. Acquired Preferences for Piquant Foods by Chimpanzees. Appetite: Journal for Intake Research. 1983; 4:69–77.
121. Gartstein M, Rothbart M. Studying infant temperament via the Revised Infant Behavior Questionnaire. Infant Behavior and Development. 2003; 26:64–86.
122. Casey R, Rozin P. Changing Children's Food Preferences: Parent Opinions. Appetite. 1989; 12:171–182. [PubMed: 2774551]
123. Schaal B, Marlier L, Soussignan R. Human Foetuses Learn Odours from their Pregnant Mother's Diet. Chemical Senses. 2000; 25:729–737. [PubMed: 11114151]
124. Wright, P. Hunger, Satiety, and Feeding Behavior in Early Infancy. In: Boakes, RA.; Popplewell, DA.; Burton, MJ., editors. Eating Habits: Food, Physiology and Learned Behaviour. John Wiley & Sons; Chichester: 1987. p. 75-106.
125. Birch, LL. The acquisition of food acceptance patterns in children. In: Boakes, RA.; Popplewell, DA.; Burton, MJ., editors. Eating Habits: Food, physiology and learned behaviour. John Wiley & Sons; Chichester: 1987. p. 107-130.
126. Rozin P, Millman L. Family Environment, Not Heredity, Accounts for Family Resemblances in Food Preferences and Attitudes: A Twin Study. Appetite. 1987; 8:125–134. [PubMed: 3592649]
127. Rozin, P. Sociocultural influences on human food selection. In: Capaldi, ED., editor. Why We Eat What We Eat: The Psychology of Eating. American Psychological Association; Washington, DC: 1996. p. 233-263.
128. Messer E. Some Like it Sweet: Estimating Sweetness Preferences and Sucrose Intakes from Ethnographic and Experimental Data. American Anthropologist. 1986; 88:637–647.
129. Mela DJ. Food choice and intake: the human factor. Proceedings of the Nutrition Society. 1999; 58:513–521. [PubMed: 10604182]
130. Lupton D. Food, memory and meaning: the symbolic and social nature of food events. The Sociological Review. 1994:664–685.
131. Simoons, FJ. Chapter 1: Introduction. In: Simoons, FJ., editor. Eat Not This Flesh: Food Avoidances from Prehistory to the Present. University of Wisconsin Press; 1994. p. 3-12.
132. Harris M. India's Sacred Cow. Human Nature. 1978:28–36.
133. Rozin, E. The Structure of Cuisine. In: Barker, LM., editor. The Psychobiology of Human Food Selection. AVI Publishing Company, Inc.; Westport, CT: 1982. p. 189-203.

134. Rozin, P. Human Food Selection: The Interaction of Biology, Culture and Individual Experience. In: Barker, LM., editor. The Psychobiology of Human Food Selection. AVI Publishing Company, Inc.; Westport, CT: 1982. p. 225-254.
135. Ogden-Smee, FJ.; Laland, KN.; Feldman, MW. Niche Construction: The Neglected Process in Evoultion. Princeton University Press; Princeton, NJ: 2003.
136. Wollonstonecroft MM. Investigating the Role of Food Processing in Human Evolution: A Niche Construction Approach. Archaeological and Anthropological Science. 2011; 3:141–150.
137. Dufour, DL. A Closer Look At the Nutritional Implications of Bitter Cassava Use. In: Sponsel, L., editor. Indigenous Peoples And The Future of Amazonia. University of Arizona Press; Tucson: 1995. p. 149-165.
138. Barnes S. Evolution of the health benefits of soy isoflavones. Proc Soc Exp Biol Med. 1998; 217(3):386–392. [PubMed: 9492352]
139. Laland KN, Brown GR. Niche Construction, Human Behavior, and the Adaptive-Lag Hypothesis. Evolutionary Anthropology. 2006; 15:95–104.
140. Gerbault P, Liebert A, Itan Y, et al. Evolution of Lactase Persistence: An Example of Human Niche Construction. Philosophical Transactions of the Royal Society B: Biological Sciences. 2011; 366(1566):863–877.
141. Guarner F, Malagelada JR. Gut flora in health and disease. Lancet. Feb 8; 2003 361(9356):512–519. [PubMed: 12583961]
142. Edwards CA, Parrett AM. Intestinal flora during the first months of life: new perspectives. Br J Nutr. Sep; 2002 88(Suppl 1):S11–18. [PubMed: 12215177]
143. Palmer C, Bik EM, DiGiulio DB, Relman DA, Brown PO. Development of the human infant intestinal microbiota. PLoS Biol. Jul.2007 5(7):e177. [PubMed: 17594176]
144. Ley RE, Peterson DA, Gordon JI. Ecological and evolutionary forces shaping microbial diversity in the human intestine. Cell. Feb 24; 2006 124(4):837–848. [PubMed: 16497592]
145. Dethlefsen L, McFall-Ngai M, Relman DA. An ecological and evolutionary perspective on human-microbe mutualism and disease. Nature. Oct 18; 2007 449(7164):811–818. [PubMed: 17943117]
146. Ley RE, Turnbaugh PJ, Klein S, Gordon JI. Microbial ecology: human gut microbes associated with obesity. Nature. Dec 21; 2006 444(7122):1022–1023. [PubMed: 17183309]
147. Ley RE, Backhed F, Turnbaugh P, Lozupone CA, Knight RD, Gordon JI. Obesity alters gut microbial ecology. Proceedings of the National Academy of Sciences. Aug 2; 2005 102(31): 11070–11075.
148. Backhed F, Ding H, Wang T, et al. The gut microbiota as an environmental factor that regulates fat storage. Proceedings of the National Academy of Sciences. Nov 2; 2004 101(44):15718–15723.
149. Cani PD, Delzenne NM. Gut microflora as a target for energy and metabolic homeostasis. Curr Opin Clin Nutr Metab Care. Nov; 2007 10(6):729–734. [PubMed: 18089955]
150. Ridoutt BG, Pfister S. A Revised Approach to Water Footprinting to Make Transparent the Impacts of Consumption and Production on Global Freshwater Scarcity. Global Environmental Change. 2010; 20:113–120.
151. Frank-White NE, Burns-Kirkness R, Frank E. The Effect of Dietary & Transportation Choices on Climate Change. Food and Nutrition Sciences. 2011; 2:482–485.
152. Sapolsky, RM. Why Zebras Don’t Get Ulcers. Holt Paperbacks; 2004.
153. Sapolsky R. Sick of Poverty. Scientific American. 2005; 293:92–99. [PubMed: 16323696]
154. Tamashiro KLK, Hegeman MA, Sakai RR. Chronic Social Stress in a Changing Dietary Environment. Physiology & Behavior. 2006; 89:536–542. [PubMed: 16843504]
155. McDermott R. Ethics, Epidemiology and the Thrifty Gene: Biological Determinism as a Health Hazard. Social Science & Medicine. 1998; 47(9):1189–1195. [PubMed: 9783862]
156. Neyrinck AM, Delzenne NM. Potential Interest of Gut Microbial Changes Induced by Non-Digestible Carbohydrates of Wheat in the Management of Obesity and Related Disorders. Current Opinion in Clinical Nutrition & Metabolic Care. 2010; 13(6):722–728. [PubMed: 20823777]

157. Mitchell M. Increasing Fish Consumption for Better Health - Are We Being Advised to Eat More of an Inherently Unsustainable Protein? Nutrition Bulletin. 2011; 36(4):438–442.

158. Tennyson CA, Friedman G. Microecology, Obesity, and Probiotics. Current Opinion in Endocrinology, Diabetes & Obesity. 2008; 15(5):422–427.

159. Galland L. Diet and Inflammation. Nutrition in Clinical Practice. 2010; 25(6):634–640. [PubMed: 21139128]

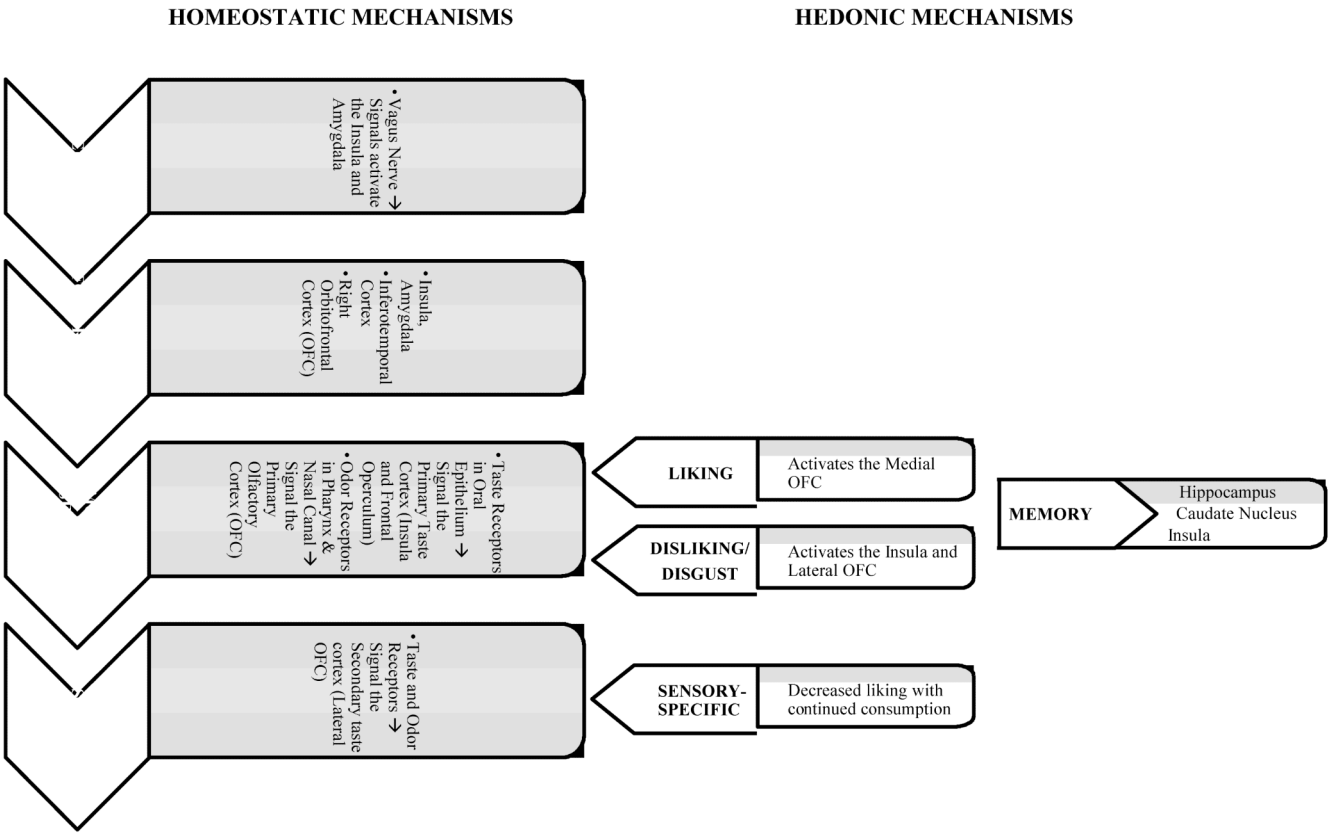

Figure 1.

Schematic representation of the areas of the brain that mediate aspects of feeding behavior in primates, including humans. Homeostatic mechanisms center on maintaining energy balance before, during, and following food consumption; Hedonic mechanisms center on the perceived liking or disliking of those consumed food resources and their effects on influencing future feeding behavior.