Understanding eating interventions through an evolutionary lens

Britt Ahlstrom, Tran Dinh, Martie G. Haselton & A. Janet Tomiyama

Accepted author version posted online: 14 Nov 2016.

Submit your article to this journal ⬀

View related articles ⬀

View Crossmark data ⬀

Understanding eating interventions through an evolutionary lens

For submission to *Health Psychology Review*

Britt Ahlstrom[a], Tran Dinh[b], Martie G. Haselton[acd], & A. Janet Tomiyama[a]

[a]*Department of Psychology, University of California, Los Angeles, Los Angeles, USA*

[b]*Department of Psychology, University of New Mexico, Albuquerque, USA*

[c]*Department of Communication Studies and* [d]*Institute for Society and Genetics,*

University of California, Los Angeles, Los Angeles, USA

Authors:

Britt Ahlstrom
Department of Psychology
University of California, Los Angeles
1285 Franz Hall
502 Portola Plaza
Los Angeles, CA 90095-1563, USA
ahlstrom@ucla.edu
310.909.9288

Dr. Martie G. Haselton
Department of Psychology
University of California, Los Angeles
1285 Franz Hall
502 Portola Plaza
Los Angeles, CA 90095-1563, USA
haselton@ucla.edu
310.206.7445

Tran Dinh
Department of Psychology
University of New Mexico
MSC03-2020
Logan Hall
Albuquerque, NM 87131-0001, USA
trandinh@unm.edu
505.277.4121

Dr. A. Janet Tomiyama
Department of Psychology
University of California, Los Angeles
1285 Franz Hall
502 Portola Plaza
Los Angeles, CA 90095-1563, USA
tomiyama@psych.ucla.edu
310.206.6875

Corresponding author: tomiyama@psych.ucla.edu

Abstract

Health psychologists aim to improve eating behaviour to achieve health. Yet the effectiveness of healthy eating interventions is often minimal. This ineffectiveness may be in part because many healthy eating interventions are in a battle against evolved mechanisms (e.g., hedonic and related systems) that promote the consumption of energy-dense foods. Such foods, once rare, are now abundant in our obesogenic society, and consequently the evolved desire for energy-dense foods can now easily lead to the overconsumption of sugary, processed, and unhealthy foods. However, humans have other evolved mechanisms that also impact eating behaviour. In this article, therefore, we review eating interventions through an evolutionary lens, and describe evolved mechanisms that are relevant to eating behaviour. We discuss how using this lens could help health psychologists design more effective eating interventions and policies. We also discuss how some prior eating interventions and policies may have failed by inadvertently working against evolved mechanisms. By learning to work with human nature, eating interventions can more effectively promote healthier eating and healthier lives.

Corresponding author: tomiyama@psych.ucla.edu

Health psychologists strive to help people eat well and be healthier. However, the effectiveness of health psychology interventions aimed at eating behaviour is often disappointing. For instance, calorie-restrictive dieting—one of the most commonly used eating interventions—has largely failed to result in sustained weight loss or health improvements (Franz et al., 2007; Mann et al., 2007; Tomiyama, Ahlstrom, & Mann, 2013). Policy interventions that raise awareness about healthy eating have also had mixed support and limited benefits (Brambila-Macias et al., 2014). There are likely many reasons for this ineffectiveness, but it may be, in part, because healthy eating interventions are often battling evolved mechanisms that promote the consumption of sugary, energy-dense foods, especially when the body feels deprived (Cummings, 2006; Saper, Chou, & Elmquist, 2002).

According to evolutionary theory, organisms have evolved mechanisms, such as those that affect eating behaviour, which would have increased their reproductive success in the ancestral past. Evolved mechanisms[1] can be operationally defined as patterns of behaviour and cognitive processes that have developed as a consequence of evolution. These mechanisms were shaped via natural selection when organisms faced adaptive challenges to fitness that recurred over evolutionary history, such as the challenges of survival and reproduction. In order for an organism to survive and reproduce in the future, the amount of energy it consumes plus the amount of energy stored from previous consumption must equal or exceed the amount of energy it expends. Through most of human evolutionary history, people hunted and gathered their food (Foley, 1995). This nomadic forager lifestyle entailed relatively high energy expenditure in an

---

[1]We use the term *evolved mechanism* because we do not posit that each mechanism in our framework is a unique, food-specific *adaptation* (a characteristic that increased survival and reproduction in the ancestral past). Some mechanisms may, indeed, be food-specific adaptations, whereas others may be byproducts of non-food-specific adaptations or of other chance factors (Buss, Haselton, Shackelford, Bleske, & Wakefield, 1998; Futuyma, 2009). Rather than focusing on the debate about what is and is not an adaptation, we therefore use the broader term evolved mechanism, defined as a characteristic that developed as one of the following: a food-specific adaptation, a byproduct of a non-food-specific adaptation, or a result of chance factors such as phylogenetic legacy, drift, or mutation (Buss et al., 1998).

Corresponding author: tomiyama@psych.ucla.edu

environment where food availability was more uncertain, energy-dense foods were scarce, and calories were harder to acquire (Eaton, Konner, & Shostak, 1988; Pontzer et al., 2012). These challenges for survival and successful reproduction likely led to the evolution of adaptive preferences for energy-dense foods—foods that were high in fat, sugar, and calories—and for the capacity to store excess calories as body fat, which provides a buffer against fluctuations in food availability (Cahill, 1976; Navarrete, van Schaik, & Isler, 2011; Tiger, 1992). Humans with adaptations that helped increase energy intake and reduce energy expenditure would have been more likely to survive in ancestral environments and to leave more descendants with genes for these traits. Yet in modern, obesogenic environments, these same adaptations can predispose individuals to obesity when the need for physical activity is low and the availability of calorie-dense foods is high (Eaton, Konner, & Shostak, 1988; Pontzer et al., 2012).

However, human eating behaviour is certainly more complex than consuming energy-dense food when it is available. The evolved mechanisms that regulate eating behaviour are not fixed—rather, they are context-dependent and contingent on environmental inputs, individual differences, and life stages. Interventions that adopt an evolutionary perspective can more effectively lead to healthier eating by manipulating environmental cues or tailoring methods to meet the evolved predispositions of target populations. In this review, we therefore view eating interventions using an evolutionary lens. We describe several evolved mechanisms that were likely designed to increase reproductive success ancestrally, and then describe interventions that tap into those mechanisms to promote healthier eating, which we define as eating more vegetables and less sugary, processed food.[2] We organize these mechanisms (Table 1) based on

---

[2]We acknowledge there are many healthy foods and that a variety of foods are essential for human health. However, we use this definition of 'healthy' because the aim of this article is ultimately to help the public eat healthier, and there is no debate that, on average, individuals in modern, industrialized nations underconsume vegetables and overconsume sugary, processed foods (Smith, Guenther, Subar, Kirkpatric, & Dodd, 2010).

Corresponding author: tomiyama@psych.ucla.edu

the earliest age at which an intervention targeting that mechanism would likely be effective. We conclude by outlining ways in which health psychologists can use knowledge gained from evolutionary psychology to improve research on and interventions for healthy eating.

Homeostatic and hedonic systems

To regulate eating behaviour in ways that would have been advantageous over evolutionary time, humans, like other animals, have numerous mechanisms that ensure adequate intake (Saper et al., 2002). Two of these mechanisms are the homeostatic and hedonic food systems (Lutter & Nestler, 2009). The homeostatic system moderates energy balance by promoting food consumption when energy reserves are low and inhibiting food consumption when energy reserves are high (Lutter & Nestler, 2009). In humans, the homeostatic system involves the hormones ghrelin, leptin, and cholecystokinin (Geary, 2004; Lutter & Nestler, 2009). These hormones interact with each other and with other neuronal (Lutter & Nestler, 2009) and environmental (Rozin & Tuorila, 1993) factors to regulate eating. When the body is food-deprived due to dieting or other food restriction, ghrelin levels increase and may promote eating initiation, the rewarding value of energy-dense foods, and weight regain (Cummings, 2006; Perello et al., 2010). After food consumption, increases in cholecystokinin, and possibly leptin, promote satiation and meal cessation (Cummings & Overduin, 2007; Geary, 2004).

The human hedonic system, on the other hand, motivates humans to eat more sugary, energy-dense foods—foods that would have been scarce and advantageous to consume in human ancestral environments (Lieberman, 2003; Lutter & Nestler, 2009). The hedonic system is based on taste and desire, rather than on physiological need. When the hedonic system is activated, the desire for food can be so strong that it overrides the homeostatic system (Lutter & Nestler, 2009; Saper et al., 2002), leading to the consumption of calories beyond that which is needed to

Corresponding author: tomiyama@psych.ucla.edu

maintain homeostasis. Such overriding would not have been very problematic in the ancestral past, when it would have taken substantial effort to obtain food and the threat of nutrient and calorie shortages would have been recurrent and potentially lethal (Lieberman, 2003). Indeed, in the ancestral environment, the hedonic system would have aided in survival and reproduction by motivating humans to engage in more flexible, adaptive behaviours to achieve desired rewards, as opposed to the primarily reflexive behaviours that result from homeostatic drives (Faster & Duncan, 1998). By doing so, the hedonic system motivated humans to not only obtain sufficient calories to achieve homeostasis, but also to seek out new food sources that promoted caloric consumption and the build up of fat stores, thereby further preventing starvation during food shortages (Lieberman, 2006; Ulijaszek, 2002).

Yet the food environment of modern, industrialized nations differs dramatically from that of the past (Lieberman, 2006). In these industrialized nations, energy-dense foods are easily accessed (often in the form of sugary, processed food), leading to 'obesogenic' environments in which energy-dense foods can be consumed at a much greater rate than they are needed by the body (Lieberman, 2006). Indeed, with the ubiquity of sugary, processed food, the hedonic system can now override the homeostatic system on a daily basis. This ubiquity of unhealthy food is a major contributor to high rates of obesity and diabetes in the modern world (Lieberman, 2003).

Because overeating of hedonically rewarding foods contributes to a broad range of poor health outcomes, eating researchers using an evolutionary perspective often focus on the hedonic system. Yet human eating behaviour is more complex than homeostatic and hedonic systems, and therein lies the potential for healthy eating interventions. Humans have numerous evolved behaviours and cognitive processes that are sensitive to ecological, social, developmental, and cultural inputs, and that help them adapt to variability in their environment, including their eating

Corresponding author: tomiyama@psych.ucla.edu

environment (Kaplan et al., 2000; Turner & Thompson, 2013). Health psychologists can tap into these evolved mechanisms to alter eating behaviour. Even if these behaviours and cognitive processes did not evolve specifically *for* regulating eating (although many of them may have), they can, nevertheless, influence eating. Therefore, even though it would be exceedingly difficult for health psychologists to eliminate the hedonic drive for sugary, energy-dense foods, they can nevertheless design interventions that work with these other evolved mechanisms to affect eating behaviour (Turner & Thompson, 2013).

Evolved mechanisms that affect eating behaviour

In this review, we discuss evolved mechanisms that affect eating behaviour, summarized in Table 1, which is organized by the earliest age at which an intervention that targets each mechanism would likely be effective. [Table 1 near here].

Familiarity bias

Familiarity bias—or growing to like foods to which an individual has been exposed many times before—is one cognitive process that would have aided in survival in ancestral environments. Foods eaten often in the past are less likely than novel foods to be poisonous or otherwise harmful. This preference for familiar foods has been observed following exposure in adulthood (Pliner, 1982), childhood (Wardle, Herrera, Cooke, & Gibson, 2003), infancy (Maier, Chabanet, Schaal, Issanchou, & Leathwood, 2007), and even in utero (Cooke & Fildes, 2011). Novel foods can be especially harmful to young children, who are capable of putting substances into their mouths but who have smaller bodies and less practice in discerning which substances are safe to eat (Cashdan, 1998). Consequently, increased liking for foods with repeated exposure would have helped infants and children learn which foods and flavours are safe, and continues to do so in the modern day (Cashdan, 1994; Cooke, 2007). However, children between ages two

Corresponding author: tomiyama@psych.ucla.edu

and six can be quite fearful of new foods—known as *neophobia* (Dovey, Staples, Gibson, & Halford, 2008). This fear of new foods is hypothesized to be an evolved aversion that prevents children from consuming harmful food (Dovey et al., 2008), but it also tends to lead to the avoidance of healthy foods, such as vegetables (Cooke, Wardle, & Gibson, 2003).[3]

In modern environments, familiarity bias can increase preferences for unhealthy food through frequent exposure to readily accessible sugary, calorie-dense, processed foods. This process is facilitated by humans' innate preference for sweet flavours over bitter ones (Birch, 1999). However, interventions that use repeated exposure to induce familiarity bias can be effective in increasing preferences for healthy foods such as vegetables (Cooke et al., 2004; Forestell & Mennella, 2007; Mennella, Jagnow, & Beauchamp, 2001). Yet because it can be difficult to get neophobic children to eat these foods to begin with, targeting children *before* the neophobic period could be more effective. Indeed, the first two to three years of life have been called a 'sensitive period,' during which children learn which foods are safe to eat (Cashdan, 1994).

Yet interventions could be effective much earlier, such as while the child is still in utero. Experiments have shown that infants are exposed to and develop a liking for the flavours of their mothers' diet through amniotic fluid and breast milk. When mothers were randomly assigned to drink carrot juice during pregnancy or lactation, their infants developed a greater liking (as indicated by fewer negative facial features) for carrot-flavoured cereal (Mennella et al., 2001). Experimental evidence also suggests that when infants were fed previously-disliked vegetable purees for several days, they quickly grew to like the vegetables and subsequently ate more of

---

[3]The bitterness of some vegetables is another likely factor in children's vegetable avoidance, as bitterness can signal toxins that could be dangerous to small bodies (Cashdan, 1998). However, distaste for the bitter flavour does not account for the avoidance of all vegetables, such as sweet vegetables, and although bitterness may underlie children's initial avoidance, neophobia likely perpetuates it (Cashdan, 1998).

Corresponding author: tomiyama@psych.ucla.edu

them (Maier et al., 2007; Mennella, Nicklaus, Jagolino, & Yourshaw, 2008). Health psychologists could therefore aim eating interventions at pregnant and new mothers, so as to elicit infants' familiarity bias for vegetables in utero (through amniotic fluid) and shortly after birth (through breast milk; Mennella et al., 2001). Educating these mothers about the importance of their diet while pregnant or breastfeeding could be an effective means of improving mothers' vegetable consumption, and in turn, the vegetable consumption of their children. Expectant and new mothers could also be provided with free or subsidized healthy foods. For example, hospitals could send a mother home with her newborn and a bag of broccoli, with a reminder that by eating the broccoli and then breastfeeding, she could facilitate her infant's liking for broccoli. If the mother followed through on eating the broccoli, both the infant and the mother herself could benefit from familiarity bias.

Sensory-specific satiety

Diverse diets promote well-rounded nutrient intakes, thereby promoting human health and survival (Hockett & Haws, 2003). One tendency that has helped humans seek out diversity is sensory-specific satiety—the tendency for people to perceive foods that they have recently eaten as less hedonically rewarding (i.e., less desirable), while foods that they have not recently eaten remain just as hedonically rewarding (Raynor & Epstein, 2001; Rolls, 1986; Rolls et al., 1981). This tendency has been documented in animals and humans, and in both infants and adults (Birch & Deysher, 1986; Rolls, 1986; Wansink, 2004). Sensory-specific satiety could have effectively motivated individuals to seek out nutrients they had not recently consumed and to avoid investing energy towards acquiring nutrients they had already obtained. This mechanism could have encouraged eating a variety of different food, thereby avoiding nutritional deficiencies (Armelagos, 2014; Rolls et al., 1981).

Corresponding author: tomiyama@psych.ucla.edu

Because sensory-specific satiety tempers the hedonic reward system, rather than working on the homeostatic system, sensory-specific satiety is primarily influenced by sensory (hedonic) qualities of food and not by the physiological consequences of absorbing and digesting food (Bell, Roe, & Rolls, 2003; Raynor & Epstein, 2001). Consequently, the mere sensory diversity of energy-dense (e.g., sugary, processed) food can lead the hedonic system to override the homeostatic system, resulting in overeating (McCrory, Burke, & Roberts, 2012). Throughout evolutionary history, the variety of diverse and hedonically rewarding foods would have been limited. However, in modern, industrialized nations, the variety of hedonically rewarding, sugary, and processed food is almost infinite (Eaton, Konner, & Cordain, 2010), not only due to the increased variety of food items, but also to increased *perceptions* of variety from the diversity of food packing, flavours, and dyes. When individuals become sensory satiated to one unhealthy food item, there is always another unhealthy item (or another brand or another flavour) to try (Armelagos, 2014). Consequently, the sensory diversity of sugary, processed food can hijack this system, preventing sensory-specific satiety from decreasing food intake (Armelagos, 2014). Nevertheless, health psychologists can design interventions to facilitate sensory-specific satiety for unhealthy foods, while promoting perceptions of variety and increased consumption of healthy foods. Interventions that target sensory specific satiety have been successfully implemented in preschool-aged children, both in school (Roe, Meengs, Birch, & Rolls, 2013) and in their homes (De Wild, de Graaf, Boshuizen, & Jager, 2015). Yet sensory-specific satiety is also active in infants (Rolls, 1986), and such interventions could therefore be applied as early as infancy. This is the time period when most children switch from breast milk to pureed food and start to have a choice in what they will and will not eat (Fox, Pac, Devaney, & Jankowski, 2004).

Corresponding author: tomiyama@psych.ucla.edu

However, it is still an infant's care provider who presents the food options to the infant, and therefore interventions that target sensory-specific satiety in infants would need to work through the infant's parents or other care providers. Health psychologists could work with this evolved tendency to promote healthier eating by sending parents and infant care providers vouchers for or samples of various pureed vegetables. Health psychologists could also provide parents with baby food processors and trial Community Supported Agriculture (CSA) memberships, so that parents can cook and puree their infant a variety of seasonal vegetables. By providing their infants with a variety of vegetable flavours, the infants would be less likely to become sensory sated on vegetables and consequently consume more of them.

Increasing *perceived*, rather than actual variety could also be effective. For instance, both children and adults select and eat less food when they are presented with fewer food colours (e.g., being presented with 7, instead of 10, colours of M&Ms; Kahn & Wansink, 2004), and interventions using this tactic have shown some success (Epstein, Carr, Cavanaugh, Paluch, & Boulton, 2011; Raynor, 2012). Consequently, health psychologists could teach parents how to add safe and natural beet, carrot, and berry dyes to otherwise bland-coloured canned baby foods. By instructing parents to add these safe dyes to vegetable dishes, while not adding the dyes to any sugary, processed dishes, the parents would be providing more varied colours of healthy foods and relatively more monotonous colours of unhealthy ones. This tactic would work with the evolved tendency of sensory-specific satiety in infants to promote healthier eating. Although further research is required, such efforts could help prevent the under-consumption of vegetables in infants (Fox, Pac, Devaney, & Jankowski, 2004) and set the stage for healthier eating throughout their lives.

Disgust

Corresponding author: tomiyama@psych.ucla.edu

Nausea and vomiting can result from consuming toxic or otherwise unsafe food; the development of conditioned food aversions following nausea or vomiting would have been evolutionarily advantageous (Bernstein, 1978; Garcia, Kimeldorf, & Koelling, 1955; Horn, 2008; Pelchat & Rozin, 1982). These aversions protect animals, including humans, from eating that food again (Garcia et al., 1955; Horn, 2008). A possible strategy for encouraging people to eat less unhealthy food is to make them sick of the food, by making them sick *from* the food. Conditioned food aversions can develop even when the food was not truly to blame for the symptoms. For instance, experiments have shown that when individuals ate ice cream prior to chemotherapy, they later expressed a decreased preference for that same type of ice cream (Bernstein, 1978; Bernstein & Webster, 1980), and that when individuals drank a beverage prior to induced motion sickness, they later drank less of that same type of beverage (Arwas, Rolnick, & Lubow, 1989). Such food aversions may contribute to lowered consumption among picky eaters and individuals receiving chemotherapy or radiation treatment for cancer (Bernstein, 1999; Scalera, 2002). However, food aversions do not appear to substantially alter overall amount of food consumed for healthy individuals who eat a varied diet, as these individuals can eat another food in place of the food to which they have developed an aversion (Scalera, 2002).

Nevertheless, conditioned taste aversion interventions could, theoretically, be tried for individuals who eat excessive amounts of particular foods. Interventionists could refer to prior work on smoking and alcohol aversion therapy as models. For example, smoking interventions that involve smoking quickly (which induces feelings of illness) have increased abstinence better than control interventions (Tahiri, Mottillo, Joseph, Pilote, & Eisenberg, 2012). Furthermore, in one alcohol aversion study, pairing alcohol with a vomit-inducer (ipecac) resulted in decreased alcohol intake, while pairing alcohol with electric shock did not (Cannon, Baker, & Wehl, 1981).

Corresponding author: tomiyama@psych.ucla.edu

However, such conditioned aversion studies have fallen out of favour because of the fear that such treatments could be dangerous to participants (Scalera, 2002; Tahiri et al., 2012). It is unlikely that many health psychologists would conduct a study that involves inducing actual illness, but it is possible that merely pairing often-overeaten foods with revolting pictures or smells could result in food avoidance by eliciting disgust (Rozin & Fallon, 1987). For example, watching *Super Size Me,* a documentary that pairs fast food with disgusting visuals (e.g., a person vomiting after eating fast food), has been has been shown to result in short term reductions in fast food intake in adults (Cottone & Byrd-Bredbenner, 2007). In another study, placing a candy, which the participant previously reported liking, on the bottom of a clean toilet led children as young as 2.5 years of age to throw away the candy that had touched the toilet, rather than to eat it (Stevenson, Oaten, Case, Repacholi, & Wagland, 2010). The minimum age for these disgust-based manipulations appears to be the age when children have the capability to be 'sufficiently attentive and independent during testing' (Stevenson et al., 2010, p. 168), which has been found to be around age 2 (Stevenson et al., 2010). Health psychologists could therefore investigate if pairing disgusting images and smells with unhealthy food could reduce unhealthy food consumption, starting with children as young as 2 years of age (Stevenson et al., 2010). There will be ethical and practical challenges to conducting disgust-based interventions in such a young population, but it is an intriguing avenue for future research.

Life history strategies

Life history theory is a framework in evolutionary biology that attempts to explain how individuals strategically allocate their limited energy supply in ways that would have increased their reproductive potential in the ancestral past (Brumbach, Figueredo, & Ellis, 2009; Chisholm et al., 1993; Del Giudice, Gangestad, & Kaplan, 2015; Ellis, Figueredo, Brumbach, & Schlomer,

Corresponding author: tomiyama@psych.ucla.edu

2009). Children are exposed not only to food in their homes, but also to larger patterns of resource availability and predictability in their environments (Belsky, Steinberg, & Draper, 1991). According to life history theory, individuals use these patterns to make trade-offs in how to allocate resources throughout development (Belsky et al., 1991; Chisholm et al., 1993). These trade-offs form coordinated patterns of life history 'strategies', which fall along a continuum from 'slow' to 'fast'. When ancestral environments were plentiful and predictable, a 'slow' strategy would have been advantageous, in which individuals save up resources, invest more in their bodily health, and delay reproduction, which ultimately allows them to invest more in their future potential or current offspring and to increase their offspring's competitive advantage (Brumbach, Figueredo, & Ellis, 2009; Chisholm et al., 1993). Yet such a strategy would have been risky in marginal, unpredictable ancestral environments, in which individuals and their children had a high risk of death by injury, illness, or starvation, regardless of the amount of parental investment they received (Griskevicius, Tybur, Delton, & Robertson, 2011). Therefore, in these environments, a 'fast' strategy would have been advantageous. This strategy involves immediate reward-seeking, impulsive behaviour, reduced investment in long-term health, and earlier reproduction to increase the chance of reproducing (Belsky et al., 1991; Brumbach et al., 2009). Longitudinal research has suggested that life history strategies may be especially dependent upon environmental conditions during the first five years of life (Simpson, Griskevicius, Kuo, Sung, & Collins, 2012).

Life history strategies have been shown to lead to different resource consumption patterns, especially during times of environmental stress (Griskevicius, Delton, et al., 2011; Griskevicius, Tybur, et al., 2011; Griskevicius et al., 2013). Because food is a resource, life history strategies have also been hypothesized to lead to different food consumption patterns, such that slow

Corresponding author: tomiyama@psych.ucla.edu

strategists might respond to environmental stressors by eating less and fast strategists might respond by eating more (Hill, Rodeheffer, DelPriore, & Butterfield, 2013). Supporting this hypothesis, across a series of experiments, young adult females from poorer, more unpredictable childhood environments responded to primes of environmental stress by choosing more food items and displaying an increased desire for unhealthy foods; females from more plentiful, predictable environments displayed the opposite pattern (Hill et al., 2013). Other research suggests that life history strategies may influence eating behaviour even when individuals are not stressed. One study found that among individuals who had grown up in higher socioeconomic status (SES) environments (an indicator of slower life history strategies), those who were energy-sated ate less than those who were energy-deprived; in contrast, individuals who had grown up in lower SES environments (a predictor of fast strategies) ate comparably high amounts of food, both when energy-sated and when energy-deprived (Hill, Prokosche, DelPriore, Griskevicius, & Kramer, 2016). These findings could potentially explain why poverty (Duncan, Ziol-Guest, & Kalil, 2010) and adversity during childhood (Thomas, Hyppönen, & Power, 2008) are longitudinally associated with greater adult body weight. Fast life history strategies are adaptive in harsh and unpredictable ancestral environments, but in modern, obesogenic environments, they can also lead to overeating and weight gain (Hill et al., 2013; Hill et al., 2016).

If life history strategies do influence eating behaviour in this way, it would open to the door to interventions that work with these strategies to promote healthier eating. Life history-based interventions could be applied as soon as life history strategies are formed, around age 5 (Simpson et al., 2012). Targeting life history strategies could be particularly useful in interventions that incentivise children (ages 5 and older) in kindergartens and nurseries to eat healthier. Similar school-based interventions have already shown promising results (Jones, 2014;

Corresponding author: tomiyama@psych.ucla.edu

Just & Price, 2012). In one field experiment on 15 elementary school lunchrooms, children who were provided with small monetary (e.g., a quarter) or prize (e.g., raffle ticket) incentives to eat at least one serving of vegetables or fruit were 80% more likely to do so (Just & Price, 2012). Incentives were more effective among lower-SES schools and when the incentives were paid immediately, rather than 1-2 weeks later (Just & Price, 2012). Health psychologists using an incentive system to encourage vegetable intake could conduct interventions personalized to individuals' life history strategies and investigate whether delayed, large incentives are more effective in higher SES schools/nurseries (e.g., children receive $1.25 at the end of the week if they ate vegetables every day) and whether immediate, small rewards are more effective in lower SES schools/nurseries (e.g., $0.25 for each serving of vegetables eaten).

However, there remain many questions about how life history strategies influence eating behaviour, such as whether life history strategies influence both amount and type of food consumed. Further research is also necessary to determine whether immediate versus delayed incentives are best implemented on an individual or school-wide basis, and whether short-term monetary or prize interventions can result in long-term changes.

Genetic predispositions

As omnivorous eaters inhabiting a diverse array of terrain with various food sources, humans have faced the adaptive problem of choosing and consuming the types and amounts of food that would have aided in their survival and reproductive success. Humans can taste five basic flavours—sweet, savoury/umami, bitter, sour, and salty—which signal the nutritional value and potential danger of food (Scott, 2005). Sweetness and umami can indicate sugars and amino acids, respectively, whereas bitterness and sourness can indicate toxic substances (e.g., toxic plants) and low pH (e.g., unripe fruit; Scott, 2005; Yarmolinsky, Zuker, & Ryba, 2009). These

Corresponding author: tomiyama@psych.ucla.edu

flavour signals explain why infants have evolved an innate disliking for bitter and sour tastes and an innate liking for sweet and possibly savoury flavours (Birch, 1999; Yarmolinsky et al., 2009). However, humans also display individual differences in these preferences. For instance, gene variants influence the preference for and consumption of sweet (Mennella, Pepino, & Reed, 2005), bitter (Keller, Steinmann, Nurse, & Tepper, 2002), and fatty foods (Keller et al., 2002) in children. Gene variants also play a role in the preference for and consumption of bitter and sweet vegetables (Dinehart, Hayes, Bartoshuk, Lanier, & Duffy, 2006). These gene variants are not necessarily adaptive, as non-adaptive genetic variation occurs frequently (Buss, 2009).

Nevertheless, health psychologists could use their knowledge of these individual differences in gene-based preferences to design more effective interventions. Diets based on genetic or genomic variation in response to food (i.e., *nutrigenetics* or *nutrigenomics*) already exist, but such diets tend to be tailored to individuals' unique genetic nutritional requirements (Arkadianos et al., 2007; Fenech, Leah, & Ferguson, 2011; Frankwich et al., 2015), rather than tailored to individuals' unique food preferences. For example, in one nutrigenetic diet study, participants with GSTM1 gene deletions were informed that the risk of lung cancer among people with these deletions was reduced by 80% when cruciferous vegetables were frequently consumed (Arkadianos et al., 2007). In support of such diets, individuals who were assigned to the nutrigenetic diet lost more weight than matched controls who received a non-nutrigenetic diet (Arkadianos et al., 2007). In another study, when individuals were randomly assigned to receive advice on a nutrigenomic or a standard diet, individuals in the nutrigenomic group were more likely to report they understood the advice, that the advice would be useful, and that they wanted to learn more (Nielsen & El-Sohemy, 2012). However, in a more recent, albeit small (N = 51) randomized controlled trial, a nutrigenetic diet did not result in more weight loss than a standard

Corresponding author: tomiyama@psych.ucla.edu

diet, and people had difficulty adhering to the nutrigenetic diet, just as people tend to have difficulty adhering to other diets (Frankwich et al., 2015). Furthermore, the *taste* of food is usually more important than the *health* of food in determining food choice (Glanz, Basil, Maibach, Goldberg, & Snyder, 1998). In fact, individuals perceive that the less healthy a food is, the better it will taste and the more they will enjoy it (Raghunathan, Naylor, & Hoyer, 2006).

Consequently, interventions could focus on tailoring diets to individuals' unique genetic food *preferences* (e.g., instructing them as to which vegetables they may like, based on their genes, that they have never had before). Individuals who have a genetic predisposition to like the sweet flavour could be taught about the variety of sweet vegetables that could be added to their diets, such as purple sweet potatoes, cooked beets, and sugar snap peas. Conversely, individuals who have a genetic predisposition to like savoury flavours could be taught about the variety of savoury vegetables that could be added to their diets, such as shitake mushrooms and cooked carrots, and be recommended to eat vegetables with savoury tomato-based sauce. Individuals with a genetic aversion to bitter foods could also be taught healthy ways to reduce the bitterness of vegetables, such as by cooking them or topping them with pineapple or lime juice. Health psychologists could investigate if these genetic predisposition-based strategies lead to greater intake of healthy foods, as well as if implementing these strategies into existing diets results in greater adherence. Because these interventions have not been sufficiently, if at all, investigated in adolescents, and because of the ethical and legal challenges with providing children and adolescents with genetic testing (Wertz, Fanos, & Reilly, 1994), these interventions would likely be most feasible and effective among individuals age 18 and older.

Perceptions of the Controllability of Mortality

Corresponding author: tomiyama@psych.ucla.edu

At any given time, individuals have a non-zero chance of mortality and finite energetic resources to invest in reproductive activities during a finite lifespan. Evolved heuristics for estimating the controllability of mortality would therefore have helped humans optimize effort and resources towards goals that were fitness-enhancing in ancestral environments. These evolved heuristics would have helped humans make trade-offs in health behaviours, including eating behaviour (Pepper & Nettle, 2014b), and these processes are the focus of the behavioural-ecological model of optimal health behaviour (Nettle, 2010). This model shares similarities to life history theory in that they both involve trade-offs. Yet unlike life history theory, the behavioural-ecological model focuses on current perceptions of mortality risk rather than on strategies that one developed during childhood (Nettle, 2010). This model posits that investing in healthy behaviours might be beneficial for individuals whose mortality risk is intrinsic (i.e., individuals who believe their lifespan is meaningfully affected by health behaviours), but such prioritizing would be costly for individuals whose mortality risk is extrinsic (i.e., individuals who believe their lifespan is unaffected by health behaviours and is instead determined by genetics and/or environmental circumstance). Individuals who perceive mortality as intrinsic would expect that health behaviours would increase their longevity, whereas individuals who perceive mortality as extrinsic, and therefore uncontrollable, would not expect health behaviours to be life extending. When mortality is extrinsic and uncontrollable, investing in health behaviours could even be costly because doing so takes up time and resources that could otherwise be used to pursue reproductively beneficial activities such as gaining status, resources, or mating opportunities (Pepper & Nettle, 2014b).

This behavioural-ecological framework shares similarities to health locus of control theory but differs in that the latter theory views locus of control as a stable individual trait, rather

Corresponding author: tomiyama@psych.ucla.edu

than a malleable belief. For instance, health locus of control interventions aim at matching health messages to an individual's locus of control beliefs (Williams-Piehota, Schneider, Pizarro, Mowad, & Salovey, 2004), rather than altering those beliefs. The behavioural-ecological framework also shares similarities to terror management theory, which has been used successfully in some health behaviour interventions (Goldenberg & Arndt, 2008), but differs from terror management theory in their prediction of what leads to healthy behaviour. Terror management theory predicts that thoughts of mortality lead to health-improving behaviours (Goldenberg & Arndt, 2008), whereas the behavioural-ecological model predicts—and indeed has shown in experiments—that primes that mortality is intrinsic lead people to engage in health improving behaviours, whereas primes that mortality is extrinsic lead people to engage in unhealthy behaviours. For instance, in a series of experiments (Pepper & Nettle, 2014a), adults were primed with the perception that life expectancy was either controllable or uncontrollable and were asked whether they would prefer to receive a box of fruit or a box of chocolates as a reward for completing the study. Adults who had been primed to believe mortality was controllable were more likely to prefer fruit, whereas adults who had been primed to believe mortality was uncontrollable were more likely to prefer chocolate. Consequently, the evolved tendency to adapt one's health behaviours to the perceived controllability of mortality may partially explain healthy food choices. It may also explain why individuals who believe that diabetes can be controlled by personal behaviours exhibit greater dietary adherence (Scollan-Koliopoulos, Walker, & Rapp, 2011). Further research is necessary to determine if perceived controllability of mortality influences actual food consumption and if it influences food consumption in children, who have not yet been experimentally studied using this model.

Corresponding author: tomiyama@psych.ucla.edu

Nevertheless, this behavioural-ecological model has been shown to influence adults' food choices (Pepper & Nettle, 2014a), and the evolved tendency to adapt health behaviours to the perceived controllability of mortality can therefore be targeted in adults age 18 and older. Health psychologists could use this behavioural-ecological model to design interventions that target adults at the moment they are about to eat or buy food. Throughout grocery stores, individuals could be primed with the thought that mortality is controllable. Public cafeterias and restaurants could also be decorated with reminders that healthy eating can contribute to longer lives. Refrigerator magnets with such reminders could target at-home food choices.

Interventions such as these could be especially beneficial for adults of lower subjective SES, as these individuals are more likely to perceive mortality as extrinsic and to invest less effort in health behaviours (Pepper and Nettle, 2013). However, merely telling adults of lower SES that their mortality is controllable may not be enough, as there is substantial research showing that living in low SES neighbourhoods *does* lead to greater risk of dying by violent or accidental causes (Adler & Ostrove, 1999).

Another tactic consistent with the behavioural-ecological model would be to tackle poverty, rather than eating behaviour directly; indeed, higher SES groups consistently eat more vegetables and 'healthy' foods, compared to lower SES groups (Darmon & Drewnowski, 2008). Of course, reducing poverty will prove difficult, but its impact on mortality perceptions may be a factor underlying the success – and failure – of interventions that rely upon people making food choices that are good for their health (Nettle, 2010). Convincing people to make healthy choices is the basis of many eating interventions (Cutler, 2004), and this problem underscores the importance of integrating an evolutionary perspective into health psychologists' eating interventions.

Corresponding author: tomiyama@psych.ucla.edu

Discussion

In this review, we used an evolutionary lens to help understand how eating interventions could work with evolved mechanisms. Due to humans' evolutionary lineage, it would be exceedingly difficult for health psychologists to rid people of their evolved preference for sugary, energy-dense food. However, health psychologists can tap into many other evolved behaviours and cognitive processes to promote healthier eating. We hope that our review can help to guide health psychologists in integrating these mechanisms and designing more effective interventions.

We organized our review by the earliest age at which an intervention that targets each mechanism would likely be effective, to provide health psychologists with better direction in selecting which evolved mechanisms to target. By providing these ages, we aim to provide guidance regarding which evolved behaviour or cognitive process would likely be effective targets, based on empirical evidence, for promoting healthy eating in a particular age group. This organization does not imply that the evolved mechanisms would be completely ineffective targets for other ages. In many instances, the evolved mechanisms that would likely be effective targets in infancy could also be effectively targeted in adulthood. For example, inducing sensory specific satiety could reduce unhealthy food consumption in adults, as well as in infants. Additionally, repeatedly exposing adults to healthy foods – thereby inducing familiarity bias – could increase their preferences for these healthy foods, as it does in utero and in infancy. In other instances, evolved mechanisms could potentially be targeted at earlier ages. For example, genetic predispositions and controllability of mortality may be effective targets in adolescents, but due to a lack of experimental research on younger ages this cannot yet be recommended. In summary, the evolved mechanisms reviewed here are organized based on the *earliest* age for which there is empirical evidence suggesting that targeting each evolved mechanism would

Corresponding author: tomiyama@psych.ucla.edu

likely be effective, but future research will provide further insight into the optimal ages for intervention.

Additionally, the evolved mechanisms we reviewed need not be targeted in isolation. Rather, by targeting multiple mechanisms simultaneously, health psychologists could potentially further increase the effectiveness of their interventions. For example, an intervention that provides schoolchildren with the vegetables that are familiar to them (which works with humans' evolved familiarity bias) could be further strengthened by providing financial incentives to eat these vegetables in low-SES schools (which works with life history strategies).

Considering multiple evolved mechanisms simultaneously could also prevent the design of interventions that work with one mechanism but orthogonal to another. For instance, delivering fresh vegetables to the homes of new mothers could lead their breastfeeding babies to develop greater liking for vegetables through the familiarity bias, but if the mothers view mortality as extrinsic and uncontrollable, they may not see as much benefit to vegetables and may be less likely to eat them. If, instead of working orthogonally to the behavioural-ecological model, the intervention took both evolved mechanisms (familiarity bias and perceived controllability of mortality) into account, researchers might send the vegetables in packaging that reminds mothers that both their lifespan and the lifespan of their children are influenced by what the mother eats (Bateson et al., 2004; Heidemann et al., 2008). If this addition led mothers to see mortality as more intrinsic, it might allow familiarity bias to have more impact.

Targeting multiple evolved mechanisms could also prevent the design of interventions that work with one mechanism but against another. For example, providing a wider variety of vegetables to children in preschool may work with sensory-specific satiety to promote vegetable consumption, but if those vegetables are entirely novel to the children, the familiarity bias may

Corresponding author: tomiyama@psych.ucla.edu

prevent children from actually eating the various options. To increase the intervention's effectiveness, researchers may want to provide varied, yet familiar vegetables, with 'familiar' vegetables being determined by asking parents what vegetables they eat at home, or even what vegetables the mother ate during pregnancy. Such interventions would need to be empirically tested to determine their effectiveness, but these examples elucidate the importance of considering multiple evolved mechanisms within a single intervention.

We anticipate that integrating evolutionary perspectives into research design will result in more effective healthy eating interventions. However, our review is not exhaustive, nor is it intended to be. Future work should expand the scope of evolutionary mechanisms and additionally consider how the evolved mechanisms interact with one another (e.g., how do life history strategies interact with the familiarity bias?) and with novel societal phenomena (e.g., fast food and the globalization of the food supply; Jabr, 2013). Future research should also test where, when, and for whom each of the evolved mechanisms most apply, so that interventions can be tailored to specific population contexts and be maximally effective. One major challenge going forth is that any intervention will be battling against humans' evolved preference for foods high in sugar, fat, and calories. Given this strong hedonic drive, it is all the more important to ensure interventions are not inadvertently doubly stacking the deck against their own success by failing to take other evolutionary mechanisms into account. Here we have focused on promoting healthy eating, but our evolved hedonic drive could promote unhealthy eating. However, increasing healthy eating alone can positively impact health. For example, in a meta-analysis of 221,080 participants, the relative risk of coronary heart disease was reduced by 4% for every one additional serving of fruits and vegetables consumed per day (Dauchet, Amouyel, Hercberg, & Dallongville, 2006). Another meta-analysis demonstrated that risk of all-cause mortality was

Corresponding author: tomiyama@psych.ucla.edu

decreased by 6% and 5% for each additional daily serving of fruits and vegetables, respectively (Wang et al., 2014).

Many of the theories discussed in this review have not yet been implemented in healthy eating interventions, and the interventions that have been conducted have often been brief in duration. Consequently, further research will be required to determine the sustainability of the healthy eating interventions that we propose. For instance, if low SES students are rewarded for eating vegetables, will they need to receive payment or prize with each meal they eat? Or would rewarding low SES students for eating vegetables lead those children to develop a liking for the vegetables from familiarity bias, leading to a spillover effect in which those children also eat more vegetables at dinner? Similarly, would low SES children need to be incentivized for eating vegetables throughout their schooling, or would paying children in a low SES elementary school to eat more vegetables lead them to develop a liking for vegetables and continue to eat more vegetables in their later years, even if they were not paid to eat them? If payment needed to be continuous to be effective, this would not necessarily make the interventions useless; for instance, governmental programs often subsidize fruit and vegetable purchases (e.g., Patlan & Mendelson, 2016). Furthermore, paying low SES students to eat just one more serving of vegetables a day could save the government money long-term. One study found that if Americans ate just one more serving of vegetables or fruits each day, it would save over five billion USD in medical costs every year (Union of Concerned Scientists, 2013). However, would continuous payment lead to continuous vegetable-eating, or would the incentives lose their effectiveness over time? These are questions that need to be explored in studies evaluating interventions' effectiveness in changing behaviour, and mechanisms underlying that behaviour change (see Aunger & Curtis, 2016).

Corresponding author: tomiyama@psych.ucla.edu

Because our review is targeted toward evolved mechanisms that affect healthy eating

behaviour, we do not address the numerous evolutionary theories focused on evolved behaviours,

predispositions, and growth patterns that affect *weight.* We refer readers who are interested in

this topic to the extensive literature on the evolution of thrifty genes (Neel, 1999), obesity genes

(e.g., FTO gene; Hofker & Wijmenga, 2009; Olszewski & Fredriksson, 2009), and the potential

adaptiveness of obesity (Speakman, 2013). We also do not address the substantial literature on

how the Western diet can conflict with humans' evolved genome to result in chronic disease. We

encourage readers who are interested in this topic to read the reviews that discuss this in depth

(Cordain et al., 2005; Lucock, Martin, Yates, & Veysey, 2014; Simopoulos, 1999). Most likely,

the evolution of obesity (Speakman, 2013), the evolution of the Western diet (Cordain et al.,

2005; Simopoulos, 1999), and the evolution of mechanisms that affect eating behaviour (this

review) all interact, and we expect that determining how these perspectives can be integrated will

be an important focus of evolutionary health psychology in the years to come.

Conclusion

Individuals interested in encouraging healthy eating are likely to benefit from including

evolutionary perspectives in their intervention toolkit (Lozano, 2010; Tybur, Bryan, & Hooper,

2012). Such integration could greatly increase the effectiveness of healthy eating interventions,

of which modern society is in desperate need. We reviewed eating interventions with an

evolutionary lens in order to ultimately help develop more effective eating interventions and

policies, with the goal of helping people achieve health.

Corresponding author: tomiyama@psych.ucla.edu

References

Adler, N. E., & Ostrove, J. M. (1999). Socioeconomic status and health: What we know and
what we don't. *Annals of the New York Academy of Sciences*, *896*, 3–15.
doi:10.1111/j.1749-6632.1999.tb08101.x

Arkadianos, I., Valdes, A. M., Marinos, E., Florou, A., Gill, R. D., & Grimaldi, K. A. (2007).
Improved weight management using genetic information to personalize a calorie
controlled diet. *Nutrition Journal, 8*, 1–8. doi:10.1186/1475-2891-6-29

Armelagos, G. J. (2014). Brain evolution, the determinants of food choice, and the omnivore's
dilemma. *Critical Reviews in Food Science and Nutrition*, *54*(10), 1330–1341.
http://doi.org/10.1080/10408398.2011.635817

Arwas, S., Rolnick, A., & Lubow, R. (1989). Conditioned taste aversion in humans using
motion-induced sickness as the US. *Behaviour Research and Therapy*, *27*(3), 295–301.
Retrieved from http://www.sciencedirect.com/science/article/pii/0005796789900491

Aunger, R., & Curtis, V. (2016). Behaviour centred design: Towards an applied science of
behaviour change. *Health Psychology Review, 10,* 425-446. doi:
10.1080/17437199.2016.1219673

Bateson, P., Barker, D., Clutton-Brock, T., Deb, D., D'Udine, B., Foley, R. A., ... & Sultan, S. E.
(2004). Developmental plasticity and human health. *Nature*, *430*(6998), 419-421.
doi:10.1038/nature02725

Bell, E. A., Roe, L. S., & Rolls, B. J. (2003). Sensory-specific satiety is affected more by volume
than by energy content of a liquid food. *Physiology & Behavior, 78*, 593–600.
doi:10.1016/S0031-9384(03)00055-6

Corresponding author: tomiyama@psych.ucla.edu

Belsky, J., Steinberg, L., & Draper, P. (1991). Childhood experience, interpersonal development, and reproductive strategy: An evolutionary theory of socialization. *Child Development*, *62*(4), 647–670. doi: 10.1111/j.1467-8624.1991.tb01558.x

Bernstein, I. L. (1978). Learned taste aversions in children receiving chemotherapy. *Science*, *200*(4347), 1302-1303. doi:10.1126/science.663613

Bernstein, I. L. (1999). Taste aversion learning: A contemporary perspective. *Nutrition, 15*(3), 229-234. doi:10.1016/S0899-9007(98)00192-0

Bernstein, I. L., & Webster, M. M. (1980). Learned taste aversions in humans. *Physiology & Behavior*, *25*(3), 363–6. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/7443805

Birch, L. L. (1999). Development of food preferences. *Annual Review of Nutrition*, *19*, 41–62. Retrieved from http://www.annualreviews.org/doi/pdf/10.1146/annurev.nutr.19.1.41

Birch, L. L., & Deysher, M. (1986). Caloric compensation and sensory specific satiety: Evidence for self regulation of food intake by young children. *Appetite*, *7*(4), 323–331. doi:10.1016/S0195-6663(86)80001-0

Brambila-Macias, J., Shankar, B., Capacci, S., Mazzocchi, M., Perez-cueto, F. J. A., Verbeke, W., & Traill, W. B. (2014). Policy interventions to promote healthy eating: A review of what works, what does not, and what is promising, *32*(4), 365–375.

Brumbach, B. H., Figueredo, A. J., & Ellis, B. J. (2009). Effects of harsh and unpredictable environments in adolescence on development of life history strategies: A longitudinal test of an evolutionary model. *Human Nature*, *20*(1), 25–51. doi:10.1007/s12110-009-9059-3

Buss, D. M. (2009). How can evolutionary psychology successfully explain personality and individual differences? *Perspectives on Psychological Science, 4*(4), 359-366. doi:1 0.1111/j.1745-6924.2009.01138.x

Corresponding author: tomiyama@psych.ucla.edu

Buss, D. M., Haselton, M. G., Shackelford, T. K., Bleske, A. L. & Wakefield, J.

(1998). Adaptations, exaptations, & spandrels. *American Psychologist*, 53, 533-548.

Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/9612136

Cahill, G. J. (1976). Starvation in man. *Clinics in Endocrinology and Metabolism*, *5*(2), 397-415.

doi:10.1016/S0300-595X(76)80028-X

Cannon, D. S., Baker, T. B., & Wehl, C. K. (1981). Emetic and electric shock alcohol aversion

therapy: Six- and twelve-month follow-up. *Journal of Consulting and Clinical

Psychology*, *49*(3), 360–368. doi:10.1037/h0077964

Cashdan, E. (1994). A sensitive period for learning about food. *Human Nature*, *5*(3), 279–291.

Retrieved from http://link.springer.com/article/10.1007/BF02692155

Cashdan, E. (1998). Adaptiveness of food learning and food aversions in children. *Social Science

Information*, *37*(4), 613–632. doi:10.1177/053901898037004003

Chisholm, J., Ellison, P., Evans, J., Lee, P., Lieverman, L., Pavlik, Z., … Worthman, C. (1993).

Death, hope, and sex: Life-history theory and the development of reproductive strategies.

Current Anthropology, *34*(1), 1–24. Retrieved from http://www.jstor.org/stable/2743728

Cooke, L. (2007). The importance of exposure for healthy eating in childhood: A review.

Journal of Human Nutrition and Dietetics, 20(4), 294–301. doi:10.1111/j.1365-

277X.2007.00804.x

Cooke, L. J., Wardle, J., Gibson, E. L., Sapochnik, M., Sheiham, A., & Lawson, M. (2004).

Demographic, familial and trait predictors of fruit and vegetable consumption by pre-

school children. *Public Health Nutrition*, *7*(02), 295–302. doi:10.1079/PHN2003527

Corresponding author: tomiyama@psych.ucla.edu

Cooke, L., & Fildes, A. (2011). The impact of flavour exposure in utero and during milk feeding on food acceptance at weaning and beyond. *Appetite*, *57*(3), 808–811. doi:10.1016/j.appet.2011.05.317

Cooke, L., Wardle, J., & Gibson, E. L. (2003). Relationship between parental report of food neophobia and everyday food consumption in 2–6-year-old children. *Appetite*, 41(2), 205-206. doi:10.1016/j.appet.2007.09.009

Cordain, L., Eaton, S. B., Sebastian, A., Mann, N., Lindeberg, S., Watkins, B. A, & Keefe, J. H. O. (2005). Origins and evolution of the Western diet: Health implications for the 21st century. *The American Journal of Clinical Nutrition*, *81*(2), 341-354. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/15699220

Cordain, L., Miller, J. B., Eaton, S. B., Mann, N., Holt, S. H., & Speth, J. D. (2000). Plant-animal subsistence ratios and macronutrient energy estimations in worldwide hunter-gatherer diets. *The American Journal of Clinical Nutrition*, *71*(3), 682–92. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10702160

Cottone, E., & Byrd-Bredbenner, C. (2007). Knowledge and psychosocial effects of the film Super Size Me on young adults. *Journal of the American Dietetic Association*, *107*(7), 1197–1203. doi:10.1016/j.jada.2007.04.005

Cummings, D. E. (2006). Ghrelin and the short-and long-term regulation of appetite and body weight. *Physiology & Behavior*, *89*(1), 71-84. doi:10.1016/j.physbeh.2006.05.022

Cummings, D. E., & Overduin, J. (2007). Gastrointestinal regulation of food intake. *Journal of Clinical Investigation*, *117*(1), 13. doi:10.1172/JCI30227

Corresponding author: tomiyama@psych.ucla.edu

Cutler, D. M. (2004). Behavioral health interventions: What works and why? In N. B. Anderson, R. A. Bulatao, & B. Cohen (Eds.), *Critical perspectives on racial and ethnic differences in health in late life* (pp. 643-674). Washington, DC: National Academies Press

Darmon, N., & Drewnowski, A. (2008). Does social class predict diet quality? *The American Journal of Clinical Nutrition*, *87*(5), 1107–1117. Retrieved from http://ajcn.nutrition.org/content/87/5/1107.full

Dauchet, L., Amouyel, P., Hercberg, S., & Dallongeville, J. (2006). Fruit and vegetable consumption and risk of coronary heart disease: a meta-analysis of cohort studies. *The Journal of Nutrition*, *136*(10), 2588-2593.

de Wild, V. W. T., de Graaf, C., Boshuizen, H. C., & Jager, G. (2015). Influence of choice on vegetable intake in children: an in-home study. *Appetite*, *91*, 1–6. doi:10.1016/j.appet.2015.03.025

Dinehart, M. E., Hayes, J. E., Bartoshuk, L. M., Lanier, S. L., & Duffy, V. B. (2006). Bitter taste markers explain variability in vegetable sweetness, bitterness, and intake. *Physiology & Behavior*, *87*(2), 304–313. doi:10.1016/j.physbeh.2005.10.018

Domínguez-Rodrigo, M. (2002). Hunting and scavenging by early humans: The state of the debate. *Journal of World Prehistory*, *16*(1), 1-54. doi:10.1023/A:1014507129795

Dovey, T. M., Staples, P. A., Gibson, E. L., & Halford, J. C. G. (2008). Food neophobia and 'picky/fussy' eating in children: A review. *Appetite*, *50*(2-3), 181–93. doi:10.1016/j.appet.2007.09.009

Duncan, G. J., Ziol-Guest, K. M., & Kalil, A. (2010). Early-childhood poverty and adult attainment, behavior, and health. *Child Development*, *81*(1), 306–25. doi:10.1111/j.1467-8624.2009.01396.x

Corresponding author: tomiyama@psych.ucla.edu

Eaton, S. B., Konner, M. J., & Cordain, L. (2010). Diet-dependent acid load, Paleolithic nutrition, and evolutionary health promotion. *The American Journal of Clinical Nutrition*, *91*(2), 295-297. doi:10.3945/ajcn.2009.29058.Am

Eaton, S. B., Konner, M., & Shostak, M. (1988). Stone agers in the fast lane: chronic degenerative diseases in evolutionary perspective. *The American journal of medicine*, *84*(4), 739-749. doi:10.1016/0002-9343(88)90113-1

Epstein, L. H., Carr, K. A., Cavanaugh, M. D., Paluch, R. A., & Bouton, M. E. (2011). Long-term habituation to food in obese and nonobese women. *American Journal of Clinical Nutrition*, *94*(2), 371–376. doi:10.3945/ajcn.110.009035

Fenech, M., Leah, E., & Ferguson, L. R. (2011). Nutrigenetics and nutrigenomics: Viewpoints on the current status and applications in nutrition research and practice. *Journal of Nutrigenetics and Nutrigenomics*, *4(2),* 69–89. doi:10.1159/000327772

Figueredo, A. J., Vásquez, G., Brumbach, B. H., Sefcek, J. A., Kirsner, B. R., & Jacobs, W. J. (2005). The K-factor: Individual differences in life history strategy. *Personality and Individual Differences*, *39*(8), 1349–1360. doi:10.1016/j.paid.2005.06.009

Foley, R. (1995). The adaptive legacy of human evolution: A search for the environment of evolutionary adaptedness. *Evolutionary Anthropology: Issues, News, and Reviews*, *4*(6), 194-203. doi:10.1002/evan.1360040603

Forestell, C. A., & Mennella, J. A. (2007). Early determinants of fruit and vegetable acceptance. *Pediatrics*, *120*(6), 1247–1254. doi:10.1542/peds.2007-0858

Fox, M. K., Pac, S., Devaney, B., & Jankowski, L. (2004). Feeding infants and toddlers study: What foods are infants and toddlers eating? *Journal of the American Dietetic Association*, *104*, 22-30. doi:10.1016/j.jada.2003.10.026

Corresponding author: tomiyama@psych.ucla.edu

Frankwich, K. a., Egnatios, J., Kenyon, M. L., Rutledge, T. R., Liao, P. S., Gupta, S., …
Zarrinpar, A. (2015). Differences in weight loss between persons on standard balanced vs
nutrigenetic diets in a randomized controlled trial. *Clinical Gastroenterology and
Hepatology*. doi:10.1016/j.cgh.2015.02.044

Franz, M. J., Van Wormer, J. J., Crain, a. L., Boucher, J. L., Histon, T., Caplan, W., … Pronk, N.
P. (2007). Weight-loss outcomes: A systematic review and meta-analysis of weight-loss
clinical trials with a minimum 1-year follow-up. *Journal of the American Dietetic
Association*, *107*(10), 1755–1767. doi:10.1016/j.jada.2007.07.017

Fraser, D., & Duncan, I. J. (1998). 'Pleasures,' 'pains' and animal welfare: Toward a natural
history of affect. *Animal Welfare, 7*(4), 383-396. Retrieved from
http://animalstudiesrepository.org/cgi/viewcontent.cgi?article=1038&context=acwp_awa
p

Futuyma, D. J. (2009). Natural selection and adaptation in evolution. In D. J. Futuyma (Ed.),
Evolution (2nd ed.). Sunderland, MA: Sinauer Associates

Garcia, J., Kimeldorf, D. J., & Koelling, R. A. (1955). Conditioned aversion to saccharin
resulting from exposure to gamma radiation. *Science*, *122*(3160), 157–158. Retrieved
from http://psycnet.apa.org/psycinfo/1956-02010-001

Geary, N. (2004). Endocrine controls of eating: CCK, leptin, and ghrelin. *Physiology &
Behaviour*, *81*(5), 719–33. doi:10.1016/j.physbeh.2004.04.013

Glanz, K., Basil, M., Maibach, E., Goldberg, J., & Snyder, D. (1998). Why Americans eat what
they do: Taste, nutrition, cost, convenience, and weight control concerns as influences on
food consumption. *Journal of the American Dietetic Association*, *98*(10), 1118–1126.
doi:10.1016/S0002-8223(98)00260-0

Corresponding author: tomiyama@psych.ucla.edu

Goldenberg, J. L., & Arndt, J. (2008). The implications of death for health: A terror management health model for behavioral health promotion. *Psychological Review*, *115*(4), 1032–1053. doi:10.1037/a0013326

Griskevicius, V., Ackerman, J. M., Cantú, S. M., Delton, A. W., Robertson, T. E., Simpson, J. A., ... & Tybur, J. M. (2013). When the economy falters, do people spend or save? Responses to resource scarcity depend on childhood environments. *Psychological Science*, *24*(2), 197–205. doi:10.1177/0956797612451471

Griskevicius, V., Delton, A. W., Robertson, T. E., & Tybur, J. M. (2011). Environmental contingency in life history strategies: the influence of mortality and socioeconomic status on reproductive timing. *Journal of Personality and Social Psychology*, *100*(2), 241-254. doi:10.1037/a0021082

Griskevicius, V., Tybur, J., Delton, A., & Robertson, T. (2011). The influence of mortality and socioeconomic status on risk and delayed rewards: A life history theory approach. *Journal of Personality and Social Psychology*, *100*(6), 1015–1026. doi:10.1037/a0022403

Heidemann, C., Schulze, M. B., Franco, O. H., Van Dam, R. M., Mantzoros, C. S., & Hu, F. B. (2008). Dietary patterns and risk of mortality from cardiovascular disease, cancer, and all causes in a prospective cohort of women. *Circulation*, *118*(3), 230–237. doi:10.1161/CIRCULATIONAHA.108.771881

Hill, S. E., Rodeheffer, C. D., DelPriore, D. J., & Butterfield, M. E. (2013). Ecological contingencies in women's calorie regulation psychology: A life history approach. *Journal of Experimental Social Psychology*, *49*(5), 888–897. doi:10.1016/j.jesp.2013.03.016

Corresponding author: tomiyama@psych.ucla.edu

Hill, S.E., Prokosch, M.L., DelPriore, D.J., Griskevicius, V., & Kramer, A. (2016). Low

childhood socioeconomic status promotes eating in the absence of energy need.

Psychological Science. Retrieved from

http://personal.tcu.edu/sehill/SES_Hunger_4_Nov_15.pdf

Hockett, B., & Haws, J. (2003). Nutritional ecology and diachronic trends in Paleolithic diet and

health. *Evolutionary Anthropology: Issues, News, and Reviews*, *12*(5), 211-216.

Retrieved from

http://users.clas.ufl.edu/krigbaum/proseminar/hockett&haws_2003_ea.pdf

Hofker, M., & Wijmenga, C. (2009). News and views: A supersized list of obesity genes. *Nature*

Genetics, *41*(2), 139-140. doi:10.1038/ng0209-139

Horn, C. C. (2008). Why is the neurobiology of nausea and vomiting so important? *Appetite*,

50(2-3), 430–434. doi:10.1016/j.biotechadv.2011.08.021.

Jabr, F. (2013, June 3). How to really eat like a hunter-gatherer: Why the paleo diet is half-baked.

Scientific American. Retrieved from http://www. scientificamerican. com/article/why-

paleo-diet-half-baked-how-hunter-gatherer-really-eat

Jones, B. A. (2014). *Incentivizing Children's Fruit and Vegetable Consumption: Evaluation and

Modification of the Food Dudes Program for Sustainable Use in U. S. Elementary

Schools* (Doctoral dissertation)*.* Retrieved from All Graduate Theses and Dissertations*.

(Paper 3699)

Just, D. R., & Price, J. (2013). Using incentives to encourage healthy eating in children. *Journal

of Human Resources, 48*(4), 855-872. Retrieved from

http://jhr.uwpress.org/content/48/4/855.full.pdf

Corresponding author: tomiyama@psych.ucla.edu

Kahn, B. E., & Wansink, B. (2004). The influence of assortment structure on perceived variety

and consumption quantities. *Journal of Consumer Research*, *30*(4), 519–533.

doi:10.1086/380286

Kaplan, H., Hill, K., Lancaster, J., & Hurtado, A. (2000). A theory of human life history

evolution: Diet, intelligence, and longevity. *Evolutionary Anthropology*, *9*(4), 156-185.

doi:10.1002/1520-6505(2000)9:4<156::AID-EVAN5>3.0.CO;2-7

Keller, K. L., Steinmann, L., Nurse, R. J., & Tepper, B. J. (2002). Genetic taste sensitivity to 6-n-

propylthiouracil influences food preference and reported intake in preschool children.

Appetite, *38*(1), 3–12. doi:10.1006/appe.2001.0441

Lieberman, L. S. (2003). Dietary, evolutionary, and modernizing influences on the prevalence of

type 2 diabetes. *Annual Review of Nutrition*, *23*(1), 345-377.

doi:10.1146/annurev.nutr.23.011702.073212

Lieberman, L. S. (2006). Evolutionary and anthropological perspectives on optimal foraging in

obesogenic environments. *Appetite, 47*(1), 3-9. doi:10.1016/j.appet.2006.02.011

Lozano, G. A. (2010). Evolutionary explanations in medicine: How do they differ and how to

benefit from them. *Medical Hypotheses*, *74*(4), 746–749.

doi:10.1016/j.mehy.2009.11.003

Lucock, M. D., Martin, C. E., Yates, Z. R., & Veysey, M. (2014). Diet and our genetic legacy in

the recent anthropocene: A Darwinian perspective to nutritional health. *Journal of

Evidence-Based Complementary & Alternative Medicine*, *19*(1), 68–83.

doi:10.1177/2156587213503345

Lutter, M., & Nestler, E. J. (2009). Homeostatic and hedonic signals interact in the regulation of

food intake. *The Journal of Nutrition, 139*(3), 629–632. doi:10.3945/jn.108.097618.629

Corresponding author: tomiyama@psych.ucla.edu

Maier, A., Chabanet, C., Schaal, B., Issanchou, S., & Leathwood, P. (2007). Effects of repeated exposure on acceptance of initially disliked vegetables in 7-month old infants. *Food Quality and Preference*, *18*(8), 1023–1032. doi:10.1016/j.foodqual.2007.04.005

Mann, T., Tomiyama, A. J., Westling, E., Lew, A.-M., Samuels, B., & Chatman, J. (2007). Medicare's search for effective obesity treatments: Diets are not the answer. *The American Psychologist*, *62*(3), 220–233. doi:10.1037/0003-066X.62.3.220

McCrory, M. A., Burke, A., & Roberts, S. B. (2012). Dietary (sensory) variety and energy balance. *Physiology & Behavior*, *107*(4), 576–583. doi:10.1016/j.physbeh.2012.06.012

Mennella, J. A., Jagnow, C. P., & Beauchamp, G. K. (2001). Prenatal and postnatal flavor learning by human infants. *Pediatrics*, *107*(6), e88–e88. doi:10.1542/peds.107.6.e88

Mennella, J. A., Nicklaus, S., Jagolino, A. L., & Yourshaw, L. M. (2008). Variety is the spice of life: Strategies for promoting fruit and vegetable acceptance during infancy. *Physiology and Behavior*, *94*(1), 29–38. doi:10.1016/j.physbeh.2007.11.014

Mennella, J. A., Pepino, M. Y., & Reed, D. R. (2005). Genetic and environmental determinants of bitter perception and sweet preferences. *Pediatrics*, *115*(2), e216–e222. doi:10.1542/peds.2004-1582

Navarrete, A., van Schaik, C. P., & Isler, K. (2011). Energetics and the evolution of human brain size. *Nature*, *480*(7375), 91-93. doi:10.1038/nature10629

Neel, J. V. (1999). Diabetes mellitus: a "thrifty" genotype rendered detrimental by "progress"? 1962. *Bulletin of the World Health Organization*, *77*(8), 353–362.

Nettle, D. (2010). Why are there social gradients in preventative health behaviour? A perspective from behavioural ecology. *PloS One*, *5*(10), e13371. doi:10.1371/journal.pone.0013371

Corresponding author: tomiyama@psych.ucla.edu

Nielsen, D. E., & El-Sohemy, A. (2012). A randomized trial of genetic information for

personalized nutrition. *Genes and Nutrition*, *7*(4), 559–566. doi:10.1007/s12263-012-

0290-x

Olszewski, P. K., Fredriksson, R., Olszewska, A. M., Stephansson, O., Alsio, J., Radomska, K. J.,

… Schioth, H. B. (2009). Hypothalamic FTO is associated with the regulation of energy

intake not feeding reward. *BMC Neuroscience*, *12*(129), 1–12. doi:10.1186/1471-2202-

10-129

Patlan, K. L. & Mendelson, M. (2016). WIC Participant and Program Characteristics 2014: Food

Package Report. Prepared by Insight Policy Research under Contract No. AG   3198   C

11   0010. Alexandria, VA: U.S. Department of Agriculture, Food and Nutrition Service.

Pelchat, M. L., & Rozin, P. (1982). The special role of nausea in the acquisition of food dislikes

by humans. *Appetite*, *3*(4), 341–351. doi:10.1016/S0195-6663(82)80052-4

Pepper, G. V, & Nettle, D. (2013). Perceived extrinsic mortality risk and reported effort in

looking after health: Testing a behavioural ecological prediction. *PeerJPrePrints*,

1(e29v2), 1–5. doi:10.7287/peerj.preprints.141v2

Pepper, G. V, & Nettle, D. (2014a). Out of control mortality matters: The effect of perceived

uncontrollable mortality risk on a health-related decision. *PeerJ, 2*(e459), 1–34.

doi:10.7717/peerj.459

Pepper, G. V., & Nettle, D. (2014b). Socioeconomic disparities in health behaviour: An

evolutionary perspective. In M. A. Gibson & D. W. Lawson (Eds.), *Applied Evolutionary*

Anthropology: Darwinian Approaches to Contemporary World Issues (225-243). New

York: Springer.

Corresponding author: tomiyama@psych.ucla.edu

Perello, M., Sakata, I., Birnbaum, S., Chuang, J. C., Osborne-Lawrence, S., Rovinsky, S. A., ... & Zigman, J. M. (2010). Ghrelin increases the rewarding value of high-fat diet in an orexin-dependent manner. *Biological Psychiatry*, *67*(9), 880-886. doi: 10.1016/j.biopsych.2009.10.030

Pliner, P. (1982). The effects of mere exposure on liking for edible substances. *Appetite*, *3*(3), 283–290. doi:10.1016/S0195-6663(82)80026-3

Pontzer, H., Raichlen, D. A., Wood, B. M., Mabulla, A. Z., Racette, S. B., & Marlowe, F. W. (2012). Hunter-gatherer energetics and human obesity. *PLoS One*, *7*(7), e40503. doi:10.1371/journal.pone.0040503

Raghunathan, R., Naylor, R. W., & Hoyer, W. D. (2006). The unhealthy = Tasty intuition and its effects on taste inferences, enjoyment, and choice of food products. *Journal of Marketing, 70*(4), 170-184. doi:10.1509/jmkg.70.4.170

Raynor, H. A. (2012). Can limiting dietary variety assist with reducing energy intake and weight loss? *Physiology and Behavior*, *106*(3), 356–361. doi:10.1016/j.physbeh.2012.03.012

Raynor, H. A., & Epstein, L. H. (2001). Dietary variety, energy regulation, and obesity. *Psychological Bulletin*, *127*(3), 325–341. doi:10.1037//0033-2909.127.3.325

Roe, L. S., Meengs, J. S., Birch, L. L., & Rolls, B. J. (2013). Serving a variety of vegetables and fruit as a snack increased intake in preschool children. *American Journal of Clinical Nutrition*, *98*(3), 693–699. doi:10.3945/ajcn.113.062901

Rolls, B. J. (1986). Sensory-specific satiety. *Nutrition Reviews*, *44*(3), 93–101. doi:10.1016/S0031-9384(03)00055-6

Corresponding author: tomiyama@psych.ucla.edu

Rolls, B. J., Rowe, E. A., Rolls, E. T., Kingston, B., Megson, A., & Gunary, R. (1981). Variety in a meal enhances food intake in man. *Physiology & Behavior*, *26*(2), 215–221. doi:10.1016/0031-9384(81)90014-7

Rozin, P., & Fallon, A. E. (1987). A perspective on disgust. *Psychological Review*, *94*(1), 23-41. doi:10.1037/0033-295X.94.1.23

Rozin, P., & Tuorila, H. (1993). Simultaneous and temporal contextual influences on food acceptance. *Food Quality and Preference*, *4*(1-2), 11–20. doi:10.1016/0950-3293(93)90309-T

Rozin, P., & Vollmecke, T. A. (1986). Food likes and dislikes. *Annual Review of Nutrition*, *6*(101), 433–56. doi:10.1146/annurev.nu.06.070186.002245

Rozin, P., Scott, S., Dingley, M., Urbanek, J. K., & Kaltenbach, M. (2011). Nudge to obesity I: Minor changes in accessibility decrease food intake. *Judgment and Decision Making, 6*(4), 323–332. Retrieved from http://journal.sjdm.org/11/11213/jdm11213.html

Saper, C., Chou, T., & Elmquist, J. (2002). The need to feed: Homeostatic and hedonic control of eating. *Neuron*, *36*, 199–211. doi:10.1016/S0896-6273(02)00969-8

Scalera, G. (2002). Effects of conditioned food aversions on nutritional behaviour in humans. *Nutritional Neuroscience*, *5*(3), 159–188. doi:10.1080/10284150290013059

Scollan-Koliopoulos, M., Walker, E. A., & Rapp, K. J. (2011). Self-regulation theory and the multigenerational legacy of diabetes. *The Diabetes Educator*, *37*(5), 669–679. doi:10.1177/0145721711416133

Scott, K. (2005). Taste recognition: Food for thought. *Neuron, 48*, 455–464. doi:10.1016/j.neuron.2005.10.015

Corresponding author: tomiyama@psych.ucla.edu

Simopoulos, A. P. (1999). *Evolutionary aspects of nutrition and health: Diet, exercise, genetics, and chronic disease* (Vol. 84). Basel: Karger Publishers. doi:10.1159/000059680

Simpson, J. A., Griskevicius, V., Kuo, S. I.C., Sung, S., & Collins, W. A. (2012). Evolution, stress, and sensitive periods: The influence of unpredictability in early versus late childhood on sex and risky behavior. *Developmental Psychology*, *48*(3), 674–686. doi:10.1037/a0027293

Smith, S. M. K., Guenther, P. M., Subar, A. F., Kirkpatrick, S. I., & Dodd, K. W. (2010). Americans do not meet federal dietary recommendations. *The Journal of Nutrition, 145*(5). doi:10.3945/jn.110.124826

Speakman, J. R. (2013). Evolutionary perspectives on the obesity epidemic: adaptive, maladaptive, and neutral viewpoints. *Annual Review of Nutrition*, *33*, 289–317. doi:10.1146/annurev-nutr-071811-150711

Stevenson, R. J., Oaten, M. J., Case, T. I., Repacholi, B. M., & Wagland, P. (2010). Children's response to adult disgust elicitors: Development and acquisition. *Developmental Psychology, 46*(1), 165. doi:10.1037/a0016692

Tahiri, M., Mottillo, S., Joseph, L., Pilote, L., & Eisenberg, M. J. (2012). Alternative smoking cessation aids: A meta-analysis of randomized controlled trials. *The American Journal of Medicine*, *125*(6), 576-584. doi:10.1016/j.amjmed.2011.09.028

Thomas, C., Hyppönen, E., & Power, C. (2008). Obesity and type 2 diabetes risk in midadult life: The role of childhood adversity. *Pediatrics*, *121*(5), e1240–1249. doi:10.1542/peds.2007-2403

Tiger, L. (1992). The Pursuit of Pleasure. Boston, MA: Little Brown and Company.

Corresponding author: tomiyama@psych.ucla.edu

Tomiyama, A., Ahlstrom, B., & Mann, T. (2013). Long-term effects of dieting: Is weight loss

related to health? *Social and Personality Psychology Compass*, 7(12), 861-877.

doi:10.1111/spc3.12076

Turner, B. L., & Thompson, A. L. (2013). Beyond the Paleolithic prescription: Incorporating

diversity and flexibility in the study of human diet evolution. *Nutrition Reviews*, *71*(8),

501-510. doi:10.1111/nure.12039

Tybur, J. M., Bryan, A. D., & Hooper, A. E. C. (2012). An evolutionary perspective on health

psychology: New approaches and applications. *Evolutionary Psychology*, *10*(5), 855–67.

Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/23253791

Tybur, J. M., Bryan, A. D., Magnan, R. E., & Hooper, A. E. C. (2011). Smells like safe sex:

Olfactory pathogen primes increase intentions to use condoms. *Psychological Science*,

22(4), 478–480. doi:10.1177/0956797611400096

Ulijaszek, S. J. (2002). Human eating behaviour in an evolutionary ecological context.

Proceedings of the Nutrition Society, *61*(4), 517–526. doi:10.1079/PNS2002180

Union of Concerned Scientists (2013). Investing in healthy food will save lives and dollars.

Retrieved from http://www.ucsusa.org/food_and_agriculture/solutions/expand-healthy-

food-access/11-trillion-reward.html#.VxLcPpMrLu4

Wang, X., Ouyang, Y., Liu, J., Zhu, M., Zhao, G., Bao, W., & Hu, F. B. (2014). Fruit and

vegetable consumption and mortality from all causes, cardiovascular disease, and cancer:

systematic review and dose-response meta-analysis of prospective cohort studies. *BMJ,*

349:g4490. doi: 10.1136/bmj.g4490

Corresponding author: tomiyama@psych.ucla.edu

Wansink, B. (2004). Environmental factors that increase the food intake and consumption volume of unknowing consumers. *Annual Review of Nutrition*, *24*(217), 455–479. doi:10.1146/annurev.nutr.24.012003.132140

Wardle, J., & Cooke, L. (2008). Genetic and environmental determinants of children's food preferences. *British Journal of Nutrition*, *99*(S1), S15-S21. doi:10.1017/S000711450889246X

Wardle, J., Herrera, M.L., Cooke, L., & Gibson, E. L. (2003). Modifying children's food preferences: The effects of exposure and reward on acceptance of an unfamiliar vegetable. *European Journal of Clinical Nutrition*, *57*(2), 341–348. doi:10.1038/sj.ejcn.1601541

Wertz, D. C., Fanos, J. H., & Reilly, P. R. (1994). Genetic testing for children and adolescents: Who decides? *JAMA*, *272*(11), 875-881. doi:10.1001/jama.1994.03520110055029.

Williams-Piehota, P., Schneider, T. R., Pizarro, J., Mowad, L., & Salovey, P. (2004). Matching health messages to health locus of control beliefs for promoting mammography utilization. *Psychology & Health, 19*(4), 37–41. doi:10.1080/08870440310001652678

Yarmolinsky, D., Zuker, C., & Ryba, N. (2009). Common sense about taste: From mammals to insects. *Cell, 139*(2). doi:10.1016/j.cell.2009.10.001

Corresponding author: tomiyama@psych.ucla.edu

Evolved Mechanism	How it Affects Eating Behaviour	Example Intervention	Suggested Age for First Intervention
Familiarity Bias	Eating more of foods to which one has been repeatedly exposed	Providing pregnant and new mothers with vegetables to eat	In utero
Sensory-Specific Satiety	Eating less after experiencing sensory specific satiety, and eating more after experiencing variety	Providing parents with various pureed vegetables to feed to infants	Infancy
Disgust	Eating less of foods that have been paired with disgust	Pairing unhealthy foods with disgust	Age 2
Life History Strategies	Eating less healthy as the result of fast life history strategies, and eating healthier as the result of slow life history strategies	Matching incentives for healthy eating with individuals' life history strategies	Age 5
Genetic Predispositions	Eating more of foods one is genetically predisposed to like	Tailoring diets to match individuals' gene-based food preferences	Age 18
Perceptions of the Controllability of Mortality	Eating healthier when mortality is viewed as controllable, and eating less healthy when mortality is viewed as uncontrollable	Reminders above grocery store doors that mortality is controllable	Age 18

Corresponding author: tomiyama@psych.ucla.edu