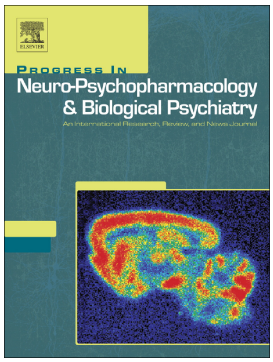

A narrative review of highly processed food addiction across the lifespan

Emma T. Schiestl, Julia M. Rios, Lindsey Parnarouskis, Jenna R. Cummings, Ashley N. Gearhardt

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A Narrative Review of Highly Processed Food Addiction across the Lifespan

Emma T. Schiestl, Julia M. Rios, Lindsey Parnarouskis, Jenna R. Cummings and Ashley N. Gearhardt

University of Michigan

Corresponding Author: Dr. Ashley N. Gearhardt, University of Michigan, 530 Church St. Ann Arbor, Michigan, 48109, agearhar@umich.edu

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Highlights

- Highly processed (HP) foods are associated with changes in reward functioning and the development of addictive-like eating behaviors.
- HP food addiction is associated with negative outcomes (e.g., elevated BMI, increased binge eating) at every life stage.
- The study of food addiction across the lifespan has important implications for treatment and the adaptation of public policy.

Abstract

Evidence is growing that highly processed (HP) foods (i.e., foods high in refined carbohydrates and fat) are highly effective in activating reward systems and may even be capable of triggering addictive processes. Unlike traditional drugs of abuse, exposure to HP foods is common very early in development. HP food addiction has been associated with negative outcomes, including higher body mass index (BMI), more frequent binge eating, greater failure in weight loss treatment trials, and poorer mental and physical health. Although most research on HP food addiction has been conducted using adult samples, research on this topic now spans across the life span beginning *in utero* and extending through older adulthood. HP food addiction and related reward-based changes are associated with negative outcomes at every life stage, which has important implications for developmentally tailored prevention and treatment efforts. Using a developmentally informed approach, the current study comprehensively reviews the existing research on HP food addiction across the lifespan and highlights important areas of future research.

Key words: food addiction, lifespan, prenatal, infancy, childhood, adolescence, adulthood

Introduction

Since the 1970s, the rate of obesity in the United States has been rising across the lifespan (Kranjac & Wagmiller, 2016). Although many factors contribute to obesity, the biggest factor is arguably the dramatic changes that have occurred in the modern food environment (Bentley, Ruck, & Fouts, 2020; Willett et al., 2019). Highly processed (HP) foods that have artificially high levels of refined carbohydrates (e.g., white flour, sugar), fat, and salt now dominate the food environment (Monteiro et al., 2018). HP foods are engineered to be highly rewarding and they are easily accessible, inexpensive, and heavily marketed (Moodie et al., 2013). Growing evidence in both animals and humans suggests HP foods may be capable of triggering addictive processes that drive compulsive patterns of intake (Ahmed, Guillem, & Vandaele, 2013; Davis, 2013; Small & DiFeliceantonio, 2019). Exposure to these potentially addictive HP foods is widespread and occurs at every stage of development from *in utero* to older adulthood (Bueno, Marchioni, César, & Fisberg, 2012; Whisner et al., 2015). Exposure to addictive substances early in development increases vulnerability for problematic patterns of use (Hingson, Heeren, & Winter, 2006). For most addictive substances, initial exposure occurs in adolescence or early adulthood (Hingson et al., 2006). By contrast, exposure to HP foods occurs very early in development. For example, the World Health Organization recently issued an alert about the excessive levels of added sugars (>30% of calories from sugar) in baby food (Beal, 2020). Moreover, 84.4% of infants and toddlers consume added sugars on a given day (Herrick, Fryar, Hamner, Park, & Ogden, 2020). Thus, it is essential to consider how the potentially addictive nature of HP foods may negatively affect people of all ages.

In the current manuscript, we conducted a narrative review of the existing research on how exposure to HP foods may result in reward-related differences and HP food addiction across

the lifespan. We identified the research studies through independent searches by all study authors via a variety of databases (Google Scholar, PsycINFO, and PubMed) and reference sections of relevant articles. We considered relevant studies regardless of study design and year of publication. However, we included only research reported in English. Our aim was to provide a developmentally informed analysis of existing studies and to highlight important areas of future research to enhance our understanding of HP food addiction across all age groups.

Prenatal Risk Factors for HP Food Addiction

The impact of exposure to HP foods begins before birth. Although most research focuses on understanding how maternal undernutrition impacts postnatal weight and dietary outcomes, some studies have examined the impacts of maternal overnutrition or HP food consumption (Howie, Sloboda, Kamal, & Vickers, 2009). Observational studies show associations between maternal gestational diabetes or excessive weight gain and child adiposity in the first years of life (Godfrey, Barker, Robinson, & Osmond, 1997; Oken, Levitan, & Gillman, 2008; Stettler et al., 2000). Maternal consumption of HP foods and calorically dense diets are linked to increased risk of obesity and related health problems for offspring from birth through adolescence (Silverman, Rizzo, Cho, & Metzger, 1998). Furthermore, maternal diet during pregnancy is linked to changes in infant eating behaviors, food preferences, and weight trajectories (Anzman, Rollins, & Birch, 2010). Such studies provide a theoretical basis to suggest that prenatal exposure to HP foods via maternal diet may be an early risk factor for addictive patterns of consumption later in life.

Converging evidence from non-human animal studies also highlights the importance of the *in utero* food environment on food preferences, eating behaviors, and body mass (Gugusheff, Ong, & Muhlhausler, 2015). Rats born to mothers fed a palatable “junk food” diet of HP foods during gestation show an amplified preference for HP foods over healthy chow and eat beyond

caloric need (Bayol, Farrington, & Stickland; Ong & Muhlhausler, 2011). In order to further isolate the effects of exposure to HP foods before birth, some studies have utilized a cross-fostering paradigm. This paradigm allows offspring born to mothers fed a junk food diet to be transferred to mothers fed control diets (and *vice versa*) immediately after birth. In an early cross-fostering study, prenatal exposure to HP foods induced an exacerbated preference for HP foods and a predisposition for obesity in the offspring, regardless of maternal diet after birth (Chang et al., 2008). However, two follow-up studies concluded that additional exposure to HP foods after birth may also be necessary (Naef et al., 2011; Vucetic et al., 2010) or even sufficient (Gugusheff et al., 2013) to produce these effects. Gugusheff and colleagues also propose that the effects of prenatal exposure to HP foods have the potential to be reversed with a healthy maternal diet during the lactation/suckling period (2015). Despite the strength of the non-human animal literature, translational experimental studies with humans are needed (e.g., nutritional interventions for pregnant women that aim to reduce intake of HP foods during gestation and assesses eating behaviors of the child later in life).

It is important to note that the development of flavor preferences may play a role in the association between prenatal exposure to HP foods and maladaptive outcomes post-birth. Exposure to certain flavors *in utero* influences the likelihood of postnatal acceptance of those flavors by the infant (Mennella, Jagnow, & Beauchamp, 2001; Schaal, Marlier, & Soussignan, 2000). High maternal consumption of HP foods, particularly in place of non-HP foods, may limit an infant's likelihood of accepting healthier foods (e.g., vegetables; Anzman et al., 2010). In turn, infant flavor preference continues to shape the child's diet composition as soon as solid foods are introduced (Anliker, Bartoshuk, Ferris, & Hooks, 1991; Mennella, Nicklaus, Jagolino, & Yourshaw, 2008). Thus, through flavor conditioning, prenatal exposure to HP foods *in utero*

may influence preference for HP foods over healthier options. Such predispositions appear to have important implications for ongoing eating behaviors and weight outcomes throughout the lifespan and should be a major focus of future research efforts (Eichen, Chen, Schmitz, Arlt, & McCloskey, 2016; Loxton & Tipman, 2017; Schulte, Grilo, & Gearhardt, 2016).

In addition to the potential effects of prenatal exposure to HP food intake, prenatal exposure to substance use may also have implications for the development of HP food addiction across the lifespan. In a meta-analysis of 14 observational studies, prenatal exposure to maternal cigarette smoking was identified as a consistent risk factor for overweight and obesity from ages 3 to 33 (Oken et al., 2008). Some observational studies have also identified prenatal exposure to maternal alcohol use as a risk factor for overweight and obesity in youth ages 2 to 19 (Fuglestad et al., 2014; Werts, Van Calcar, Wargowski, & Smith, 2014). Although these studies establish that prenatal exposure to substance use may be a critical risk factor for overweight and obesity, there is less research investigating whether prenatal exposure to substance use increases risk specifically for HP food addiction.

In particular, if prenatal exposure to substance use selectively impacts the intake of HP foods, but not less processed foods (Schulte, Avena, & Gearhardt, 2015), this would provide further support that prenatal exposure to substances is directly related to the development of HP food addiction. Some evidence for this theory already exists. For example, in one observational study, prenatal exposure to maternal cigarette smoking predicted greater fat intake among adolescents ages 13 to 19 (Haghighi et al., 2013). Additionally, in our own analysis of data from the Fragile Families and Child Wellbeing Study (N = 4,898), prenatal exposure to maternal cigarette smoking and alcohol use predicted greater HP food (i.e., soda, candy or sweets) intake among 5-year-old youth but did not predict the intake of other foods (i.e., fruits and vegetables;

Cummings & Gearhardt, in press). Furthermore, prenatal exposure to maternal illicit drug use predicted greater HP food intake (but did not predict intake of other foods) among youth at ages 5, 9, and 15 (Cummings & Gearhardt, in press). These results suggest that prenatal exposure to substances may have lasting effects on HP food intake during childhood and adolescence. However, more research is needed, and these observational studies are limited in identifying potential mechanisms.

One mechanism that potentially explains the effects of prenatal exposure to both HP food intake and substance use on risk for HP food addiction is altered neural-reward circuitry. In bovine and rodent models where offspring are prenatally exposed to high-calorie or HP diets, offspring show amplified opioid and dopamine signaling (i.e., increased sensitivity of μ-opioid receptor and dopamine active transporter) at six weeks of age (Muhlhausler, Adam, Findlay, Duffield, & McMillen, 2006; Ong & Muhlhausler, 2011). These are the same neural pathways that underlie the compulsive consummatory behaviors characteristic of addiction (Adinoff, 2004) and are associated with heightened preference for HP foods across the lifetime (Bayol, Farrington, & Stickland, 2007; Ong & Muhlhausler, 2011). Moreover, in rodent models where pups are prenatally exposed to addictive substances (i.e., alcohol, cocaine, morphine, marijuana, and nicotine), they demonstrate similar changes to neural-reward circuitry (Malanga & Kosofsky, 2003). They are also more responsive to multiple drugs as shown in intravenous self-administration, intracranial self-stimulation, and conditioned place preference paradigms (Malanga & Kosofsky, 2003). Prenatal exposure to HP food intake and substance use may therefore impact the risk for HP food addiction through neural reward mechanisms, but more research is needed.

HP Food Addiction Risk in Newborns and Infants

The first years of life are a critical period where early exposure to HP foods may have high potential for lifelong effects on food preference, eating behavior, and obesity risk (Anzman et al., 2010). Neural plasticity is highest in early development, allowing phenotypic, epigenetic, and genetic potential to be dramatically altered by environmental factors (Gluckman & Hanson, 2008). As such, any relations between increased HP food consumption and dietary preferences for high-sugar or high-fat foods (Thanarajah & Tittgemeryer, 2020) or excessive eating behaviors (Schulte et al., 2015) may be particularly impactful during this stage of life. Furthermore, children at this age may also be especially vulnerable to developing predilections for HP foods due to their enhanced preference for sweeter foods compared to adults (Schwartz et al., 2009). This is crucial to consider because infants in the US are often exposed to HP foods within the first year of life (Archibald, Dolinsky, & Azad, 2018). Additionally, infancy is a period of rapid learning marked by important transitions in consummatory behaviors (Alberts, 1994) and evidence indicates that early life eating habits tend to persist over time (Fletcher et al., 2017; Rose, Birch, & Savage, 2017).

From the beginning of life, neural reward mechanisms in response to HP foods influence eating behaviors. Sucrose, in particular, activates the endogenous opioid system, which underlies experiences of pleasure and hedonic response (Berridge, Ho, Richard, & DiFeliceantonio, 2010). Babies are born liking sugar, and individual differences in hedonic reactivity to sweetness can be measured within the first weeks of life (Anzman et al., 2010; Steiner, 1974). Young children who have stronger hedonic response to HP foods may be more likely than their lower-hedonic response counterparts to exhibit non-homeostatic eating behaviors (i.e., consuming HP foods over healthier alternatives, overconsuming HP foods beyond caloric need, continuing to eat HP foods even when satiated; Ziauddeen, Alonso-Alonso, Hill, Kelley, & Khan, 2015). Therefore,

inherent hedonic responsiveness and access to HP foods immediately after birth are important risk factors for adverse eating and weight outcomes.

Sucrose also has well-documented analgesic effects in newborns (Lumeng et al. 2020) and provides a precise index of opioid reward functioning during this period (Segato et al., 1997; Blass et al., 1987; Bertino et al., 1991; Fatino et al., 1986). The ability of sucrose to act as an analgesic in the newborn stage is associated with hedonic response to sweet food, propensity to overeat, and adiposity (Pepino & Mennella, 2005; Lumeng et al., 2020). Furthermore, individual differences in behavioral pain-blocking effects of sucrose in the newborn period are associated with greater infant weight gain (Lumeng et al., 2020). Individual differences in the analgesic effects of sucrose appear to remain relevant to sweet taste preferences in childhood, although this association was less pronounced in children with overweight or risk of overweight (Pepino & Mennella, 2005). This may be explained by evidence that chronic consumption of foods higher in carbohydrate and sugar modifies opioid functioning (Colantuoni et al., 2001; Levine et al., 2003) and interferes with the analgesic effects of sucrose (Kanarek et al., 1997; D'Anci et al., 1996). These combined findings provide support for a conceptual model in which stronger opioid response to sucrose in newborns predicts enhanced preference and intake of sweet foods across the lifespan, thereby predisposing such individuals to overeating, overweight, and dampened analgesic response to sucrose over time.

Patterns of HP food consumption in early development may also set the stage for HP food addiction later in life. While hedonic response predicts intake of HP foods, intake of HP foods also exacerbates hedonic response (Bayol et al., 2007). In non-human animal models, repeated overconsumption of HP foods results in neurobiological changes in reward-related regions of the brain (e.g., increase of extracellular dopamine in the nucleus accumbens)

mimicking the effects of other drugs of abuse (Avena, Rada, & Hoebel, 2008). In turn, these neurobiological adaptations lead to the onset of behaviors associated with addiction (Lüscher & Malenka, 2011). Thus, excessive exposure to HP foods in infancy may create long-term changes to neural reward systems especially among infants inherently more reward responsive to HP foods (Davis, Strachan, & Berkson, 2004; Stice, Spoor, Bohon, Veldhuizen, & Small, 2008). Despite this, few studies have examined how consumption of HP foods in the earliest stages of development may have long lasting effects on neural reward functioning in human populations (Sullivan, Smith, & Grove, 2010; Crume et al., 2016; Martin et al., 2016). This will be an important direction for future research to further our understanding of complex and multidirectional relationships between hedonic response and HP food consumption.

Collectively, these findings illustrate how early life predispositions and exposures may lead at-risk infants to be particularly vulnerable to HP food addiction throughout the lifetime. Specifically, constructs such as reward responsivity and food preference in infancy appear to have utility in predicting obesity and HP food addiction later in life. Considering how early markers of reward are indicative of addictive phenotypes is critical to advancing our understanding of etiological factors and the application of intervention approaches earlier in the lifespan. Furthermore, identification of environmental features that promote the consumption of healthy foods and reduce the overconsumption of HP foods in the earliest stages of development may be particularly important for minimizing negative eating-related outcomes. Financial incentives for parents to select healthier food options, the provision of nutritional information for all food products and menus, removal of HP foods in schools and children's programs, and the inclusion of more healthy food choices in home and school settings have all been identified as

potential strategies warranting further research attention (Lowe, 2003; Goldman, Radnitz, & McGrath, 2012; Fox et al., 2004).

HP Food Addiction in Middle Childhood

HP food addiction in childhood is a growing area of interest due to increasing levels of childhood obesity and diet-related disease (Hales, Fryar, Carroll, Freedman, & Ogden, 2018). As such, developmentally sensitive measures have been created to determine if HP food addiction may contribute to the rise of obesity and other problematic eating behaviors early in childhood. The Yale Food Addiction Scale for Children (YFAS-C) is a commonly used measure of HP food addiction in children and has been validated in samples ranging from 4 to 18 years old (Gearhardt, Roberto, Seamans, Corbin, & Brownell, 2013). The YFAS-C was developed as a self-report measure (Gearhardt, et al., 2013), but parent-report versions have been used with younger children (Burrows, et al., 2017). Items on the YFAS-C represent symptoms of DSM IV substance dependence (e.g., loss of control, inability to cut down, withdrawal, tolerance) to probe for symptoms of addictive-like eating.

The prevalence of HP food addiction in childhood varies by sample, ranging from 4-to-24% (Filgueiras et al., 2019; Laurent & Sibold, 2016). However, an even greater proportion of children meet criteria for three or more symptoms of HP food addiction, but do not meet clinical levels of distress or impairment (Filgueiras et al., 2019; Gearhardt et al., 2013). HP food addiction in childhood is associated with increased emotional eating, greater *ad libitum* food intake in laboratory settings, higher body mass index (BMI), increased consumption of HP foods and added sugars, and appetitive responsiveness (Filgueiras et al., 2019; Gearhardt et al., 2013; Laurent & Sibold, 2016). Due to these negative associations, HP food addiction appears to be a clinically relevant condition in childhood.

Like enhanced hedonic responsivity in infancy, enhanced reward sensitivity to HP foods in childhood has been proposed as an important risk factor for the development of HP food addiction (Stice & Burger, 2019; Stojek & MacKillop, 2017). Reward sensitivity in childhood can be assessed in many ways. Behavioral tasks measuring the relative-reinforcement value (RRV) of food (e.g., willingness to work for food reward over alternative reinforcers) and degree of eating beyond homeostatic need (e.g., eating in the absence of hunger (EAH)) are particularly well utilized (Epstein, Paluch, Roemmich, & Beecher, 2007; Fisher & Birch, 1999; Temple, Legierski, Giacomelli, Salvy, & Epstein, 2008). In a sample of children aged 8-10 years old, greater RRV for food was positively associated with increased caloric intake, higher body weight, and amplified obesity risk (Temple et al., 2008). Similarly, a systematic review of 19 cross-sectional and prospective EAH studies in children aged 3-12 years old found that EAH was positively correlated with increased weight gain, and overweight and obese weight status (Lansigan, Emond, & Gilbert-Diamond, 2015). Thus, childhood RRV and EAH studies suggest that reward sensitivity may be an important risk factor for HP food addiction in childhood.

Another growing area of interest is the potential existence of a withdrawal syndrome in children with HP food addiction. In substance use disorders, withdrawal refers to the cascade of aversive physical, affective, and cognitive symptoms that occur when use of an addictive substance is reduced or stopped (American Psychiatric Association, 2013). Avoidance of withdrawal often serves as a barrier to initiating quit attempts and experiences of withdrawal are one of the most powerful predictors of relapse (Connors, Maisto, & Donovan, 1996). Pediatric weight loss studies have extremely high attrition rates, with up to 73% of families dropping out of treatment, often within the first few weeks (Skelton & Beech, 2011). Withdrawal from HP

foods may therefore be an important, understudied contributor to families' lack of treatment adherence.

Preliminary research suggests that children experience a withdrawal syndrome when their parents restrict access to HP foods. The Highly Processed Food Withdrawal Scale for Children (ProWS-C) was developed to measure parent-reported HP food withdrawal in children ages 3 to 11, and examines physical, affective, and cognitive symptoms (i.e., physical, affective, and cognitive; Parnarouskis, Schulte, Lumeng, & Gearhardt, 2020). High scores on the ProWS-C are associated with higher levels of parent-reported child HP food addiction and higher child BMI. A recent qualitative study also found that, when children were not able to access sugar-sweetened beverages, they reported cravings and negative affective symptoms (Sylvetsky et al., 2020). To confirm and expand upon these preliminary findings of HP food withdrawal in children, future prospective research using multiple informants (i.e., teachers, self-report for older children) is needed. Furthermore, it will be necessary to determine if this pattern of behavior is specific to HP foods or if it also occurs when a child's access to any rewarding stimulus is restricted.

The concept of children as young as three years old experiencing withdrawal is novel, as most addictive substances are not commonly used until adolescence or early adulthood (Johnston et al., 2019). In order to develop effective interventions, it will be important to understand how addictive processes like withdrawal present differently in children compared to adolescents and adults. Even if the HP food withdrawal symptoms in children are relatively mild compared to substances like nicotine, they should be addressed directly due to the potential for addictive processes to harm long-term development. Future research should continue to examine this construct in younger children.

HP Food Addiction in Adolescents

Due to biological, psychological, and social changes, adolescence is a high risk period for the consumption of addictive substances (Gray & Squeglia, 2018). Although most adolescents do not go on to develop clinical levels of pathology, persistent use of substances during adolescence is highly associated with the development of substance use disorders later in life (SAMHSA, 2017). Adolescent substance use is also associated with cognitive consequences (e.g., slower psychomotor speed, poor verbal memory, poor visuospatial functioning) and negative psychological experiences (e.g., anxiety, depression), although the directionality of these associations remain unclear (Fishbein, Rose, Darcey, Belcher, & VanMeter, 2016; Nguyen-Louie et al., 2015).

Adolescence is also a high risk period for the development of obesity (Abarca-Gómez et al., 2017) and epidemiological research indicates that the prevalence of obesity in adolescence is growing (Hales et al., 2018). Adolescents also demonstrate a stronger preference for sweeter substances relative to young adults, potentially making them more prone to the overconsumption of HP foods (Mennella, Lukasewycz, Griffith, & Beauchamp, 2011). Furthermore, neurobiological research indicates that adolescents whose parents are overweight show increased reward circuitry activity in response to food cues, despite being normal weight (Stice, Yokum, Burger, Epstein, & Smail, 2011). In adults, elevated reward activity is associated with lower weight loss success, suggesting that these adolescents may be at risk for future weight concerns (Murdaugh, Cox, Cook III, & Weller, 2012). In light of these findings, research into HP food addiction in adolescence is of critical importance.

Research on HP food addiction in adolescents is growing. Prevalence rates vary by sample, ranging from 2.6% in a community sample of Dutch adolescents to 38% in a sample of adolescents with obesity seeking weight loss treatment (Meule, Hermann, & Kübler, 2015; Mies

et al., 2017). The most common symptoms endorsed by adolescents are the inability to cut down, use despite negative consequences, and giving up important activities. Symptoms such as tolerance and withdrawal are less common (Albayrak et al., 2017; Meule et al., 2015; Schulte, Jacques-Tiura, Gearhardt, & Naar, 2018). HP food addiction in adolescence is associated with numerous consequences including elevated added sugar intake, elevated consumption of HP foods, objective binge episodes, objective over eating, more frequent food cravings, higher rates of depression, and increased motor and attentional impulsivity (Lin, Imani, Griffiths, & Pakpour, 2020; Mies et al., 2017; Schulte et al., 2018; Zhao et al., 2018). Furthermore, one study found that adolescents with HP food addiction completing outpatient weight loss treatment were more likely to leave treatment early compared to their peers without HP food addiction (Tompkins, Laurent, & Brock, 2017). Similar results have been found for adolescents outside of the US suggesting that HP food addiction in adolescence may be relevant across cultures (Ahmed, Sayed, Mostafa, & Abdelaziz, 2016; Borisenkov, Tserne, & Bakutova, 2018; Chen, Tang, Guo, Liu, & Xiao, 2015).

Although many aspects of HP food addiction in adolescence parallel HP food addiction in childhood and adulthood, some associations differ. For example, although dietary restraint (e.g., intentions or actions to restrict food intake) is not associated or is negatively associated with food addiction in children and adults (Carter, Van Wijk, & Rowsell, 2019; Gearhardt, Corbin, & Brownell, 2016), positive associations have been found in adolescence (Schiestl & Gearhardt, 2018). Additionally, Richmond, Roberto, & Gearhardt (2017) found that symptoms of HP food addiction were associated with greater *ad libitum* calorie intake in children ages 4 to 8, while no association was found between HP food addiction and calorie intake in older children and adolescents (ages 9 to 16). The lack of association in older children may be a manifestation

of dietary restraint due to increasing body shape and weight concerns that do not yet exist in younger children (Richmond, Roberto, & Gearhardt, 2017). Furthermore, in adulthood, perpetual attempts and failures to engage in dietary restriction may reduce motivation to continue dieting over time (Gearhardt et al., 2016), potentially eliminating the association between HP food addiction and dietary restraint. Interestingly, some studies conducted outside of the US did find a positive relationship between cognitive restraint and HP food addiction in young adult samples (Aloi et al., 2017; Brunault et al., 2017; Khine et al., 2019), suggesting that there may be cultural differences. However, it is important to note that these studies were conducted on samples of college students where body image concerns may still be developmentally relevant (Maezono et al., 2019). It will be important for future research to focus on how the correlates of HP food addiction vary cross-culturally and based on the social pressures associated with different developmental stages.

One potential barrier to examining food addiction in adolescence may be related to our current clinical conceptualization of substance use disorder. When the DSM-5 was released, symptoms of addiction became more problem-focused, with approximately half of the symptoms relating to the consequences of use (e.g., interpersonal problems, failure to fulfil obligations; American Psychiatric Association, 2013). Compared to adults, addictive-eating in adolescence may not have resulted in significant consequences, which could emerge later on in the progression of the disorder (Piontek, Kraus, Legleye, & Bühringer, 2011). Supporting this hypothesis, one study found that adolescents endorsed symptoms such as craving, loss of control, and the inability to cut down on HP foods, while they did not endorse symptoms such as interpersonal problems, use in hazardous situations, and giving up activities (Schiestl & Gearhardt, 2018). When a dimensional rather than a categorical approach to scoring was

employed with this sample, food addiction was positively associated with emotional eating, eating to cope, and BMI z-score even when symptom thresholds were not met (Schiestl & Gearhardt, 2018). This suggests that it may be necessary to conceptualize and approach the measurement of HP food addiction in adolescents differently than adults, emphasizing the importance of more sensitive scoring procedures. In light of these results, it will be important for future researchers to determine how the nature of HP food addiction differs between children, adolescents, and adults.

The presence of HP food addiction symptoms during adolescence may also have unique consequences for the development of substance-related behaviors. Indeed, during adolescence, many youth have their first experiences with substances like tobacco, cigarettes, or alcohol (Steinberg, 2008). One possible consequence of HP food addiction earlier in development is cross-sensitization, or the phenomenon where sensitization to one drug causes an increased drug-seeking response to another type of drug (Avena & Hoebel, 2003). In a landmark study, Avena and Hoebel (2003) found that rats demonstrating signs of sugar dependence showed an increased drug-seeking response to amphetamine through cross-sensitization relative to rats that only ate chow. These findings suggest that the presence of HP food addiction symptoms during adolescence could potentially increase vulnerability to the rewarding effects of substances when adolescents first try them.

Another possible consequence of HP food addiction in this age group is that adolescents may become motivated to use substances as a way to reduce HP food intake. For example, adolescents who have concerns about their body weight and engage in dieting behaviors are more likely to smoke tobacco cigarettes (Potter, Pederson, Chan, Aubut, & Koval, 2004). A small group of observational studies also shows that younger adolescents who gain more weight

are more likely to smoke cigarettes later in adolescence and early adulthood (Gearhardt, Waller, Jester, Hyde, & Zucker, 2018; Huang, Lanza, Wright-Volel, & Anglin, 2013; Lanza, Grella, & Chung, 2015).

However, a growing body of research shows negative associations between greater body mass and other substance use. For instance, individuals with chronically-obese versus non-obese growth trajectories from age 6 to 18 were less likely to initiate alcohol use at age 14 or to binge drink across adolescence (Huang et al., 2013). Furthermore, adolescents with obese versus non-obese growth trajectories from age 8 to 21 had fewer drinking and illicit drug problems in early adulthood (Gearhardt et al., 2018). Results of these studies are in accordance with the food-drug competition hypothesis, or that greater HP food intake might "compete" with substance use via adaptations in shared neural-reward pathways (Gearhardt & Corbin, 2009). Indeed, one study that specifically examined associations between HP food intake and alcohol use across adolescence found that adolescent women who ate more high-fat and -sugar foods from age 16 to 19 were less likely to drink alcohol at these ages (Cummings, Ray, & Tomiyama, 2017). In sum, the emergence of HP food addiction symptoms during adolescence could potentially increase or decrease risk for greater substance-related behaviors. More research is needed to identify under what conditions, for whom, and why these effects may emerge.

Parenting Factors Relevant to HP Food Addiction from Infancy through Adolescence

Parents play an important role in their children's eating patterns, which may have implications for the development of HP food addiction. In substance use disorder research, three key mechanisms for intergenerational transmission of the disorder have been identified: a direct genetic route (i.e., a child inherits vulnerabilities that increase the likelihood of substance use), an epigenetic route (i.e., environmental factors like *in utero* substance use increase a child's risk

for later use), and a social learning route (i.e., the child learns the behavior by observing and imitating parents) (Elam, Sternberg, Waddell, Blake, & Chassin, 2020; Pasqualini, Pieroni, & Tomassini, 2019). The HP food addiction perspective posits that genetic and epigenetic mechanisms of HP food addiction may broadly mirror those seen in substance use disorder (Davis & Bonder, 2019). However, social learning mechanisms may be especially relevant to HP food addiction because, unlike with substance use, parents play a direct role in feeding their children and teaching them what, how, and when to eat (Yee, Lwin, & Ho, 2017).

Most recent research on infant feeding has focused on the role of maternal sensitivity to infants' hunger and satiety cues (Hetherington, 2020). Per this line of research, mothers risk overfeeding their infants if they feed according to their own ideas about when and how much is appropriate (Hetherington, 2020). The proposed solution is responsive feeding, in which mothers feed only in response to their infants' hunger and satiety cues (Hetherington, 2020). Some responsive feeding interventions have been found to aid in preventing rapid weight gain in infants (Daniels et al., 2012; Savage, Birch, Marini, Anzman-Frasca, & Paul, 2016). However, these approaches may place undue blame on mothers for their children's weight by neglecting the fact that both the children and the parents have unique characteristics that dynamically interact. Recent research found that infants' reward responsiveness to sucrose in the first weeks of life for predicted weight outcomes at 9 and 18 months (Lumeng et al., 2020). This may reflect innate tendencies for some infants to seek more food than others, which poses an important limitation to feeding interventions that solely address parent responsiveness. Further research illuminating how parent and child factors interact will allow for the development of more compassionate feeding guidance for parents of infants with a higher reward drive for food.

Responsive feeding has also been suggested for children as their diets expand from mostly milk or formula to a range of solid foods (Black & Hurley, 2017). Parents are advised to decide which foods are available to their children and when, but to validate children's feelings and allow their children to decide how much to eat (Black & Hurley, 2017). Responsive feeding interventions are generally accepted by parents of preschoolers (Ledoux, Robinson, Baranowski, & O'Connor, 2018). However, a recent randomized controlled trial found that a responsive feeding intervention did not produce significant differences in obesity prevalence during children's first two years of life (Morandi et al., 2019). The effectiveness of responsive feeding strategies for mitigating children's risk for HP food addiction has not yet been explored.

To our knowledge, only one study to date has directly examined the relationship between parental feeding practices and HP food addiction (Burrows et al., 2017). This study found that parents who engaged in more restriction and pressure to eat had children with more HP food addiction symptoms (Burrows et al., 2017). These findings are consistent with previous research demonstrating that restriction was associated with eating in the absence of hunger (EAH) in 4-6 year old girls (Birch, Fisher, & Davison, 2003) and increased adiposity (Spruijt-Metz, Lindquist, Birch, Fisher, & Goran, 2002). Although this preliminary research highlights a potentially important relationship between parent feeding practices and child eating behavior, it does not warrant the conclusion that parents are causing negative eating patterns in their children. In fact, a longitudinal study found that parental restriction did not predict EAH at future age points (Bauer et al., 2017). Thus, it remains unclear whether feeding practices like restriction and pressure to eat cause children to develop addictive relationship with HP foods or if parents are using restraint as an attempt to modulate the behavior of children that begin to display HP food

addiction. Future research that accounts for dynamic, dyadic relationships between parents and children is necessary to make inferences about the direction of effects.

Research on childhood HP food addiction also highlights a positive association between parent and child HP food addiction symptoms (Burrows et al., 2017). This pattern mirrors substance use disorder research, which shows that children are more likely to develop a substance use disorder if they have a parent with the condition (Merikangas et al., 1998). Parent HP food addiction symptoms may be particularly important for child diet change, as most pediatric weight loss trials emphasize changing the diet of the entire family (Chai et al., 2019.) If parents and children are simultaneously experiencing symptoms like loss of control and tolerance, this may contribute to dynamics that undermine diet change attempts. For example, parents experiencing HP food addiction symptoms may be more likely to keep HP foods in the home, thus providing more HP food cues for both parents and children. This counters the advice of family-based weight management programs, which emphasize reducing environmental cues for HP foods and increasing environmental cues for healthier foods like fruits and vegetables (Wilfley, Hayes, Balantekin, Van Buren, & Epstein, 2018).

Likewise, there is a positive association between parents' experiences of HP food addiction and children's experiences of HP food withdrawal (Parnarouskis et al., 2020). Parents who reported more withdrawal symptoms in their children also reported less success at changing their children's diets (Parnarouskis et al., 2020). HP food withdrawal thus may be an overlooked barrier to family-wide diet change, as parents and children may be experiencing withdrawal or other symptoms of HP food addiction at the same time. It will be important for family-based weight management programs to take this into account and address these symptoms in both parents and children to potentially decrease attrition rates and improve outcomes for families.

During adolescence, it becomes developmentally appropriate for teens to seek independence from their parents with regard to food choices (Kell, 2008) but continued parental modeling of healthy eating may still be beneficial (Fleary & Ettienne, 2019). Teens and parents negotiate autonomy around food in complex ways, which may have implications for HP food addiction (Bassett, Chapman, & Beagan, 2008). For example, both parent concerns about their children's weight and adolescents' food reward responsiveness were associated with adolescent stress eating of HP foods (Smith et al., 2020). Given that adolescents are at increased risk for addictive behaviors (Gray & Squeglia, 2018), more research is needed to probe dyadic interactions between teens and parents and to develop scientifically-informed methods for parents to effectively guide their children as they transition to young adulthood.

HP Food Addiction in Early and Middle Adulthood

The majority of the research on HP food addiction has been conducted in early and middle adulthood. Systematic reviews have been published that provide an in-depth view of this literature (Meule & Gearhardt, 2019; Pursey, Stanwell, Gearhardt, Collins, & Burrows, 2014). Given these prior reviews, we will briefly review key findings related to HP food addiction in adulthood. The prevalence of YFAS food addiction in community samples within the US is 15% (Schulte & Gearhardt, 2018) with higher rates found in clinical samples (i.e., 32% in patients seeking bariatric weight loss surgery (Pepino, Stein, Eagon, & Klein, 2014) and 57 % in individuals with obesity and binge eating, (Gearhardt et al., 2012)). Higher HP food addiction is associated with higher BMI, waist-to-height ratio, binge eating episodes, and binge-type eating disorders (e.g., binge eating disorder, bulimia nervosa; Borisenkov et al., 2020; Meule & Gearhardt, 2019). The foods most strongly associated with food addiction are HP foods, particularly those with a high glycemic index (i.e., cause rapid rises in blood glucose; Lennerz &

Lennerz, 2018; Schulte, Smeal, & Gearhardt, 2017). Furthermore, recent research suggests that hyperinsulinemia in response to high-glycemic food intake may be responsible for driving neurochemical changes associated with HP food addiction (Caravaggio et al., 2015; Caravaggio et al., 2018; Dunn et al., 2012). However, more research into the direct mechanisms between high-glycemic food intake and HP food addiction is needed.

HP food addiction during early and middle adulthood is also associated with heightened cravings (particularly for HP foods) and increased motivations to eat HP foods in response to negative affect (Joyner, Schulte, Wilt, & Gearhardt, 2015; Meule & Kübler, 2012). Inhibitory control difficulties, especially negative urgency and emotion dysregulation, are associated with food addiction in adults (Borisenkov et al., 2020; Maxwell, Gardiner, & Loxton, 2020; Murphy & MacKillop, 2019; Murphy, Stojek, & MacKillop, 2014). Additionally, HP food addiction is associated with negative health outcomes, including hypercholesterolemia (Flint et al., 2014), and poorer psychological functioning (Chao et al., 2017). Although some studies have not found HP food addiction to predict weight loss treatment outcomes (Chao et al., 2019), a recent large scale study with over 600 patients found that HP food addiction was the biggest psychosocial predictor of poor weight loss treatment outcomes (Fielding-Singh, Patel, King, & Gardner, 2019). Thus, HP food addiction in adults likely has important clinical utility.

HP Food Addiction in Older Adulthood

Although research on HP food addiction in infancy, childhood, adolescence, and middle adulthood is growing, few studies focus on HP food addiction in older adulthood (i.e., adults ages 65 and older (Administration on Aging, 2020)). Previous generations of older adults demonstrated decreasing rates of problematic substance use across the lifespan (Schulte & Hser, 2013). As such, older adulthood is often viewed as a low risk period. However, recent research

shows higher rates of illicit drug and substance use among older adults today relative to previous generations (Yarnell, Li, Mac Grory, Trevisan, & Kirwin, 2019). Furthermore, as baby boomers age into older adulthood, the number of older adults requiring substance use disorder treatment is expected to rise to 4.4 million in 2020 (Gfroerer, Penne, Pemberton, & Folsom, 2003). Substance use in older adulthood is highly correlated with medical complications such as hypertension and liver disease relative to younger substance users (Yarnell et al., 2019). Additionally, due to age-related neurobiological changes and the potential for negative interactions with prescribed medications, drugs or alcohol can potentially exacerbate underlying physical and mental health conditions (Moore, Whiteman, & Ward, 2007). As such, substance use in older adulthood particularly risky.

In light of these findings, the examination of HP food addiction in older adulthood is critically important. To date, no study has examined the prevalence or characteristics of HP food addiction in older adulthood. In line with early research on substance use, one study did find significantly lower endorsement of HP food addiction in a group of women aged 62-88 (2.7%) relative to women 45-64 (8.4%; Flint et al., 2014). However, if trajectories of HP food addiction follow patterns of substance use, rates of HP food addiction in older adulthood may increase. Due to dramatic shifts in the food environment, including increased access to HP foods in the 1970s and 80s (Bentley et al., 2020), cohort effects for HP food addiction may be especially dramatic. Because HP foods were uncommon when current older generations were young, they may have been protected from the development of HP food addiction. However, as younger generations who were exposed to HP foods in childhood age into older adulthood, risk will likely increase as early exposure to addictive substances increases the risk of substance use disorder later in life (Hingson et al., 2006).

In light of these considerations, the examination of HP food addiction in older adulthood is an important future direction. Researchers should examine the prevalence of HP food addiction in older adulthood, with a specific focus on cohort effects due to differences in childhood food environments. Additionally, researchers should determine if neurobiological and cognitive changes that occur in older adulthood further increase the risk the HP food addiction similar to other substances.

Conclusions and Future Directions

The changing modern food environment has influenced individuals across all developmental stages. HP foods are now cheap, accessible, and heavily marketed, leading to their domination of the food supply. This review highlights how HP foods may be implicated in changes in reward functioning and the development of HP food addiction across the lifespan. A number of developmentally focused questions about HP food addiction require further study. Longitudinal studies that investigate the consequences of HP food exposure and HP food addiction symptoms across the lifespan will be extremely important for advancing our scientific understanding. It will also be important to study cohort effects, as the degree of exposure to HP foods across vulnerable periods of the lifespan (e.g., *in utero*, early childhood, adolescence) has increased drastically in the last 40 years. A greater scientific understanding of HP food addiction symptoms (and related mechanisms like reward dysfunction) across the lifespan will be essential for the creation of effective prevention, treatment, and policy interventions that target critical developmental periods. For example, interventions that aim to improve the diet quality of pregnant mothers with HP food addiction may reduce the risk of inter-generational transmission. Policies that reduce aggressive HP food marketing to adolescents may reduce triggers to overeat. Moreover, to reduce excessive intake of HP foods in older adults with diet-related disease,

therapeutic approaches that target addictive mechanisms (e.g., urge surfing or naltrexone/buproprin) may be needed to improve diet quality. In sum, deepening our knowledge about HP food addiction across the lifespan may lead to clinical advancement and improved health outcomes.

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]AUTHOR DECLARATION AND ETHICS STATEMENT

We the undersigned declare that this manuscript is original, has not been published before and is not currently being considered for publication elsewhere.

We wish to confirm that there are no known conflicts of interest associated with this publication and there has been no significant financial support for this work that could have influenced its outcome. We confirm that the manuscript has been read and approved by all named authors and that there are no other persons who satisfied the criteria for authorship but are not listed. We further confirm that the order of authors listed in the manuscript has been approved by all of us. We confirm that we have given due consideration to the protection of intellectual property associated with this work and that there are no impediments to publication, including the timing of publication, with respect to intellectual property. In so doing we confirm that we have followed the regulations of our institutions concerning intellectual property.

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Authors:
Emma T. Schiestl
Julia M. Rios
Lindsey Parnarouskis
Jenna R. Cummings
Ashley N. Gearhardt

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Highlights

- Highly processed (HP) foods are associated with changes in reward functioning and the development of addictive-like eating behaviors.
- HP food addiction is associated with negative outcomes (e.g., elevated BMI, increased binge eating) at every life stage.
- The study of food addiction across the lifespan has important implications for treatment and the adaptation of public policy.