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Missing The Forest For The Trees: The Causes Of The Obesity Epidemic

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Abstract

Arguably the most pressing public health issue in the United States today is the obesity epidemic, which has affected children, adolescents, and adults alike. A lack of longitudinal research on distinct factors related to obesity in different groups has made addressing the epidemic difficult. Many lifestyle and environmental factors, including nutrition, sleep, stress, the gut microbiome, screen time, and marketing, are all related to obesity, but many of these factors are also related to each other. The current study investigates these systemic relationships, presents a hypothesis on the complementary causes of obesity, and contributes to contemporary longitudinal research by investigating obesity rates at different levels of daily television and video watching time in the United States. Analysis of long-term obesity trends combined with a systems thinking approach is needed to inform population-level interventions and policy decisions aimed at combatting the obesity epidemic.

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Table of Contents

Introduction 6

What Is Obesity? 6

Obesity Risk Factors 6

The Obesity Epidemic 8

Potential Causes of the Obesity Epidemic 10

Sleep 11

Environmental Contamination 13

Nutrition and Food Environments 16

Gut Microbiome 20

Stress 23

Screen Time 24

Marketing 26

Solutions 31

Current Insights 34

Methods 35

Results 37

Discussion 38

Tables and Figures 42

References 46

Appendix 55

List of Tables

Table 1. Frequency of individuals in each TV time group, 2003-2015.

Table 2. Results of individual regression analyses of average change in BMI over time among different TV time groups, 2003-2015.

Table 3. Results of multiple regression using indicator variables to evaluate the effect of TV time groups on estimated change in BMI over time, 2003-2015.

Table 4. Results of analysis comparing regression estimates between groups using interaction terms (year*TV time group), 2003-2015.

Appendix 1. TV time by race and ethnicity group, 2003-2015.

Appendix 2. TV time by income group, 2003-2015.

Appendix 3. TV time by education group, 2003-2015.

List of Figures

Figure 1. Systems thinking hypothesis of obesity epidemic.

Figure 2. Mean BMI by daily hours of TV and video time, biannually 2003-2015.

Figure 3. Linear regression by TV and video time group, 2003-2015.

Introduction

What Is Obesity?

Obesity is excessive or higher-than-normal accumulation of adipose tissue or fat in the body (Panuganti 2022). Body mass index (BMI) is a common benchmark system used to estimate obesity and is calculated by dividing an individual's weight in kilograms by their height in meters squared. Once a BMI estimate is obtained for an individual, it can be sorted into categories. The four commonly used categories include "underweight," "normal weight," "overweight," and "obese," although these categories differ between race and ethnicity groups (Weir 2022). For non-Hispanic White, Black, and Hispanic individuals, an overweight BMI is a BMI of at least 25, and an obese BMI is at least 30 (WHO 2000). For Asian and South Asian individuals, these numbers are 23 and 25, respectively, since the categories for other groups do not accurately reflect obesity in these populations (WHO 2000; Weir 2022). BMI is a good measure of obesity for populations but can be inaccurate in individuals, since muscle mass and true excess adiposity may not be reflected by weight alone in all cases. In pediatric populations, BMI is compared as a percentile in comparison with others of the same sex and age to determine whether an individual is underweight, overweight, or obese (Weir 2022).

Obesity Risk Factors

Several studies in the past 20 years have analyzed the differences in various socioeconomic groups at different time points and found that generally, obesity prevalence has increased overall and that prevalence at any given point is higher in groups with lower SES, with some variability in different race and ethnicity groups (Hinkle 2012; Kelley 2016; Ogden 2017; Heo 2018; Min 2021). Among race and ethnicity groups, obesity is most prevalent in non-Hispanic Black adults, then Mexican-Americans, compared to non-Hispanic White Americans. Asian adults generally

have the lowest rates of obesity (Yang 2015; Fruh 2017; Wang 2017). Cross-sectional studies reveal some inequities by race and ethnicity in terms of BMI, waist circumference, and other health indicators (Krieger 2014). These inequities are apparent at any given time point, but they do not reveal information about the obesity epidemic, which is characterized by a rapid rate of change in obesity prevalence over time (Zimmerman 2011; Banas 2021). Cross-sectional studies highlight race and ethnicity differences in obesity prevalence, but prevalence has apparently changed at a similar rate over time: all races/ethnicity groups are experiencing an increase in obesity prevalence (Zimmerman 2011; Krieger 2013; Hales 2020; Banas 2021). In other words, there are disparities in obesity rates at any given time, but there are apparently few disparities in the *rate of change* in obesity prevalence over the last 60 years (Banas 2021).

There are several other risk factors for obesity, but these factors apparently differ by race and ethnicity. For example, physical activity level, smoking status, and gender appear to be more important for obesity in non-Hispanic White and Black adults compared to Hispanic adults (Wang 2017). In the United States, people with low food security are more likely to be overweight or obese, even when controlling for age, other poverty factors, education, marital status, and race/ethnicity (Ryan-Ibarra 2017). Geographical location may also play a part: four U.S. states (Alabama, West Virginia, Louisiana, and Mississippi) had the highest obesity rates in the U.S. at over 35% in 2015 (Fruh 2017). A 2022 analysis using data from 2013-2017 that compared obesity rates by time spent living in the United States found that in every race and ethnicity group, a longer time in the U.S. meant a higher prevalence of obesity (Cofie 2022). Women who immigrated to the U.S. and stayed at least ten years and had very low food security had a higher prevalence of overweight and obesity than women who did not have food insecurity or who lived in the U.S. less than ten years (Ryan-Ibarra 2017). People who are obese as children

and adolescents are about five times more likely to be obese in adulthood compared to people who were not obese during youth. 55% of children who are obese are also obese during adolescence. 80% of people who are obese in adolescence are obese in adulthood. That being said, 70% of adults with obesity did not have obesity during youth (Simmonds 2016).

The Obesity Epidemic

About 1.3 million people worldwide struggle with excess adiposity (Global BMI Mortality Collaboration 2016). In the United States, estimates of adult obesity prevalence in recent years have ranged from about 33-42%, including all adult age groups and both sexes (Ogden 2014; Yang 2015; Flegal 2016; Fruh 2017; Hales 2020). From 1975 to 2016, mean BMI and obesity prevalence increased in children and adolescents worldwide (NCD Risk Factor Collaboration 2017). There have also been significant increases in obesity in men, women, and children in the U.S. since the 1980s, but not much is known about the interlocking causes of overall obesity trends (Inoue 2018; Harris 2009). More specifically, based on NHANES data, the mean body mass index (BMI) in the top income group increased from about 24.5 in 1960 to about 28.5 in 2007; the mean in the lowest income group increased from about 25.2 in 1960 to about 29.4 in 2007 (Zimmerman 2011). The difference between years is apparently much larger than between income groups. In fact, year of birth involves the largest disparities in obesity in the United States, larger than race/ethnicity, socioeconomic status, gender, age, or location (Zimmerman 2011). Another study measured body height, weight, and BMI in US-born white and black non-Hispanic adults aged 20-44 from 1959-1962, finding that the shift toward high rates of change (rapid increases in weight) started in the late 1980s for both white and black populations. Rapid increases in BMI started in the late 1990s for both groups, particularly for middle income quintiles (Krieger 2013). This is one of very few longitudinal studies on obesity trends from

before 1999, but it does not include Hispanic or older adults (Krieger 2013). Other longitudinal studies in the published literature begin earlier and focus only on specific groups (Wang 2023).

Obesity also affects children; in 2011-2012, 8.1% of infants and toddlers had high weight compared to length, and 16.9% of two to 19-year-olds were obese (Ogden 2014). High overweight and obesity prevalence is spreading to younger and younger ages. People who were born from 1956 to 1965 had an obesity prevalence of 20% by age 30-39 years old. Concerningly, people who were born later, between 1966 and 1985, had an obesity prevalence of 20% by 20-29 years old (Lee 2010; Fruh 2017). Among different birth cohorts from 1930 to 1993, the prevalence of child obesity increased from 0-14% in boys and 2-12% in girls. However, this was not a completely linear trend: the severe increase in obesity prevalence in boys did not begin until after the birth year 1970 and until after 1980 for girls. However, overweight prevalence was increasing in birth cohorts from the 1930s and 1940s (von Hippel 2013). The child obesity epidemic is recent, and it became severe very fast and very suddenly (Harris 2009; von Hippel 2013). Children are developing obesity during elementary school years. Kindergarten children who did not have obesity in 2010 had a 4.5% increase in relative obesity by the end of fifth grade compared to kindergarten children in 1998 (Cunningham 2022). Causes need to be identified, since genetics alone cannot explain these severe population-level changes (Harris 2009). A 2016 systematic review on childhood obesity incidence found that only three of nineteen studies included nationally representative U.S. data. Studies of this nature often use “convenience data” that was not intended for obesity studies (meaning sub-optimal measurement of obesity), and most are not nationally-representative. BMI categories in children based on percentage are potentially but not proven to be useful for measuring health risks. Longitudinal studies using

nationally- representative data with accurate BMI or other obesity measurements are still needed (Cheung 2016).

Potential Causes of the Obesity Epidemic

There are two subtypes of obesity: "acquired" and "inherited." Acquired obesity is broadly based on behavioral patterns. The cause of this type of obesity is not completely clear. However, there is some evidence to suggest that it stems partially from a lack of physical activity, an increase in sedentary behavior, and an increase in caloric intake (Archer 2022). This type of obesity may also be related to psychological or social disorder (Rubino 2020, Archer 2022). Acquired obesity can be addressed and prevented in individuals with an increase in overall physical activity level and considerations to moderate caloric intake (Archer 2022, Mozaffarian 2022). Inherited obesity is apparently not caused by an individual's behavior alone but involves an underlying inherited condition, including genetics, epigenetics, or the gut microbiome. This type of obesity is more complicated and more difficult to treat and prevent than acquired obesity (Archer 2022). While we have seen an increase in obesity prevalence over the long term, it is difficult to measure the underlying physiological patterns of obesity subtypes (Zimmerman 2011; Banas 2021).

Fat is the body's energy store. Fat is regulated and protected by regulation of energy intake, which happens through hedonic and homeostatic pathways, and by energy expenditure through thermogenesis (Wright 2012; Maruvada 2016). Obesity is thought to be a simple imbalance of energy intake and energy expenditure, but these two systems are interconnected and regulated by complicated mechanisms that go beyond simply "energy in, energy out" (Maruvada 2016). "Energy in" and "energy out" are affected by more than just food intake and

physical activity. This system is complex and can be impacted by genetic, physiologic, social, economic, psychological, and environmental factors (Wright 2012).

As discussed, there is evidence from the NHANES survey data of an increase in BMI over time and also an increase in overall caloric intake (Brown 2015; Lind 2016; Banas 2021). However, it is worth noting that for each given amount of caloric intake or physical activity, the BMI that was predicted was much higher in 2006 compared to 1998. In other words, for each increase in calorie intake, obesity increased more than expected over time, so caloric intake alone cannot explain these trends (Brown 2015; Lind 2016).

There are many factors that likely contribute to obesity in adulthood: smoking, alcohol consumption, personality, diet, socioeconomic status, maternal BMI, marital status, education, stress, race/ethnicity, genetics, the gut microbiome and the brain-gut axis, physical activity level, social isolation, culture and social influences, marketing, gestational weight, childhood obesity, sleep, and others (Patel 2009; Zimmerman 2011; Safaei 2021). There is evidence for many hypotheses—all of them are systemic and challenge the individual-focused prevention and treatment options and are all interrelated. In the media, more news articles attribute obesity to individual choices—the public perception of obesity is framed around only individual agency rather than environmental-level attribution (Chiang 2020). The true cause is likely a combination of both individual and environmental factors.

Sleep

Where there has been a huge increase in obesity prevalence the Western world, there has also been a significant decline in duration of sleep (Patel 2009). Prospective epidemiological and laboratory studies have shown evidence that consistently low sleep duration is an independent predictor of weight gain and obesity incidence (Patel 2009; Beccuti 2011). In a systematic

review from 2008, all three longitudinal studies analyzing sleep loss and weight gain found that short sleep duration was positively associated with "future weight," especially in young age groups (Patel 2008). Cross-sectional studies measuring sleep habits in an objective way have shown an association between reduced sleep and obesity. Furthermore, these studies have shown that the association between sleep loss and obesity is not because of sleep apnea alone and that weight gain is specifically due to an increase in fat mass, not muscle mass (Patel 2009).

Circadian rhythms are important for metabolic function. They are involved in energy use and storage, fasting and feeding, sleeping and waking, all of which are part of energy homeostasis. People who experience a disruption in the circadian rhythm from changes in dietary intake, work patterns, and jet lag are at risk of developing obesity (Maruvada 2016). Sleep in itself is an important factor in glucose metabolism and neuroendocrine function. Sleep loss can lead to decreased insulin sensitivity, decreased glucose tolerance, increased appetite and hunger, and high levels of cortisol in the evenings (Beccuti 2011). There is some evidence that sleep could impact hormonal appetite regulation; sleep deprivation decreases activity in the brain regions that evaluate appetite and regulate food desirability (Patel 2009; Greer 2013). There is a specific brain mechanism where we can see insufficient sleep leading to the development and maintenance of obesity, which involves subcortical limbic system sensitivity and a high propensity towards foods that would likely lead to weight gain (Greer 2013). Observational evidence shows that during periods of sleep deprivation, people experience an increase in desire for fatty, high-calorie foods that can promote weight gain (Greer 2013; St-Onge 2016). This desire comes after sleep loss and can be predicted by the severity of sleep deprivation (Greer 2013).

On the other hand, obesity is considered a main contributor to obstructive sleep apnea (OSA), which can detrimentally affect sleep duration and quality (Jehan 2017). A four-year longitudinal study showed that a change in weight is directly proportionate to "sleep disordered breathing," where people with more weight gain have more severe sleep apnea (Peppard 2000). OSA risk increases with age, BMI, and an increase in sedentary lifestyle (Jehan 2017). Furthermore, an estimated 3-7% of men and 2-5% of women have undiagnosed OSA. Up to 82% of men and 93% of women that actually have OSA could be undiagnosed (Beydoun 2016). Obesity can lead to sleep disturbances, and in turn, sleep disturbances can also contribute to obesity in a cyclical manner (Muscogiuri 2019).

Environmental Contamination

Among developing countries, obesity is more prevalent among women. In the United States, however, obesity rates for men and women are not as dramatically different (Grantham 2014; Yang 2015; Flegal 2016; Fruh 2017). Since weight gain varies by sex, hormone and metabolic factors are apparently important contributors to weight gain in addition to energy consumption (Wright 2012; Grantham 2014). As living conditions improve, male and female obesity rates become more similar; there is clear positive, statistically significant linear correlation between national gross domestic product (GDP) and male-female obesity ratio (Grantham 2014).

Many medications that are commonly prescribed and used today are associated with weight gain. These include antihistamines, protease inhibitors, steroid hormones, contraceptives, diabetic treatments, and psychotropic medications (Wright 2012). Estrogen exposure is known to cause weight gain through thyroid inhibition and modulation of the hypothalamus (Wright 2012; Grantham 2014). Xenoestrogens are molecules that are so close to the true estrogen molecule that they can bind to estrogen receptors in the body. These molecules can be found in soy

products and may contribute to obesity; the United States is a prolific consumer of soy products (Grantham 2014). Polyvinyl chloride (PVC) is another source of xenoestrogen, and plastic use also correlates with GDP (Grantham 2014).

There are several other hypotheses for chemical and hormonal influences on obesity. Prenatal exposure to several different endocrine disrupting chemicals (including perfluoroalkyl substances, or PFAS) is associated with risk of excess adiposity and obesity later in life. PFAS were associated with increased birth weight, which can be predictive of obesity later in life (Braun 2017). In animal models, those that are exposed to various chemicals that are common environmental contaminants, including bisphenol A (BPA), phthalates, dichlorodiphenyl-trichloroethane (DDT), dioxins, tributyltin (TBT), and perfluorooctanoic acid (PFOA), during gestation more frequently have offspring with obesity due to increased fat accumulation. Prenatal exposure to a metabolite of dichlorodiphenyltrichloroethane (DDT) called dichlorodiphenyldichloroethylene (DDE), as well as high maternal levels of DDE in the blood during the prenatal period, is associated with severe and quick weight gain in the children and obesity when those children become adults (Lind 2016).

More specifically, BPA has been the subject of increased epidemiological research due to several existing health concerns. Urinary BPA, in addition to bisphenol S (BPS) and bisphenol F (BPF), common replacements for BPA, are all associated with general and abdominal obesity in children and adolescents, even after adjusting for demographic, socioeconomic, and lifestyle, among other factors (Trasande 2012; Bhandari 2013; Liu 2019). Children with the highest quartile of urinary BPA had about 2.5 times the odds of obesity compared to the lowest quartile (Bhandari 2013). Higher BPA exposure in US adults is associated with higher rates of central and abdominal obesity, based on NHANES data from 2003-2006 (Carwile 2011).

The mechanisms behind the connection between BPA and obesity are somewhat known. After chronic BPA exposure, neuropeptide Y (NPY) and agouti related peptide (AgRP) levels rise. A rise in NPY levels leads to a boost in hunger; AgRP is related to appetite regulation. When NPY and AgRP levels increase, obesity can develop (Naomi 2022). BPA can also change the normal endocrine system and metabolic pathway specifically in adipose tissue, increasing obesity risk (Carwile 2011; Naomi 2022). BPA exposure can lead to enhanced adipocyte differentiation and lipid accumulation, chronic low-grade inflammation and altered lipid homeostasis, and neurological disruption that affects food-seeking behavior, all of which can contribute to obesity (Naomi 2022). Furthermore, BPA level may be associated with fewer sleep hours based on the NHANES survey, which may also contribute to obesity development (Beydoun 2016; Muscogiuri 2019).

People in the U.S. are heavily exposed to BPA through the environment; BPA can leach into food and drink from plastic containers—92.6% of 2003-2004 NHANES participants had detectable levels of BPA in their urine, a measurement that was consistent with previous studies (Rubin 2011). A 2015 study showed some evidence of decreasing BPA exposure over time following action taken by the U.S. FDA, but trends in BPA exposure prior to 2003 are still somewhat unclear, especially because determining actual BPA exposure is difficult (LaKind 2015). Even if BPA exposure prevalence has decreased in recent years, BPA can lead to obesity through multiple generations, since BPA exposure can be inherited through both male and female germlines and affect offspring (Naomi 2022). The CDC regularly reports exposures of environmental chemicals based on NHANES data, including BPA (https://www.cdc.gov/exposurereport/).

Nutrition and Food Environments

Consumption of processed foods containing sugar, white flour, and vegetable oil and ready-to-eat meals increased from less than 5% of foods consumed in 1800 to more than 60% of foods in 2019, a dramatic increase that very much resembles the severe rise in obesity; in 2016, at least half of American youth consumed a poor-quality diet (Liu 2020; Lee 2022). The food supply of high-income countries (like the U.S.) is overwhelmingly full of ultra-processed food products (Monteiro 2013). The displacement of minimally processed foods with highly processed, packaged, convenient food products has been dubbed the "nutrition transition," characterized by increases in consumption of hyperpalatable foods containing refined grains, added sugar, and animal fat and protein (Malik 2013; Scrinis 2015; Monteiro 2019). Food preferences are malleable—many foods, such as insects, beef, and pork, are highly sought-after in some cultures and disliked in others, indicating some level of social and/or cultural influence on food preferences (Harris 2009; De Dominicis 2020). There has been insufficient research on how to encourage healthy dietary habits, particularly in a culturally appropriate way (De Dominicis 2020).

Ultra-processing develops hyperpalatable, inexpensive, ready to eat, attractive food products that by their nature are sugary or salty, fatty, and energy dense (Monteiro 2013). "Processing" alone is not necessarily bad and can even promote food safety and reduction of food waste, but some types of processing, like hydrogenation and adding sugars to drinks, have demonstrably harmful effects (Monteiro 2013; Monteiro 2019). Ultra-processed foods are convenient in that they are imperishable and ready to consume, they are attractive to customers in that they are hyperpalatable, and they are also very profitable for manufacturers due to

inexpensive ingredients and a long shelf life (Monteiro 2019). Ultra-processed foods are also highly caloric: the average energy density of food in the Canadian diet was 3.2 kcal/g in the highest quintile of ultra-processed food intake compared to 1.9kcal/g in the lowest quintile (Monteiro 2019). A randomized controlled trial from the NIH showed that when people are exposed to an *ad libitum* ultra-processed diet, meaning that 81.3% of total energy came from those ultra-processed foods, energy intake was 508 kcal more per day on average compared to *ad libitum* diets that did not have ultra-processed food. Both types of available diet were matched for total energy, energy density, fiber, macronutrients, sugar, and sodium. Despite this, the ultra-processed group gained about two pounds in two weeks, whereas the group without ultra-processed food lost about two pounds over the same time frame (Monteiro 2019).

Large food companies specifically develop foods that induce cravings, many of which have high amounts of sugar, fat, and salt (Akerlof 2016; Monteiro 2019; Wood 2021). People often report strong food cravings, emotional eating, binge eating, and struggling to regulate consumption of high-calorie foods, even when they know the consequences (Gearhardt 2011). A study on nurses was able to attribute a 3.35-pound weight gain over time to potato chips (1.69 pounds), French fries (1.28 pounds), and sugar-sweetened beverages (one pound) (Akerlof 2016). Foods and recreational drugs likely induce similar behaviors and motivations, like cravings, diminished control over consumption, and continuation of use despite known and experienced adverse effects (Gearhardt 2011).

Food intake is regulated by two complementary systems. The homeostatic pathway involves energy stores and increases desire to eat based solely on energy need. The hedonic pathway involves motivation to eat foods that are “highly palatable.” In other words, the homeostatic pathway is geared toward energy balance, and the hedonic pathway is oriented

around palatability and pleasure. Even when the body has sufficient energy stores, the hedonic pathway can override the homeostatic pathway (Lutter 2009). Chronic exposure to highly palatable foods (generally, foods high in sugar and salt) can impact neuronal reward pathways. There is a good deal of evidence that consumption of highly palatable foods revolves around the same pathway as many recreational drugs (the mesolimbic dopamine pathway); limbic domains demonstrate similar neurological changes (neuroadaptations) after exposure to both food and drug rewards. These changes also shift the motivation to access both kinds of reward (Lutter 2009). Many recreational drugs work on this pathway, and "natural rewards" (foods) do, as well—there are analogous food and behavioral effects between hyperpalatable foods and addictive drugs (Lutter 2009; Gearhardt 2011).

Activation of the mesolimbic dopamine pathway when even looking at a picture of a highly palatable food is different in obese and normal-weight women, indicating an altered neurological perception of a "food reward" and an unusually high motivation to consume highly palatable foods. In other words, people with obesity have demonstrably higher dopamine responses to high-salt/high-fat/high-sugar foods (Lutter 2009). In human beings, diminished availability of the striatal dopamine receptor, which mediates the relationship between motivational and cognitive control, and dysfunction of the striatum, which is involved the partnership of movement and reward, are both associated with obesity and future weight gain (Gearhardt 2011).

In addition to being potentially addictive, highly caloric and fatty foods are inexpensive and accessible, highly palatable, and available in large quantities (Lutter 2009; Wright 2012). The cost of a healthy diet is a major concern for individuals in the United States. There is a positive association between energy-adjusted diet cost and HEI-2010 (diet quality) scores. This

association is stronger in women. Low diet costs are also associated with lower consumption of vegetables, fruits, seafood, and whole grains, as well as higher consumption of added sugars, alcohol, refined grains, and solid fat, based on the 2007-2010 NHANES cycles (Rehm 2015). Diet quality is higher when people eat at home compared to when they eat outside of home (i.e. at restaurants). Furthermore, higher diet quality is associated with a higher cost of food, both at home and away from home (Conrad 2021).

Diet can also impact sleep quality (St-Onge 2016). A study on female Japanese workers responding to lifestyle questionnaires found that a high intake of sweets and noodles was positively associated with poor sleep quality. A high intake of fish and vegetables was associated with good sleep. There was a positive, statistically significant trend towards poorer sleep quality with an increase in carbohydrate intake (Katagiri 2014; St-Onge 2016). Specific foods, like milk, vegetables, fruit, and fish (generally seen as "healthy" foods) have been shown to have some sleep-promoting effects (St-Onge 2016).

Artificial sweeteners, or low-calorie sweeteners (LCS) increasing in the American diet are another concern. Based on NHANES data from 1999-2008, consumption of low-calorie beverages with artificial sweeteners increased in both children (from 6.1-12.5%) and adults (from 18.7-24.1%) in the U.S. Low-calorie sweetener consumption overall increased from 8.7% to 15% in children 26.9% to 32.0% in adults during the same time period. These increases were observed regardless of weight, socioeconomic status, and race/ethnicity in both children and adults but were also more pronounced in women than men (Sylvetsky 2012; Sylvetsky 2016). While artificial sweeteners are likely well-intentioned and marketed as a healthy alternative to sugar, artificial sweeteners are associated with increased caloric consumption and with weight gain (Pearlman 2017). Artificial sweeteners have negative effects on neurohormonal regulation

of satiety, energy regulation, and body weight, and they can shift glucose homeostasis (Pearlman 2017; Christofides 2021). Changes to the gut microbiome due to LCS seem to be an important reason behind the change in glucose metabolism and overall nutrient absorption in the gut (Pearlman 2017). People who use LCS also have changes in the gut microbiome that predispose them to increased gut inflammation regardless of their weight, and those who use LCS in the long term experience damage to neurohormonal control of satiety (Christofides 2021).

LCS have not been evaluated properly for the neurohormonal and physiologic impacts to the human body (Christofides 2021). The rationale behind LCS use focuses on a simple thermodynamic relationship between energy intake and expenditure, but the pathophysiology of obesity is complicated (Christofides 2021; Archer 2022). There are not many studies investigating gender or comorbidity differences and how individuals respond to LCS (Christofides 2021). Studies that did control for these factors showed that LCS were actually detrimental to people wanting to address obesity and other diseases related to adiposity like diabetes and dyslipidemia (Pearlman 2017; Christofides 2021).

Gut Microbiome

There has been a good deal of research on the relationship between the gut microbiome and obesity. When germ-free mice are given a fecal microbiota transplant from an obese donor, they become obese (Maruvada 2016). People with obesity tend to have gut microbial genes that are involved in membrane transport functions and production of butyrate (which is produced when dietary fiber is broken down). They have depletions in microbial genes related to vitamin and nucleotide metabolism or transcription (Gomes 2018). Children and adults with obesity have a high *Firmicutes* to *Bacteroidetes* bacteria ratio compared to normal weight children (Aoun 2015; Gomes 2018). Furthermore, an increase in relative number of *Firmicutes* is inversely related to

resting energy expenditure and is positively correlated with obesity development (Aoun 2015). When obese people lose weight, the *Firmicutes* to *Bacteroidetes* ratio decreases (Gomes 2018). An increase in amount of *Bacteroidetes* is positively correlated with weight loss (Aoun 2015).

There are some other mechanisms by which the gut microbiome could affect obesity. Certain bacterial metabolisms can help with extraction of calories from food intake. They can also increase fat deposition in adipose tissue and impact hepatic inflammatory processes (Gomes 2018). Gut bacteria can affect secretion of inflammatory cytokines. Inflammation is related to obesity, as high concentration the inflammation biomarkers interleukin-6 (IL-6), C-reactive protein, and tumor necrosis factor (TNF) are all associated with obesity (Aoun 2015). The microbiome can affect lymphoid structures, the immune system, nutrient metabolism, lipid metabolism, and hormones/satiety, all of which affect obesity and weight overall (Gomes 2018). Bacteria may induce expression of genes involved in carbohydrate and lipid metabolism, meaning that more energy is absorbed from food intake, causing weight gain (John 2016).

Diet and the gut microbiome are closely related. As discussed, highly palatable, high-calorie foods are increasingly available, heavily marketed, and inexpensive. Availability of this type of food can shift food patterns to hedonic eating through changes to dopamine signaling pathways and also mechanisms in the intestine, but it can also affect those relating to the gut microbiome (Gupta 2020). These types of food products can change vagal afferent function, which involves the pathway between the gut and the central nervous system, systemic and local metabolic inflammation, systemic immune activation, and shifts in the gut microbiome and metabolome (Gomes 2018; Gupta 2020). Food addiction in itself is related to the brain-gut-microbiome relationship (Gupta 2020).

Overuse of antibiotics has been linked to obesity onset. Gut dysbiosis from dietary or environmental changes can lead to chronic inflammation and is linked with metabolic disease. Antibiotic use has been linked to changes in the gut microbiome and also an increased risk of inflammatory disorder development. When people are exposed to antibiotics at age two, they have a higher likelihood of developing obesity later on. Changes to the gut microbiome from antibiotic use early in life may be the cause of subsequent weight gain (Aoun 2015). Animal models have shown strong evidence for a link between antibiotic use and obesity, but this relationship is not completely clear in humans (Leong 2018).

Obesity treatment can sometimes change the gut microbiome, and at the same time, addressing the gut microbiome can also address obesity (Aoun 2015). The gut microbiome changes after bariatric surgery. This change is specific to the actual surgery and not to the weight-loss diet, indicating a role in the surgery and gut microbiome effects in the efficacy of bariatric surgery (Maruvada 2016). Oral supplementation with probiotics (live microorganisms), has been shown to reduce waist circumference, BMI, body weight, and overall body fat, especially visceral fat (Aoun 2015; Gomes 2018). Prebiotics (specific types of fiber) can reduce insulin resistance and contribute to weight loss (Gomes 2018). Food intake triggers that lead to weight gain may be prevented by supplementation with probiotics, prebiotics, and synbiotics (combinations of probiotics and prebiotics), which regulate hormonal, neurotransmitter, and inflammatory systems (Aoun 2015).

The gut microbiome can be affected by sleep, and sleep can affect the gut microbiome (Matenchuk 2020). Gut microbiota respond to circadian rhythms in the host, but this response changes based on the diet (Maruvada 2016). When people have an obesogenic diet, chronic jet lag, and a deficiency in clock genes, which regulate circadian rhythms, the gut bacteria's normal

oscillatory status is disrupted, although this can be reversed. Interestingly, metabolites from the gut microbiome can influence both central and hepatic clock gene expression, as well as sleep duration and body composition in the host (Maruvada 2016; Matenchuk 2020). When people have fragmented or shortened sleep, they are at risk of gut dysbiosis. This could be from activation of the hypothalamic-pituitary-adrenal axis (Leong 2018; Matenchuk 2020). Metabolic disturbances that happen because of sleep loss may happen through increased growth of certain types of bacteria in the gut (Matenchuk 2020). Changes to the gut microbiome have been linked to anxiety and stress, both have which been connected to sleep loss (Patel 2009; Maruvada 2016; Leong 2018; Tomiyama 2019). Interestingly, probiotic supplementation improves sleep quality (Matenchuk 2020).

Stress

From longitudinal studies, we see that there is a positive relationship between stress and weight gain (Wardle 2011). Stress can play a role in obesity development and maintenance through multiple pathways (Tomiyama 2019). There is a strong relationship between energy homeostasis and neuroendocrine pathways, specifically the hypothalamo-pituitary-adrenal axis (Hewagalamulage 2016). Our stress-response system is responsible for the release of glucose into the bloodstream during the fight-or-flight response. Most modern-day stress-inducing events are largely psychological and not physical, and the excess energy has nowhere to go except to be deposited as body fat (Tomiyama 2019). Patients with obesity, in particular excess central adiposity, have high cortisol levels (Hewagalamulage 2016).

Furthermore, stress is involved in food intake regulation and also energy expenditure, where high-stressed individuals eat more and do less activity (Hewagalamulage 2016). Stress can affect behavior and cause a higher desire for more palatable foods, like those high in fat, salt, and

sugar, and it can induce overeating. Stress in itself can trigger physiological changes in brain reward centers (Tomiyama 2019). Finally, stress also shortens sleep, which can exacerbate the neurological changes in appetite and food desires caused by stress (Patel 2009; Greer 2013; Tomiyama 2019).

Screen Time

Screen time has often been considered as a potential cause for a rise in obesity rates, although screens as a cause of sedentary behavior alone do not necessarily explain the complete cause of the average BMI rise. While physical activity level has remained relatively stable since 1980, television (TV) appears to have simply displaced other forms of sedentary time, like reading (Zimmerman 2011). For example, a study found that when older adults watched less TV, they were less likely to be obese, regardless of sedentary behavior overall (Inoue 2012). Screen use has especially increased at mealtimes (Zimmerman 2011). The American Academy of Pediatrics recommends limiting screen time for young children overall, including by completely excluding mealtimes from screen-viewing periods (Council on Communications and Media 2016). Many children and adolescents consume meals while watching screens, and socioeconomic status is inversely related to screen time during meals (Gebremariam 2015; Jensen 2022). There is a demonstrated propensity for individuals to eat more food overall during screen viewing compared to when they are not viewing screen entertainment (Jusiene 2019). Higher weekly TV viewing was associated with a less healthy diet and consumption of more sweets by children and SSBs in adolescents (Jensen 2022).

Television viewing is associated with adverse dietary practices in children as young as three (Miller 2008). In an intervention study that looked at the association between reduced television and computer screen viewing overall and body mass index in young children who had

a high BMI, the intervention was significantly associated with reduced BMI. Interestingly, the intervention was associated with reduced energy intake but was not associated with any change in physical activity in the children, sparking questions about the role of the screens themselves in BMI. In this particular study, the intervention was also more effective in children coming from families with low socioeconomic status than in higher-SES families (Epstein 2008).

Although the recommendation for screen time in children aged two to five years is less than one hour per day, only 35.6% of individuals met this guideline in 2020 (MacArthur 2022). 26% of US children in 1998 watched at least four hours of TV per day, and 67% watched at least two (Andersen 1998). In 1998, both boys and girls who watched at least four hours of TV per day had higher body fat and a higher BMI compared to children who watched less than two hours of TV per day (Andersen 1998). In the more recent years of social media, when 14-year-old girls spent a long time (>=5 hours/day) on social media, they had a significantly higher BMI z-score. This association was attenuated when controlling for sleep duration, body weight satisfaction, depressive symptoms, and overall well-being (Foubister 2023).

Increased screen time in children and adolescents reduces psychophysiological resilience, which is related to stress (Lissak 2018). Too much screen time is associated with poor sleep. It is also associated with obesity and poor stress regulation, including cortisol dysregulation and high sympathetic nervous system arousal (Lissak 2018; Manwell 2022). People with obesity are more likely than non-obese people to watch more TV, but they also tend to get less sleep (Vioque 2000). Addictive screen time use can involve craving behavior, which is similar to that with substance use disorder. Digital media use is related to structural changes in the part of the brain related to cognitive control in children (Lissak 2018). The recent increase in screen time is concerning for population psychological health—biopsychosocial research has

shown that too much screen time affects normal development of the brain and increases the risk of several psychological disorders, including those related to emotional regulation and addictive behaviors (Manwell 2022).

Marketing

A suggestion for the underlying cause behind the rise of obesity over time in all socioeconomic groups, which would align with the country-specific context, the dietary environment, and the increase in screen time is an increase in "savvy marketing," wherein targeted advertisements rooted in psychological principles lead people to unconsciously develop diet and lifestyle habits that lend themselves to obesity (Zimmerman 2011; Cofie 2022; Lee 2022). A study from 2010 found that children who watched television with commercial advertisements were more prone to obesity than children who watched television without advertisements (PBS) for the same amount of time—in this case, the screen time itself made no difference, but the marketing did (Zimmerman 2010; Zimmerman 2011). Another systematic review found an increase in the prevalence of overweight and obesity in children after they were exposed to food advertisements on television (Pourmoradian 2021).

A 2014 sociological study found that parents generally found food advertisements to be harmful to children (very few of them found the food advertisements to be "innocuous"). Many of the parents felt "disempowered" by the very high prevalence of advertising of unhealthy foods and believed that it negatively affected their children (Pettigrew 2014). In fact, marketing high fat, salt, and sugar foods to children using characters does influence the affected children's food preferences and choices (Packer 2022). Both adults and children are affected by food advertisement, specifically on the internet and on television (Pettigrew 2013). A systematic review published in 2022 identified that promotion of unhealthy snacks by social media

influencers led to higher snack intake in children, but promotion of vegetables did not increase vegetable consumption—any amount of time spent watching food and brand videos was associated with increased food intake overall (McCarthy 2022). There is a high predominance of advertisements for high-calorie, low-nutrient foods (Harris 2009). Marketing involves both overt advertisements and product placements; when children watched a movie where a Pepsi bottle was in a movie scene, they tended to choose Pepsi over Coke, regardless of if they had noticed the Pepsi bottle in the movie (Auty 2004; Harris 2009).

Food and beverage marketing are very prevalent throughout social media and live streaming gaming platforms, including YouTube Gaming, Twitch, and Facebook Gaming, especially in relation to energy drinks. The prevalence of this type of advertising increased substantially during the time surrounding the COVID-19 pandemic (Edwards 2022). Food advertising is prevalent on websites targeted toward children. In one study, Syndicated Internet exposure data examined food advertisement viewing on popular children's web sites between July 2009 and June 2010. 3.4 billion advertisements for food were seen on popular children's websites. Most of these ads (64%) were for breakfast cereal and fast food. 84% of the products advertised were high in fat, sugar, and/or sodium (Ustjanauskas 2014). YouTube banned food advertising on "made-for-kids" channels in 2020. An article published in 2023 analyzed appearance of food in child-influencer videos and analyzed a sample of 400 influencer videos targeted towards children. They found 260 (two-thirds of videos) videos that contained food appearances, including 153 with branded product appearances of salty/sweet snacks, ice cream, candy, and sugary drinks. Only one of the videos disclosed a paid sponsorship (Fleming-Milici 2023). Food and beverage marketing on Facebook were most attractive to young adults and adolescent users (Freeman 2014).

Food marketing and obesity are clearly related, especially for young people (Martinho 2020). By age nine, children can recognize advertisements on websites. By age five, they can recognize ads on television (Ali 2009). A national sample of US youth in 2010-2011 found that obesity, consumption of sugar-sweetened beverages (SSBs), frequency of fast-food restaurant visits, and weekday TV time were all positively associated with receptivity to TV fast food advertising. For each additional point increase in receptivity, the odds of obesity in those youth increased by 19% (McClure 2013). As of 2006, the average U.S. child viewed 15 TV food ads per day or about 5500 per year, but food advertising exposure has increased for all youth (Federal Trade Commission 2006; Harris 2009; Fleming-Milici 2018). A study using Nielsen panel data analyzed television viewing times and exposure to food advertising in youths from 2008-2012. They compared the results by network type, age group, and race. They found that black youth viewed about 50% more food advertisements than white youth of the same age, but this was due to a combination of greater television viewing and more advertisements on black- and youth-targeted networks (Fleming-Milici 2018). That being said, youth in all groups viewed large numbers of TV ads for unhealthy foods and beverages, but Hispanic children appear to see fewer food TV ads and have higher obesity rates (Fleming-Milici 2013).

Large food companies employ many marketing strategies, only some of which are the focus of public health research—efforts to reduce regulation of those marketing strategies are also present (Wood 2021). For instance, the food industry apparently spent about $1 billion USD lobbying against the traffic light labeling system in the U.K. (Scrinis 2015). Many dominant food companies have concentrated market power, which allows them to exert control over food environments, especially when many of their food products are highly processed (Wood 2021). Many large food companies (often ones that are transnational) operate in many different areas

(i.e., dairy and confectionary products) and in many different food processing areas (Wood 2021). High-profile food companies invest heavily into development of their products, which involves engineering the products to be visually appealing, have a favorable texture, be hyperpalatable, and almost or actually addictive (Moodie 2013; Wood 2021). Food companies can also use misleading marketing strategies, like using names and phrases that sound healthy, empty comparisons, and "greenwashing." The types of marketing and advertising that companies use are often specifically intended to be misleading, manipulate consumers, and alter their judgment (Wood 2021). Another relevant strategy is to address disruptors of the market and advance dietary displacement of marketed products compared to others, shifting consumption habits in certain ways. An example of this is the promotion of snacking over meals (Scrinis 2015; Wood 2021). "Big Food" and the transnational food corporations, including Nestle, Coca-Cola, and others bring in high revenues each year (Nestle sold almost $100 billion USD in 2013) and can displace certain diet staples with highly processed and packaged foods (Scrinis 2015).

There has been some psychological research on the mechanisms of marketing and how it affects both children and adults (Harris 2009). Neuromarketing methods encourage children to prefer taste when they make food choices, and the television and internet are effective ways to influence consumers. Television cooking shows, children's website, and social media all influence children and adolescent consumption patterns. Food marketing focuses on taste as an attribute to encourage customers to purchase food products (Martinho 2020). There are specific styles of typeface and product shape that are more attractive to consumers — but these appearances only affect "hedonic" goods (for emotional or sensual pleasure, like many types of food products) and not "utilitarian" goods (for basic needs and functional aspects, like detergents). Companies know what types of food they are selling and know how to make the

packaging look appealing for that specific type of food (Li 2020). Point-of-purchase marketing strategies are difficult for consumers to resist because they work through non-cognitive pathways and are hard to recognize (Cohen 2016). Interestingly, food advertising combined with a cognitively demanding task predisposes people to pick less-healthy foods and more calories (Zimmerman 2014).

In 2009, companies spent $1.79 billion marketing food to youth. From 2006 to 2009, food marketers spend 50% more on "new media" (online, mobile, viral marketing), 7% of total youth-targeted expenditures in 2009 (Federal Trade Commission 2012). Young people spend a good deal of money on food purchases, and marketing can increase childrens' knowledge about certain food products and influence their opinion of and interest in purchasing those products (Harris 2009). Companies can also use power asymmetries with consumers. They can use marketing to specifically target vulnerable groups, like young children, who cannot comprehend and understand marketing strategies (Wood 2021). Advertisers can collect personal data via the internet to deliver targeted advertising to children and youths, and they often use multiple marketing strategies to target children and have ads on TV, online, via product placement in movies, and in-school ads (Europe WHO 2016; Wood 2021).

Finally, companies target people in lower income neighborhoods with targeted outdoor advertising (Wood 2021). Outdoor advertising is also understudied. One study found that for each 10% increase in food or beverage advertising outdoors, any given individual in that neighborhood had 1.05 greater odds of being overweight or obese. This was consistent when accounting for race, income, and education level (Lesser 2013; Cassady 2015).

Solutions

There is limited work on effective treatments for obesity other than surgery, which can benefit individuals but comes with many risks (Ruban 2019). Bariatric surgery has been effective with sustained, long-term weight loss when combined with an intervention that reduces the obesogenic environment (Albaugh 2022). After losing weight from bariatric surgery, sleep apnea improved, along with metabolic levels (Jehan 2017). Bariatric surgery is seen as the preferred treatment when others have failed. It is more effective at inducing weight loss than non-surgical interventions. However, these can lead to nutritional deficiencies and some negative psychological impact (Ruban 2019).

Sleep quality improves after bariatric surgery, although the long-term effects are not clear (Lodewijks 2023). For patients who underwent bariatric surgery, the mean percentage of excess weight loss was 61.2% across all surgery types and ranged from 47.5% to 70.1% based on the specific surgery type. Interestingly, obstructive sleep apnea was resolved in 85.7% of patients who had surgery (Buchwald 2005). Among patients who had bariatric surgery at 21 years old or younger, mean total body weight decreased by 31.3%, even ten to 18 years after surgery. Sleep apnea went from 16.7% to 1.0%, and depression went from 27.1% to 4.2%, with a statistically significant difference in all measures (de la Cruz-Muñoz 2022).

Dietary therapy, focused on a net deficit of calories, is one approach to obesity treatment. This can be effective in individuals if a calorie deficit is maintained and adhered to (Ruban 2019). People who adhere to their nutritional plans lose more weight at six months post-bariatric surgery. General impulsiveness, along with adherence to dietary regimen and planning, were reliable predictors of weight loss after bariatric surgery (Marchitelli 2022). The gut microbiome

can be benefitted by diet, prebiotics, probiotics, careful use of antibiotics, surgery, and fecal transplantation, which could help improve the obesity epidemic (John 2016).

The impact of gut microbes on obesity needs to be better understood. However, dietary approaches that focus on improving gut microbiota, like an inclusion of fiber, polyphenols, and reducing consumption of high-fat foods and certain additives could be helpful long-term strategies for addressing obesity. Fetal microbial transplantation has not proven effective at addressing metabolic disorders in humans, and patients respond very differently (Maruvada 2016). There have not been effective approaches to address the adolescent and child obesity epidemic. Most youths with obesity have obesity as adults, and treatment during adolescence is complicated. There are behavioral, pharmaceutical, and surgical approaches to address this, but they have not worked at the population level (Cardel 2020).

Several pharmacological treatments for obesity are available. These drugs can have unpleasant side effects, and long-term effectiveness of many of them is unknown (Ruban 2019). The weight loss medication Tirzepatide—in the RCT, participants who received the lowest (5mg) dose had an average 11.9% reduction in body weight, specifically with a significant reduction in fat mass compared to lean mass (Jastebroff 2022). One drawback to these medications is that they generally need to be taken at a "maintenance dose" indefinitely, and it is uncertain if people will be able to take breaks from the medication (Rosen 2022). In the LEAN study, both telephone and in-person counseling improved C-reactive protein levels and were effective for sustained weight loss that lasted at least six months after the trial was completed. This was only for women treated for breast cancer (Harrigan 2016). The intragastric balloon has been used since the 1980s, but there is not much evidence to support its efficacy when compared to other approaches (Ruban 2019).

Personal agency is an important consideration, but the increase in fast food consumption can also be viewed in the context that companies can work within the "taste-engineering frame" and specifically engineer foods that taste good and intentionally increase over-consumption of those foods over time. Taking this context into account increases support for limits on food advertising and specifically limits for high-fat, high-sugar food advertising targeted to children (Ortiz 2016). Arguably, most individuals do not want to be obese, but people often make choices that result in unwanted effects, often after some type of manipulation; this principle directly relates to marketing issues and the obesity epidemic (Akerlof 2016). Food can have a similar neurological response to abused drugs, but these foods are also consumed more often and at younger ages. Early and frequent exposure to harmful food products in youth may have detrimental and difficult-to-address consequences in the long term, so approaches targeting youth may be particularly important (Gearhardt 2011).

While there is a preference for industry self-regulation, this has led to uneven and inconsistent improvements in food product quality; there are some regulatory approaches to obesity that use law (Sisnowski 2015). Even when marketing is somewhat regulated, products can still include misleading health and nutrition information. An example of this is misleading nutrient-content claims on the packages of highly processed foods being allowed even when marketing is regulated (Scrinis 2015). Exploitative marketing practices could be reduced by prohibiting marketing in certain times and places, like digital marketing of unhealthy foods, unhealthy food marketing at schools, and marketing of unhealthy food at times when children generally watch TV. Point-of-purchase policy options, like warning labels and marketing restrictions on highly processed foods would be implemented at the local level and still allow for consumer choice (Cohen 2016). Based on data from the 2017-2018 NHANES, 57% of total

energy would be subject to front-of-pack nutrition labeling schemes. People who consume more food away from home (i.e., at restaurants) are less exposed to front-of-pack labeling (FOPL), and people who are a healthy weight and who generally ate a healthier diet were more exposed to FOPL (Roark 2022). Front-of-pack nutrition labeling schemes have been suggested often in recent years, but less than 60% of the American diet would be subject to FOPL, making this strategy potentially helpful but less than ideal (Wood 2021; Roark 2022).

While food packaging can be to the consumers' detriment, it can help inform healthy choices and help consumers. Having cartoons on the front of unhealthy food packages can influence children and persuade them to choose those foods, but this strategy also works for fruits and vegetables (Scrinis 2016; Martinho 2020). Food marketing power could be wielded to promote healthy eating (Martinho 2020). Campaigns to increase fruit and vegetable consumption in the population may be helpful, but they need to take into account different demographics of the target population (Li 2016). People who prioritize nutrition while shopping do have higher-quality diets than those who don't, regardless of gender, education, or income. Taste is considered "very important" by 77% of US adults when purchasing food. This is true for 59.9% of adults in terms of nutrition, 39.9% in terms of cost, and 29.8% in terms of convenience. The perceived importance of nutrition strongly and positively predicts diet quality (HEI) scores. The perceived preference for taste and convenience is inversely related with overall diet quality (HEI score) (Aggarwal 2016). Encouraging importance of healthy food choices and nutrition over taste may improve diet, which could benefit the obesity epidemic at a population level.

Current Insights

The present study seeks to shed light on the interrelated nature of the factors relating to the obesity epidemic. The hypothesis, which has been informed by an in-depth analysis of the

literature, is that obesity is directly related to the human diet in addition to the gut microbiome, stress, sleep, screen time, marketing exposure, and environmental contaminants. However, these factors are also hypothesized to be connected to each other, creating potential causal pathways that may be important to investigate (Figure 1). The current analysis intends to conduct an analysis of prospective observational data in order to generate specific hypotheses surrounding the relationship between screen time and obesity that are informed by the evidence of a systemic cause of the obesity epidemic.

Methods

The statistical methods used in this project build on and complement those in a previous study; details on the methods used in the previous iteration are available elsewhere (Banas 2021). The previous study focused on obesity trends in the United States from 1959 to 2018 by race/ethnicity, income, and education. That study used an interrupted time series (ITS) method to analyze demographic variables and BMI over time, and the current study considered an update to that analysis (Banas 2021). The current study uses data from the National Health and Nutrition Examination Survey (NHANES) from 2003-2015, the years for which data on television and video time were available after variable ascertainment and data cleaning. NHANES data is nationally representative and collected biannually, with approximately 5,000 individuals included in each wave of the survey (Banas 2021, CDC [2]).

In order to complete an update of the methods from the 2018 study, a biostatistician from the Yale Center for Analytical Science was consulted independently to examine the ITS method, which was confirmed to be the current recommended statistical method for this type of project. The present study also analyzed the potential to update the previous iteration with newly published NHANES data. However, due to the COVID-19 pandemic starting in 2020, NHANES

data collection was stopped mid-2020. Therefore, the NHANES 2019-2020 data are not nationally representative. These data were combined with the previous iteration from 2017-2018 to create a 2017-2020 dataset that is nationally representative (CDC [1]). The 2017-2020 NHANES data are not included in the current project, as it would distort the nature of the intended analysis. This is because the previous analysis focused on small increments of data over time, with the point of interest on changes in obesity prevalence over time. Adding in the 2017-2020 dataset would not add additional data to the previous analysis (as the 2017-2018 data is already included there), and adding a four-year cycle to the analysis of two-year cycles would make comparison difficult.

SAS 9.4 was used for data analysis (SAS Institute Inc). The current study sought to analyze screen time and BMI in addition to the demographic variable analysis from the 2018 study (Banas 2021). NHANES 1999-2000 and NHANES 2001-2002 include a measurement of TV, phone, and computer use (PAQ480 and PAD480, respectively), although none of the NHANES cycles include a "screen time" questionnaire variable that encompasses all or most forms of screen time. NHANES 1999-2001 includes a measure of TV and video watching time from the previous day. Most recent cycles include a measurement of average daily TV and video watching over the past 30 days (PAD590 for 2001-2010 and PAQ710 for 2011-2016) (CDC [2]). Data on TV and video time per day was not collected for adults starting in 2017. The measurement of TV and video watching duration in adults per day is the subject of the present analysis.

Information on the TV and video variables was not available for 1999-2002 after data processing for the other variables. Therefore, TV and video time per day from NHANES 2003-2004 through NHANES 2015-2016 is included in the present analysis. After data cleaning,

information on TV and video time by race/ethnicity, income, and education level is reported for years 2003-2015. TV time was reported in incremental groups by hour of exposure, from zero hours to four or more hours. Four was chosen as the time cutoff because of evidence that at least four hours of TV or screen exposure is related to increased risk of obesity (Andersen 1998, Vioque 2000). Regression lines were created using data from 2003-2015 to reflect the reported and available data.

An additional ITS analysis on TV and video time per day was not conducted in the present study for several reasons. This study is meant to be a complement and update to the 2021 study, which analyzed data from 1959-2018; the available screen time data included here only ranges from 2003-2015, which would not provide for a parallel analysis with the previous iteration of the study (Banas 2021). There were also five screen time groups, and because the estimates of screen time are based on questionnaire data, the average number of hours of TV and video time per day are most likely not distinct; in other words, the TV time categories are less clear than the demographic variables, for which the ITS was recommended. Another reason is that there is a large amount of missing data for the screen time variable (Appendix 1; Appendix 2; Appendix 3). Alternatively, F-tests along with interaction terms in the regression models were used to compare the regression slopes. Figures for BMI and TV/video time were created using SAS 9.4 (SAS Institute Inc). The conceptual system hypothesis was created using Canva (McDowell Cook 2023; Canva Inc).

Results

Results from the previous iteration of the study are available elsewhere; the current results serve as a complement and update to the previous analysis (Banas 2021). Among the available screen time observations from 2003-2015 over time, 3,345 (12.37%) were zero TV or video hours,

4,376 (16.19%) were one hour, 6,827 (25.25%) were two hours, 4,532 (16.76%) were three hours, and 7,957 (29.43%) were four or more hours (Table 1). The regression analysis of the zero-hour group from 2003-2015 was not statistically significant at the 0.01 level, indicating no significant increase in BMI over the data years. The four other groups had positive slope estimates that were statistically significant. The one-hour group had an estimated 0.056 increase in BMI per year. For the two-hour group, this was an increase of 0.066 per year; for the three-hour group, there was a BMI increase of 0.073 per year, and there was a BMI increase of 0.090 per year for the four or more hours group (Table 2). A breakdown of the screen time data by race/ethnicity, income, and education level are available in the appendix (Appendix 1; Appendix 2; Appendix 3). From the tests comparing regression slopes, none of the coefficients for individual groups or interaction terms were statistically significant, and none of the interaction terms between TV time group and midpoint year were significantly different from zero (Table 3; Table 4).

Discussion

Based on the results of regression analyses, there is evidence that obesity has increased over time, particularly in groups with a high amount of daily TV and video time (Table 2). This is consistent with the previous study results, which showed that obesity rates increased over time overall and for all demographic groups (Banas 2021). That being said, mean BMI was well within the overweight range (>=25) for all TV and video time groups for the entire time period, although only the highest TV and video time group (4+ hours per day) had an average BMI in the obese range (>=30), and this was only in later years (Figure 2) (WHO 2000). Interestingly, the present study did not find evidence of a difference in regression slopes between TV time groups; in other words, there is no evidence from this analysis that duration of TV and video

time per day differentially impacts obesity rates over time (Table 3; Table 4). This is consistent with previous literature, which suggests that screen time in itself may not cause obesity and that the true relationship may be with a factor related to screen time, such as marketing or screen time during meals (Zimmerman 2011; Gebremariam 2015; Jensen 2022). It is important to note that the statistical tests conducted in the present study are intended as hypothesis-generating tests, as there are many additional factors needed to draw conclusions based on the test results.

This study has several strengths. The background information provides a robust and systemic hypothesis about the interconnected nature of factors involved in the obesity epidemic, and it provides an important context for results reported in the previous study (Banas 2021). The longitudinal nature of the analysis of TV and video time and average BMI using NHANES data is a novel addition to the existing literature, which largely focuses on cross-sectional and short-term estimates of screen time and BMI (Epstein 2008; Jusiene 2019; Jensen 2022). Additionally, the statistical analysis portion of this study as a complement to the extensive background information provides a unique approach and adds new hypotheses to the discussion surrounding the obesity epidemic.

This study also has several limitations. In particular, the television and videos variable used has several flaws. This variable, which records estimated daily number of hours of television and videos watched per individual, most likely does not provide a good estimate of the behavior of interest, which is screen time in general. It does not include time spent on other types of screens, like non-video computer time or time spent on social media without video watching. There were many instances of missing observations of the TV time variable, especially in comparison to the demographic variables used in the previous iteration of the study (Banas 2021; Appendix 1; Appendix 2; Appendix 3). This may have led to bias in the results. The regression

models created in this analysis do not account for demographic or other factors that may impact obesity. Finally, the statistical analysis portion of this study was originally meant to complement the previous iteration and generate additional hypotheses, but the limited 2003-2015 time frame makes comparison of the TV time variable analysis with that of the demographic variables difficult (Banas 2021).

Before settling on a path for treatment, the actual causes of obesity hypothetically need to be investigated and addressed, although we may be able to identify effective treatments without knowing how or why they work. There is evidence for several, interrelated disease physiologies and causes (Wright 2012; Archer 2022). For example, screen use and sleep are both related to each other and to obesity (Foubister 2023). Another example is the interrelated nature of diet, sleep, stress, and the microbiome—and the relationships of all of these factors to the development of obesity itself (Patel 2009; Zimmerman 2011; Maruvada 2016; St-Onge 2016; Leong 2018; Tomiyama 2019). Each one of these different topics warrants a systematic review and likely more original research, but they are clearly interrelated, so looking at the entire picture through a systems thinking approach is critical to identify the true cause of the obesity epidemic.

To find a solution, we must look at the unintended consequences of interventions. For example, the marketing of unhealthy foods is clearly a problem (Zimmerman 2011). Beyond any philosophical concerns for heavily regulating marketing, serious market interventions, when done incorrectly, could have devastating economic effects (both on the marketing and food product industries)—and since stress is related to obesity, and healthy diets cost more money, these are important concerns (Hewagalamulage 2016; Tomiyama 2019; Conrad 2021). The effects of market intervention here are beyond the scope of this paper, but it is important to take all aspects of the obesity epidemic into account when making these decisions.

Much more analysis needs to be completed on all of these factors, including food marketing, antibiotic use, sleep duration, screen time, the gut microbiome, stress, and BPA exposure. These need to be analyzed in a longitudinal way with comparisons of obesity rates, as well as the synergistic effects of them all. Perhaps we should think about the "drunkard search principle." If we only look under the streetlight, where the light is good, then we will never find the keys that we dropped in the park (Freedman 2010). Only ever focusing in on specific, evidence-based interventions will never fully reveal the depth and breadth of the obesity problem.

Tables and Figures

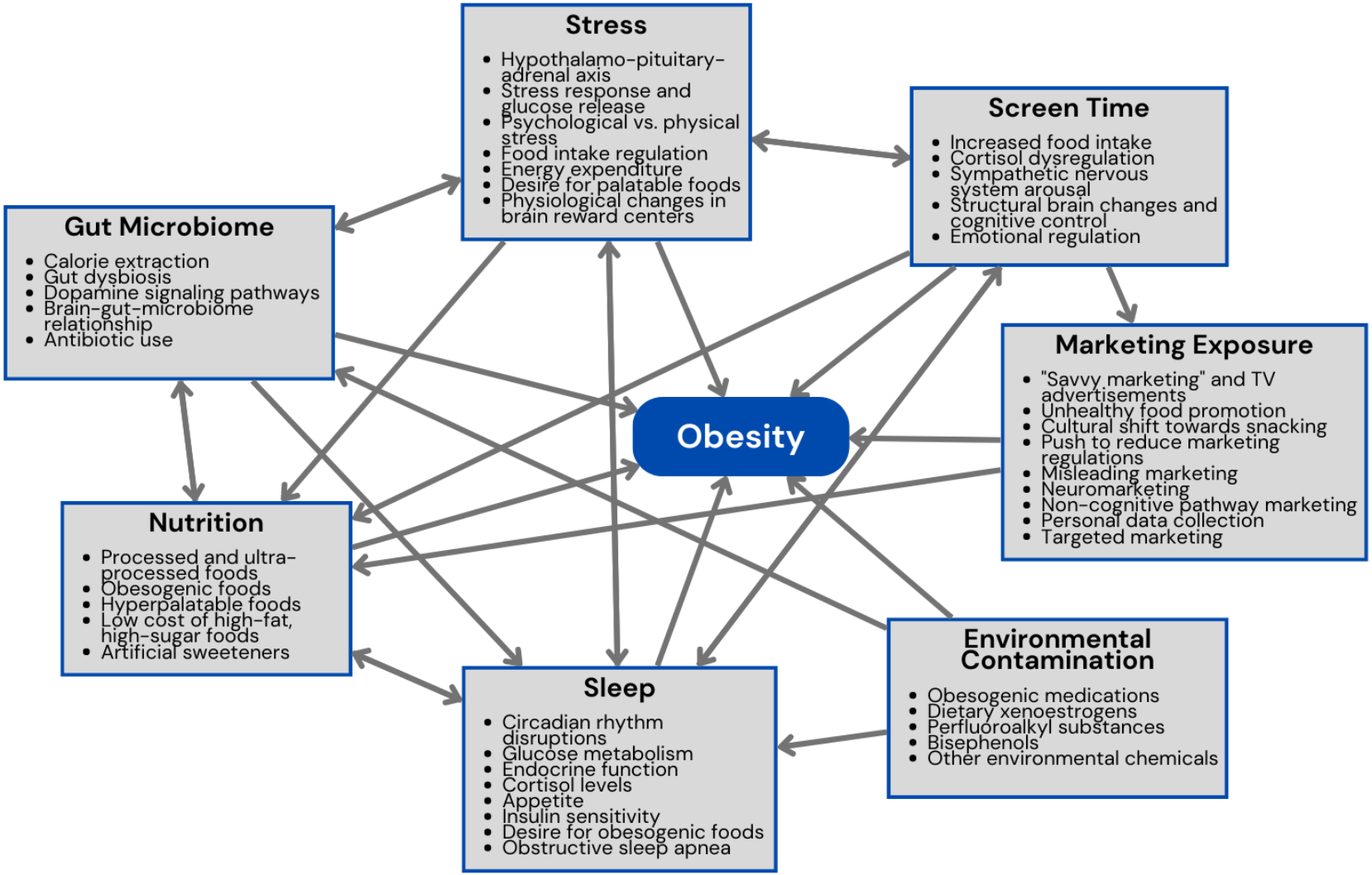

Figure 1. Systems thinking hypothesis of obesity epidemic (McDowell Cook 2023). Different factors in each category are all related to obesity, and many factors are hypothesized to be related to each other.

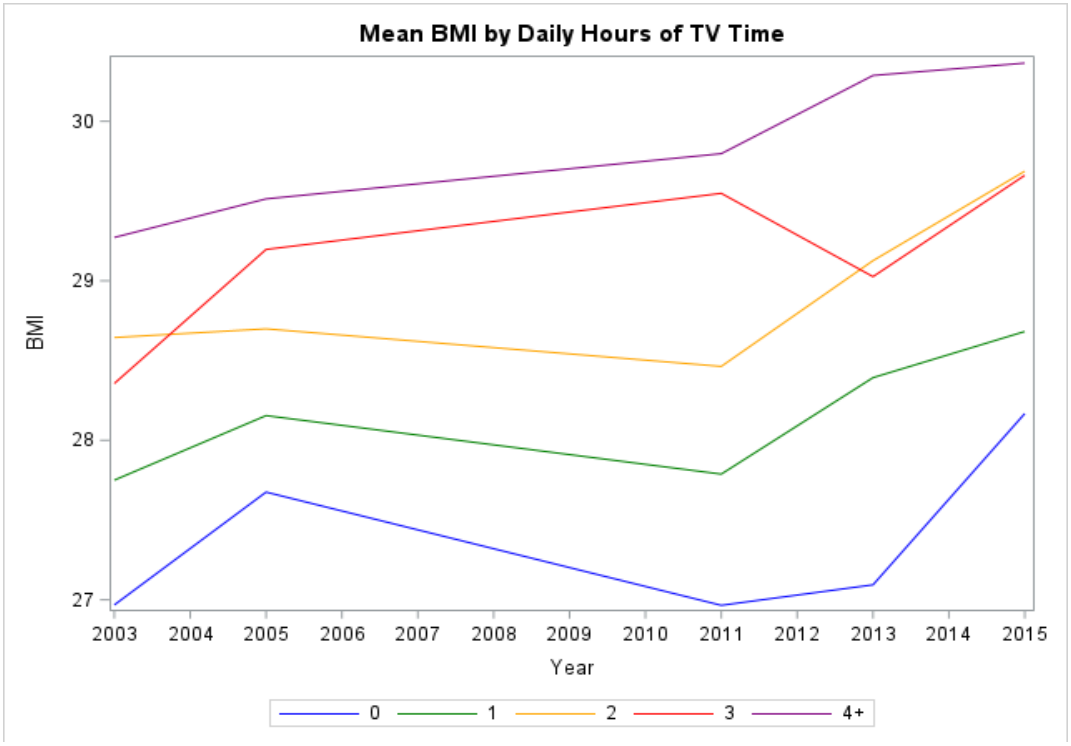

Figure 2. Mean BMI by daily hours of TV and video time, biannually 2003-2015.

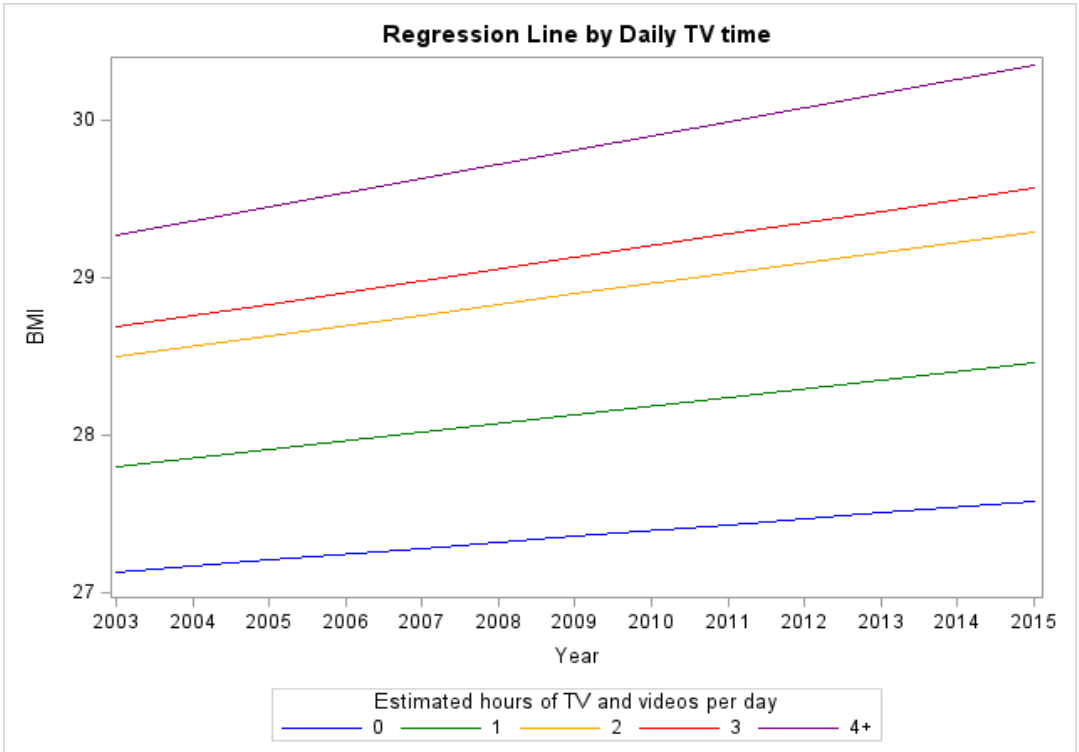

Figure 3. Linear regression by TV and video time group, 2003-2015 (see Table 2).

Group	Frequency	Percent
0 hours	3,345	12.37
1 hour	4,376	16.19
2 hours	6,827	25.25
3 hours	4,532	16.76
4+ hours	7,957	29.43

Table 1. Frequency of individuals in each TV time group, 2003-2015. There were 27,037 complete and 12,184 missing observations. Individual percentages may not add to 100.00% due to rounding.

Group	Estimate	Standard Error	P-value
0 hours	0.037	0.024	0.116
1 hour	0.056	0.021	0.009*
2 hours	0.066	0.018	<0.001*
3 hours	0.073	0.022	0.001*
4+ hours	0.090	0.020	<0.001*

Table 2. Results of individual regression analyses of average change in BMI over time among different TV time groups, 2003-2015 (see Figure 3). P-values that are significant at the 0.01 level are marked with an asterisk.

Group	Estimate	Standard Error	P-value
Intercept	-47.066	53.027	0.375
Year	0.037	0.026	0.160
1 hour	-36.647	70.447	0.603
2 hours	-57.166	64.619	0.376
3 hours	-71.537	69.908	0.306
4+ hours	-104.191	63.711	0.102
1 hour*year	0.019	0.036	0.595
2 hours*year	0.029	0.032	0.364
3 hours*year	0.036	0.035	0.294
4 hours*year	0.053	0.031	0.094

Table 3. Results of multiple regression using indicator variables to evaluate the effect of TV time groups on estimated change in BMI over time, 2003-2015. P-values that are significant at the 0.01 level are marked with an asterisk.

Group	Estimate (F-value)	P-value
0 hours (reference)	-	-
1 hour	0.28	0.595
2 hours	0.83	0.364
3 hours	1.10	0.294
4+ hours	2.80	0.094

Table 4. Results of analysis comparing regression estimates between groups using interaction terms (year*TV time group), 2003-2015. From testing for a difference between the interaction term and zero, P-values that are significant at the 0.01 level are marked with an asterisk.

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Appendix

Group	White	Black	Hispanic	Other	All
0 hours	1,009 (13.49%)	508 (8.56%)	1,256 (11.95%)	572 (18.40%)	3,345 (12.37%)
1 hour	1,308 (17.49%)	696 (11.72%)	1,752 (16.66%)	620 (19.94%)	4,376 (16.19%)
2 hours	1,997 (26.71%)	1,355 (22.82%)	2,676 (25.45%)	799 (25.70%)	6,827 (25.25%)
3 hours	1,298 (17.36%)	970 (16.34%)	1,816 (17.27%)	448 (14.41%)	4,532 (16.76%)
4+ hours	1,865 (24.94%)	2,408 (40.56%)	3,014 (28.67%)	670 (21.55%)	7,957 (29.43%)
Column Total	7,477 (100.00%)	5,937 (100.00%)	10,514 (100.00%)	3,109 (100.00%)	27,037 (100.00%)

Appendix 1. TV time by race and ethnicity group, 2003-2015. There were 27,037 complete and 12,184 missing observations. Percentages included are column percentages by race/ethnicity group. Individual percentages may not add to 100.00% due to rounding.

Group	Not low-income	Low-income	All
0 hours	734 (16.66%)	251 (10.92%)	985 (14.69%)
1 hour	928 (21.07%)	290 (12.62%)	1,218 (18.17%)
2 hours	1,301 (29.53%)	503 (21.89%)	1,804 (26.91%)
3 hours	644 (14.62%)	329 (14.32%)	973 (14.52%)
4+ hours	798 (18.12%)	925 (40.25%)	1,723 (25.70%)
Column Total	4,405 (100.00%)	2,298 (34.28%)	6,703 (100.00%)

Appendix 2. TV time by income group, 2003-2015. There were 6,703 complete and 32,518 missing observations. Percentages included are column percentages by income group. Individual percentages may not add to 100.00% due to rounding.

Group	Less than high school	High school diploma	Greater than high school	All
0 hours	802 (11.78%)	543 (8.83%)	1,995 (14.21%)	3,340 (12.37%)
1 hour	1,041 (15.28%)	847 (13.77%)	2,482 (17.68%)	4,370 (16.19%)
2 hours	1,534 (22.52%)	1,474 (23.96%)	3,813 (27.16%)	6,821 (25.26%)
3 hours	1,142 (16.77%)	1,088 (17.69%)	2,299 (16.38%)	4,529 (16.77%)
4+ hours	2,292 (33.65%)	2,199 (35.75%)	3,449 (24.57%)	7,940 (29.41%)
Total	6,811 (100.00%)	6,151 (100.00%)	14,038 (100.00%)	27,000 (100.00%)

Appendix 3. TV time by education group, 2003-2015. There were 27,000 complete and 12,221 missing observations. Percentages included are column percentages by education group. Individual percentages may not add to 100.00% due to rounding.