The Relationship between Obesity and Disorders of the Alimentary Canal

**Corresponding Author:**
Dr. John Zenian,
Research Scientist, n/a, 1341 Portsmouth Ave, 60154 - United States of America

**Submitting Author:**
Dr. John Zenian,
Research Scientist, n/a, 1341 Portsmouth Ave, 60154 - United States of America

**Article ID:** WMC003983  
**Article Type:** Original Articles  
**Submitted on:** 09-Feb-2013, 01:40:46 AM GMT  
**Published on:** 09-Feb-2013, 11:43:38 AM GMT  
**Article URL:** [http://www.webmedcentral.com/article_view/3983](http://www.webmedcentral.com/article_view/3983)  
**Subject Categories:** OBESITY  
**Keywords:** United States Obesity Epidemic, Physical Activity, Energy Dense Foods, Dental Caries, Dentine Sensitivity, Gastro-epophageal Reflux Disease, Diarrhea, Antacids, Antibiotics  
**How to cite the article:** Zenian J. The Relationship between Obesity and Disorders of the Alimentary Canal. WebmedCentral OBESITY 2013;4(2):WMC003983  
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**Source(s) of Funding:** None  
**Competing Interests:** None
The Relationship between Obesity and Disorders of the Alimentary Canal

Author(s): Zenian J

Abstract

The increase in the prevalence of obesity in the United States has been attributed to an environment that promotes a sedentary lifestyle and offers an abundance of energy rich affordable foods. Close examination of historical trends in occupational physical activity and the consumer price index for various food categories do not necessarily support such a view. It is proposed that the increase in the prevalence of obesity in the United States can be attributed to progress in the prevention and the treatment of oral and gastrointestinal disorders which reached a critical mass during the period from 1958 to 1970 prior to the onset of United States obesity epidemic. Time trend analysis shows that the prevalence of dental caries, peptic ulcer and the mortality rate from diarrhea decreased, while the prevalence of gastro-esophageal reflux disease increased prior to or concurrent with the increase in the prevalence of obesity. Furthermore, the number and scope of preventive or treatment measures for dental caries, malocclusion, dentine sensitivity, gastro-esophageal reflux disease and diarrhea increased during the same period. Improvements in the prevention and treatment of dental caries and malocclusion produced a more efficient masticatory apparatus which enabled people to consume more food, while treatment of dentine sensitivity enabled people to consume more sugar containing foods and beverages. Effective suppression of gastric acid enabled people to eat more foods without experiencing heartburn, and better treatment of diarrheal diseases enabled people to absorb more nutrients from the ingested food. The overall improvement in the health and efficiency of the alimentary canal should be considered as a possible alternative explanation for the development of the United States obesity epidemic.

Introduction

The United States population experienced a dramatic increase in the prevalence of obesity in the latter part of the twentieth century. This increase, which began in 1978 (1,2) and continues to this day, is commonly referred to as the United States obesity epidemic (3). Many theories have been formulated to explain this increase in obesity (4,5). Most of those theories however, explain why some people gain weight while others don’t, without explaining the sudden increase in obesity in the late 1970s that developed into an epidemic. The most widely accepted theory that addresses the epidemic of obesity in the United States attributes the increase in the prevalence of obesity to an environment which promotes a sedentary lifestyle and encourages the consumption of relatively cheap energy dense foods (6,7,3).

The increase in the prevalence of obesity began at a time when the United States experienced a fitness revolution, as evidenced by the popularity of jogging in the 1970s, aerobic exercise in the 1980s, and resistance training in the 1990s. Furthermore, the number of girls and women competing in sports grew exponentially as a result of the passage of the 1972 federal law giving women equal opportunity in sports (8). At the same time, the American public became more aware of the importance of nutrition in health due to the increased attention given in the mass media to the role of lipids and cholesterol in heart disease which led to the proliferation of diet programs (9), and a decline in fat consumption after 1980 (10). The historical context of the United States obesity epidemic therefore, contradicts the environmental theory of obesity which is currently the prevailing opinion concerning obesity in the United States.

The purpose of this communication is to examine critically the environmental theory of obesity and propose an alternative theory which postulates that progress in the prevention and treatment of the disorders of the alimentary canal was responsible for the United States obesity epidemic.

Methods

General Considerations: Only those diseases or conditions of the alimentary canal that affect a substantial portion of the U.S. population and have a demonstrable effect on body weight or nutrient intake, or their prevention or treatment has an effect on body weight or nutrient intake, were selected for inclusion.
Data about diarrheal disease burden was obtained from the National Health and Nutrition Examination Survey (NHANES) (17,18). The caries status of each person was assessed by the total number of decayed, missing and filled teeth known as the DMFT score. The median age of the groups selected fell in the range from 28 to 29.5 years. (17,18). Water fluoridation data were based on the Fluoridation Census carried out by the Centers for Disease Control (19,20). Data on the number of dental procedures were based on the Survey of Dental Practice and Services Rendered published by the American Dental Association. (21).

Gastro-esophageal Reflux Disease and Peptic Ulcer:
The prevalence of Gastro-esophageal reflux disease was based on the number of hospitalizations for white male veterans at all Veterans Administration hospitals throughout the United States obtained from the records of the Department of Veterans Affairs (22). The data for peptic ulcer referred to patient visits to the physicians’ office and antacid therapy referred to the percentage of antacid drugs among all drugs used to treat gastric and other ulcers of unspecified site. Both data were based on statistics from the National Diseases Therapeutic Index compiled by IMS America (23).

Diarrhea:
Data about diarrheal disease burden was based on the mortality rate for diarrheal diseases among American children published by the Centers for Disease Control (24). Although these data do not represent the actual prevalence of diarrheal diseases in the United States, their change suggests improvements in prevention or treatment of diarrheal diseases in the United States or perhaps in the prevalence of diarrheal diseases. Data about antibiotic and antimicrobial drug use came from the National Prescription Audit (25) which records all antibiotics sold in pharmacies and the National Ambulatory Medical Care Survey (26) which gathers prescription data from practicing physicians. Since the second survey does not cover all prescriptions issued in the United States, the two surveys were treated separately and comparisons were made only within each survey.

Results

The Environmental Theory of Obesity:
The first part of the environmental theory postulates that the U.S. obesity epidemic was brought about by a shift from an active to a sedentary lifestyle. In adults, this shift was primarily caused by the transition from an industrial to a service based economy (7, 16). As more and more...
Americans left manufacturing jobs and joined service related jobs, nationwide occupational physical activity declined, causing Americans to spend less energy (16, 6). In support of this theory, a recent comprehensive study (16) showed that both occupational physical activity and occupational energy expenditure declined between 1960 and 2000. However, time trend comparison of the decrease in occupational energy expenditure with the increase in the prevalence of obesity does not support such a conclusion. While occupational energy expenditure declined at essentially the same rate between 1960 and 1980, (4.3%), and between 1980 and 2000, (4.9%), the increase in the prevalence of obesity between 1980 and 2000 (15.9 extra percentage points) was ten times greater than the increase in the prevalence of obesity between 1960 and 1980 (1.6 extra percentage points) (Table 1). The lack of proportionality between the rate of decrease in occupational energy expenditure and the rate of increase in obesity at different intervals seriously undermines the idea that a decrease in occupational activity is one of the principal causes of the U.S. obesity epidemic. Further evidence against the role of occupational activity comes from studies that have compared obesity and occupation. The prevalence of obesity among employees who engaged in occupations involving low levels of physical activity was found to be only 1.7 percentage points higher than the prevalence of obesity among employees engaged in occupations involving high levels of physical activity(27). Even if we assume that there was a total shift from high to low physical activity occupations in the U.S. work force between 1960 and 2000, this difference would account for only a fraction of the increase in the prevalence of obesity in the United States in the same period, which amounted to 17.5 percentage points. Results from a study of time trends in the prevalence of obesity among various occupational groups also do not support the role of occupational physical activity in preventing obesity (28). A comparison of trends in the prevalence of obesity among five occupations involving low physical activity (managers, management related occupations, teachers, sales personnel and financial records keepers) and five other occupations involving high physical activity (farm workers, mechanics, assembly workers, mail carriers, and cleaning workers) between 1986 and 1995, showed that the pooled average annual increase in obesity was 10% higher among men engaged in occupations involving low physical activity than among men engaged in occupations involving high physical activity. In contrast, the pooled average annual increase in obesity was 24% lower among women engaged in occupations involving low physical activity than among women engaged in occupations involving high physical activity. Once again, occupational physical activity did not seem to have an overall protective effect against obesity.

The prevalence of obesity among children has been attributed to excessive television viewing (4, 29). It has been argued that increased time spent watching television would promote a sedentary lifestyle among children and discourage their participation in physical forms of recreation. Historical studies of trends in television ownership and viewing do not support such a theory. Thus, a 22% increase in television viewing between 1963 and 1980 (15) was accompanied by a 0.4 percentage point increase in the prevalence of obesity among boys and girls between the ages of 12 and 19 (3), whereas a 14.2% increase in television viewing between 1980 and 2000 (Table 1) was accompanied by an 11.1 percentage point increase in the prevalence of obesity in the same group (3). Even though television viewing increased at a lower rate between 1980 and 2000 than it did between 1963 and 1980, it was accompanied by an increase in obesity that was nearly 28 times greater between 1980 and 2000 than it was between 1963 and 1980. While it is conceivable that some parents limited their children’s television viewing, there is no reason to believe that such parental guidance declined suddenly after 1980 when obesity rates increased dramatically. Once again the lack of proportionality in the relationship between television viewing and the prevalence of obesity at two different periods suggests that the two phenomena are not causally related. It should be remembered that television viewing had become widespread long before the onset of the obesity epidemic. The percentage of U.S homes owning a television set increased from 9% in 1950 to 90% in 1962 (30), sixteen years before the onset of the obesity epidemic.

The problem of inactivity among American children is not a new phenomenon. A study of children living in New York in 1940 concluded that 72% of them were inactive (31). Another study published in 1956, observed that suburban high schoolgirls were not physically active (32). Before the advent of television, other forms of sedentary recreation could attract children, such as attending movie theaters, listening to the radio, or reading comic books, yet such forms of recreation did not apparently trigger an epidemic of obesity among American children.

The second part of the environmental theory suggests that the obesity epidemic is the result of increased consumption of relatively cheap calorie dense
processed foods. It is argued that since price is a major factor in making purchasing decisions, the sudden increase in the consumer price index for fruits and vegetables that took place in the 1980s may have forced Americans to consume the cheaper calorically dense processed foods and gain weight (7). However, data published by the Department of Agriculture show that the per capita consumption of fruits and vegetables increased in every decade between 1970 and 2000 including the 1980s when the consumer price index for fruits and vegetables showed a sudden increase (Table 1). Furthermore, the increase in the per capita consumption of calories from carbohydrates declined significantly from 5.1 percentage points in the 1980s to 0.9 percentage point in the 1990s, even though the combined consumer price index for sugar and sweets, and cereals and bakery products, which include cakes cookies and pies, registered a smaller increase in the 1990s (29%), than it did in the 1980s (52%) (Table 1). These results suggest that factors other than a sedentary lifestyle and the affordability of calorically dense processed foods may be responsible for the United States obesity epidemic.

The Relationship between Obesity and Disorders of the Alimentary Canal: Results from historical studies of public health have led to the conclusion that a mutual relationship exists between health and body weight (33). Many diseases such as tuberculosis, anemia and peptic ulcer were found to be associated with lower BMI(34,35). Other diseases such as bronchitis, emphysema and asthma showed a U shaped relationship with body weight where the prevalence of the disease was higher at both ends of the BMI spectrum (35). Improved health due to eradication or treatment of such diseases would be associated with increased body weight (7). Consequently, inductees to the United States Armed Forces during World War II were found to be heavier than inductees during World War I (36). However, weight gain associated with recovery from such diseases was likely to be modest, since many of those diseases had a U shaped relationship with body weight and/or did not involve the alimentary canal. In contrast, the eradication and treatment of the diseases of the alimentary canal are likely to have a more profound effect on body weight, because in addition to restoring well being, they can improve the function of the alimentary canal in processing, digesting and absorbing food. Among the disorders of the alimentary canal that are likely to have such an impact on body weight are dental caries, malocclusion, dentine sensitivity, gastro-esophageal reflux disease, peptic ulcer and diarrhea.

Dental health and body weight: For most people, solid food represents the principal source of caloric intake. Before being swallowed, solid food needs to be shredded by mastication. For optimum masticatory performance, an individual needs a full complement of intact teeth and a functionally stable occlusion (37). Having decayed or missing teeth and an unstable occlusion, undermine one’s ability to chew food properly and may result in diminished caloric intake (38) and lower body weight (39). Dentine sensitivity, on the other hand could affect one’s ability to enjoy sugar containing foods and beverages.

Dental caries and body weight: Dental caries is the most common cause of tooth loss (40). Those with fewer or decayed teeth possess lower bite force (41, 42) and experience chewing disability (43, 44) and oral pain (45, 43) during eating. Furthermore, tooth loss can result in reduced total energy intake (46) and dietary restriction (44, 47). In children, severe dental caries is associated with lower body weight (48, 49, 50), whereas accelerated dental development (50), or dental rehabilitation is associated with subsequent weight gain (49, 51). Such findings suggest that a reduction in the prevalence of dental caries would result in better mastication, greater food consumption, and subsequent weight gain.

Several advances in the prevention and treatment of dental caries in the second half of the twentieth century helped reduce the prevalence of dental caries in the United States. Until the 1940s, tooth extraction was a popular form of treatment due to the widespread acceptance of the theory of focal infection (52). Subsequent improvements in the diagnosis and treatment of dental caries reduced the need for tooth extraction. The use of preventive procedures, such as periodic check ups, plaque removal and topical fluoride application, had a significant impact on the dental health of the U.S. public. Preventive dentistry, which began to increase in the 1950s, became so popular that by 1976, 45% of all U.S. dentists reported employing dental hygienists (53). However, the greatest advance in the prevention of dental caries was achieved by fluoridation of the drinking water. The anti cavity property of fluoride, which was confirmed from epidemiological evidence gathered in the 1930s and 1940s (19), led to the idea that fluoridation of drinking water could help prevent tooth decay. Fluoridation of drinking water in the United States began in 1945. By 1975, almost half of the U.S. population was exposed to fluoridated drinking water (19) (Table 3). The combined effect of these advances in prevention and treatment of dental caries was...
Malocclusion and body weight: A proper intercuspal relationship which leads to a functionally stable occlusion between the upper and lower molars is important for the optimal performance of the masticatory apparatus (54). An improper or abnormal intercuspal relationship can give rise to malocclusion. According to a recent survey, only 35% of the adult U.S. population has optimal occlusion (55). Malocclusion, especially in severe cases, can affect one’s ability to break down and swallow food (54). Subjects with malocclusion have poorer masticatory performance than subjects with normal occlusion (56). They perform more irregular and inconsistent strokes during mastication (57), and execute more masticatory cycles, and take longer to eat the same amount of food than those with normal occlusion (58), possibly as an adaptive response to compensate for the smaller occlusal contact area that they possess (59). Furthermore, malocclusion is associated with a greater risk for temporomandibular dysfunction, (60) a disease which impairs masticatory function (61) and dietary intake (62).

Since malocclusion impedes masticatory function and masticatory function is related to body weight (63, 64) it would be expected that there would be a negative relationship between malocclusion and body weight. There are no large scale studies that have addressed this relationship, but some studies suggest that such a relationship may exist. One study has found that the prevalence of overweight is 5% less and the prevalence of underweight 2.5% more among adolescents with dental crowding (65). Another study has shown that the occurrence of two or more malocclusion traits was significantly higher among premature babies (66), who are known to achieve lower BMI later in life (67). Finally, obstructive sleep apnea patients who were obese had less craniofacial abnormalities including class II malocclusion than those who were not obese (68).

A negative association between malocclusion and body weight would imply a downward trend in the prevalence of malocclusion parallel to the upward trend in the prevalence of obesity. A recent survey has shown that the prevalence of overbite in the United States declined between 1966 and 1988 (69). This progress can be attributed to the six fold increase in the popularity of orthodontic treatment between 1959 and 1979 (Table 3). Orthodontic treatment reduces the number of occlusal interferences (70), improves mastication (71), reduces the incidence of temporomandibular disease (72) and leads to increased body weight (73).

Dentin Sensitivity and Body Weight: Dentin sensitivity is a condition that is characterized by sharp pain arising from external stimuli such as dietary sugar, in otherwise intact or normal teeth. It is estimated that it affects 15-20% of the adult U.S. population (74). Nerve sensitization due to occlusal or other trauma (75) can make the dental pulp vulnerable to osmotic shock caused by high concentrations of sugar found in certain foods and drinks. Soft drinks and fruit juices are particularly harmful to sensitive teeth (76), since they contain citric acid and sugar in high concentrations. The citric acid exposes the dental pulp by opening the dentinal tubules, and the sugar delivers the osmotic shock. Those who suffer from dentine sensitivity cannot enjoy soft drinks unless they receive treatment for dentine sensitivity.

Treatment of dentine sensitivity first gained scientific basis in the early 1940s when a preparation of sodium fluoride paste applied topically proved to be effective in clinical trials (77). As a result, office treatment of dentine sensitivity with fluoride paste increased across the United States in the following decades (Table 3). Eventually, office treatment of dentine sensitivity branched into other methods such as dentine coating and laser treatment with longer lasting effects (78). In home treatment of dentine sensitivity began in 1961 with the launching of Sensodyne, the first desensitizing toothpaste (77). It was followed by other forms of do it yourself treatments such as mouth washes and chewing gum (79).

At present, there are no studies of time trends in the prevalence of dentine sensitivity in the United States. However, there is reason to believe that a decline in the prevalence of dentine sensitivity may have occurred, because the prevalence of many dental disorders which contribute to the development of dentine sensitivity, such as malocclusion, (75) dental caries, large restorations (80) and tooth loss (81) have all declined in recent decades (69) (Table 2). The prevention and treatment of dentine sensitivity, can in the long run, enable more people to consume sugar containing foods and beverages without suffering adverse consequences. Consequently, the consumption of sugar containing soft drinks, which are
considered to be a contributing factor to the obesity epidemic (82), increased by 60% from 22.2 gallons per person per year in 1970 to 35.6 gallons in 1990 (Table 1).

Gastro-esophageal reflux disease and body weight: Gastro-esophageal reflux disease or heartburn is a common disorder that affects almost 25% to 40% of the U.S. population (83, 84). Several studies have shown that it is positively associated with obesity (85, 86). This association can be explained by the findings that both obesity (87, 82) and reflux disease (88, 89, 90, and 84) are caused by the same type of diet that consists of soft drinks and foods that are high in fat and carbohydrates and low in fiber. It is therefore not surprising that the prevalence of obesity and gastro-esophageal reflux disease increased in tandem between 1970s and 1990s (Table 2). People with heartburn often learn how to avoid foods and beverages that cause heartburn (84). However, successful treatment of heartburn could free some individuals to consume such offending foods and beverages and gain weight, or free others who are already overweight from changing their eating habits and losing weight. The findings that long term use of proton pump inhibitors is associated with undesired weight gain (91), and that overweight people are more likely to use proton pump inhibitors than people of normal weight (92), lend support to such an argument.

Progress in antacid therapy began shortly before the obesity epidemic. Until the early 1950s antacid therapy consisted mainly of a milk diet and alkaline powders (93, 94). Modern antacid therapy began in the early 1960s with the use of Aluminum/Magnesium antacid tablets (93) which neutralize excess gastric acid. Subsequent research into the mechanism of acid secretion yielded pharmacological antacids in the form of Histamine-2 receptor antagonists in the 1970s and proton pump inhibitors in the 1980s (94), which are more effective than chemical antacids because they act in a more sustained fashion by preventing acid production (94). The improvement in the effectiveness of acid suppression was accompanied by a dramatic increase in the use of antacids for the treatment of gastric ulcer between the 1970s and the 1990s (table 30). A temporal relationship between the increase in obesity on one hand and the prevalence of reflux disease (Table 2) and antacid therapy (Table 3) on the other, gives additional support for the role of antacids in the development of the obesity epidemic.

Helicobacter pylori and body weight: Helicobacter pylori is a common pathogen that plays an etiologic role in the development of gastritis and peptic ulcer (95). The prevalence of infection with H. pylori in the United States was estimated to be 30% in 1960 but declined to about 20% in 1990 (96). There was a parallel decline in the prevalence of peptic ulcer between the 1960s and the 1990s (Table 2). The decrease in the prevalence of infection with H. pylori could be attributed, at least in part, to its unintentional eradication through the large scale use of antibiotics (95) and antacids which increased before and during the obesity epidemic (Table 3).

Numerous clinical studies have consistently shown that eradication of H. pylori results in weight gain (97, 98), suggesting that there could be a negative relationship between H. pylori infection status and body weight. However, epidemiological studies have failed to show such a relationship in the general population (99). One explanation for the lack of an association between H. pylori infection and lower body weight could lie in a recent study which has shown that the success rate for H. pylori eradication is significantly lower among overweight individuals than among individuals of normal weight (100). Therefore, the unintentional eradication of H. pylori through the use of antibiotics between the 1960s and 1990s may have been more successful among individuals of lower body weight, thus erasing any association between H. pylori infection and lower body weight. Such a scenario leaves open the possibility that unintentional eradication of H. pylori may have contributed to the U.S. obesity epidemic.

Diarrhea and Body Weight: Diarrhea is an important cause of morbidity and mortality worldwide. In the 1950s, diarrhea represented 16% of all illnesses reported for children in the United States (101), whereas among adults, diarrhea represented 1.5% of all hospitalizations between 1979 and 1995 (102). Diarrhea affects body weight through its detrimental effect on nutritional absorption, causing a loss of 500 calories per day (103). An episode of diarrhea of more than a few days' duration can cause significant weight loss (103). Therefore it would be reasonable to suggest that there would be a negative relationship between diarrhea and body weight and that eradication and treatment of diarrhea could lead to weight gain in the general population. Early advances in the fight against diarrhea came through public health measures such as sewage disposal, water treatment and food safety (104). Nevertheless, diarrhea remained a serious problem because treatment of diarrheal diseases lacked scientific basis. Withholding food and intestinal purge, which
exacerbated nutrient loss, were often part of the treatment in the early part of the twentieth century (105). The use of oral antibiotics in the 1950s was effective in improving symptoms and reducing the excretion of pathogens into the environment (106). Therefore, the increase in the use of antibiotics between the 1960s and the 1970s, as well as between the 1980s and the 1990s (Table 3) may have had a role in curtailing the incidence of diarrheal diseases in the United States. Improved knowledge of electrolyte physiology and glucose transport in the 1950s and 1960s led to the development of oral rehydration therapy which reversed nutritional loss associated with diarrhea (107). The treatment of diarrhea gained another modality in the 1960s with the use of anti-motility drugs which inhibited excessive bowel motility and promoted fluid and salt absorption (105). As a result of such advances in the prevention and treatment of diarrhea the childhood mortality rate improved significantly between the 1960s and 1990s (Table 2), while the number of hospitalizations for gastroenteritis among adults declined by 20% between 1979 and 1995 (102).

Advances in the understanding of lactose intolerance, were also helpful in reducing the incidence of diarrhea in the United States. Lactose intolerance, which is caused by the reduced activity of the enzyme lactase (108), is found in over two thirds of Blacks, Hispanics, Native Americans, Jews and Orientals, and in about 5-15% of Northern European Americans (109). It is an important cause of non infectious diarrhea, particularly among children (110,103). Adolescents who suffer from lactose intolerance have lower BMI than those who do not (111). Treatment of lactose intolerance in children by elimination of lactose from the diet results in significant weight gain (110). Lactose free formulas and lactase tablets which were developed in the 1970s (112) can also prevent lactose induced diarrhea.

**Discussion**

The combined effects of advances in the prevention and treatment of dental caries, malocclusion, dentine sensitivity, gastro-esophageal reflux disease and diarrhea may have contributed to the obesity epidemic by enabling Americans to ingest a greater variety of foods and beverages and absorb more nutrients, with the least likelihood of experiencing adverse consequences.

Table 4 presents the advances in the prevention and treatment of oral and gastrointestinal disorders in chronological order. Such progress, which began several decades before the obesity epidemic, reached a critical mass in the period between 1958 and 1970. It is conceivable that this critical mass may have triggered the obesity epidemic which began eight years later in 1978. The effect of this critical mass may have been delayed because many of those advances would have required time to transform into practical treatments and gain widespread acceptance. Furthermore, the effects of some treatments are likely to be slow due to the sporadic (e.g. diarrhea) or gradual (e.g. dental caries) nature of the disease. Over the ensuing decades, the combined effects of those advances may have contributed to the increase in the prevalence of obesity by decreasing the prevalence and duration of oral and gastrointestinal disorders.

If the proposed theory is valid, a slowdown in the rate of increase in the prevalence of obesity should take place, because progress in the prevention and treatment of oral and gastrointestinal diseases has also slowed down (Table 4). The prevalence of a few existing measures such as water fluoridation and antacid therapy have already reached a plateau. (Table 3) As the prevalence of those and other measures reaches saturation point, their impact on the prevalence of obesity is likely to diminish. A recent meta-analysis has concluded that the worldwide epidemic of obesity, including that of the United States has leveled off since 1999 (120). The prevalence of obesity in the United States in 2010 was 35.7%, (121) which represented a 4.8 percentage point increase since 2000. (Table 1) Such increase was considerably less than the increase in the prevalence of obesity between 1990 and 2000 which was 7.7 percentage points (Table 1). These findings lend further support to the theory that progress in the prevention and treatment of oral and gastrointestinal diseases has contributed to the United States obesity epidemic.

The present theory does not contradict the basic concept that excess energy can cause weight gain. Nor does it exclude other possible factors that may have contributed to the obesity epidemic. Rather, it explains how the American public was able to consume and absorb more caloric nutrients as a result of advances in the prevention and treatment of oral and gastrointestinal disorders.

**Abbreviations**

BMI: Body Mass Index
DMFT: Decayed, Missing, and Filled Teeth
NHANES: National Health and Nutrition Examination Survey

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Illustrations

Illustration 1

Table 1: Time Trend Comparison of the Prevalence of Obesity with Determinants of Energy Balance.

<table>
<thead>
<tr>
<th>Time Period (year)</th>
<th>Prevalence of obesity (a)</th>
<th>Consumer price index (b) of Cereals and bakery products</th>
<th>Sugar and sweets</th>
<th>Fruits and vegetables</th>
<th>Per capita consumption of Total calories (c)</th>
<th>Calories from carbohydrates(d)</th>
<th>Calories from fats</th>
<th>Fruits and vegetables (e)</th>
<th>Carbonated soft drinks (f)</th>
<th>Television viewing (g)</th>
<th>Occupational energy expenditure (h)</th>
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</thead>
<tbody>
<tr>
<td>1960</td>
<td>13.4</td>
<td>37.1</td>
<td>30.5</td>
<td>37.8</td>
<td>1996</td>
<td>43.9</td>
<td>36.5</td>
<td>574.5</td>
<td>22.2</td>
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<td>1444</td>
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<td>1970</td>
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<td>83.9</td>
<td>90.5</td>
<td>82.1</td>
<td>1981</td>
<td>44.3</td>
<td>36.4</td>
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<td>149.0</td>
<td>2232</td>
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<td>33.7</td>
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<td>32.7</td>
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<td>1990</td>
<td>23.2</td>
<td>188.3</td>
<td>154.0</td>
<td>150.3</td>
<td>2248</td>
<td>50.3</td>
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<td>2000</td>
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</table>
a) Percent of U.S. adult population with BMI of greater than or equal to 30.
b) Annual average.
c) In kilocalories.
d) As percentage of total calories.
e) Pounds per year.
f) Regular (sugar containing), gallons per year.
g) Minutes per household per day.
h) Daily expenditure in calories
Illustration 2

Table 2: Time Trend Comparison of the Prevalence of Obesity with Selected Disorders of the Alimentary Canal.

<table>
<thead>
<tr>
<th></th>
<th>1960s</th>
<th>1970s</th>
<th>1980s</th>
<th>1990s</th>
<th>2000s</th>
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<td>Obesity (a)</td>
<td>13.9</td>
<td>14.8</td>
<td>18.9</td>
<td>27.4</td>
<td>34.6</td>
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<tr>
<td>Dental caries (b)</td>
<td>18.0</td>
<td>16.0</td>
<td>12.5</td>
<td>9.2</td>
<td>7.1</td>
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<td>Gastro-esophageal reflux disease (c)</td>
<td>69</td>
<td></td>
<td>217.5</td>
<td>354</td>
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<tr>
<td>Peptic ulcer (d)</td>
<td>67.2</td>
<td>45.4</td>
<td>30.5</td>
<td>13.7</td>
<td></td>
</tr>
<tr>
<td>Diarrhea (e)</td>
<td>32.8</td>
<td>16.2</td>
<td>6.3</td>
<td>6.8</td>
<td></td>
</tr>
</tbody>
</table>

a) Percent of U.S. adult population with BMI greater than or equal to 30.
b) Average number of decayed, missing and filled teeth in adults.
c) Proportional rates per 10,000 hospitalizations.
d) Physician visits per 1,000 population
e) Mortality rates per 100,000 live births.
Illustration 3

Table 3: Time Trend Analysis of Preventive and Therapeutic Procedures of Possible Relevance to Weight Gain.

<table>
<thead>
<tr>
<th>Time period</th>
<th>1950s</th>
<th>1960s</th>
<th>1970s</th>
<th>1980s</th>
<th>1990s</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obesity (a)</td>
<td>13.9</td>
<td>14.8</td>
<td>18.9</td>
<td>27.4</td>
<td></td>
</tr>
<tr>
<td>Water Fluoridation (b)</td>
<td>15.8</td>
<td>35.8</td>
<td>48.8</td>
<td>54.6</td>
<td>56.1</td>
</tr>
<tr>
<td>Desensitization Therapy (c)</td>
<td>2.2</td>
<td>12.2</td>
<td>33.9</td>
<td></td>
<td>49.6</td>
</tr>
<tr>
<td>Orthodontic Treatment (d)</td>
<td>8.8</td>
<td>20.0</td>
<td>53.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antacid Therapy (e)</td>
<td></td>
<td>41.1</td>
<td>42.7</td>
<td>75.4</td>
<td>72.2</td>
</tr>
<tr>
<td>Antimicrobial Therapy</td>
<td>123.2(f)</td>
<td>192.(f)</td>
<td>87(g)</td>
<td>110(g)</td>
<td></td>
</tr>
</tbody>
</table>

a) Percent of U.S. adult population with BMI greater than or equal to 30.
b) Percent of U.S. population exposed to fluoridated water.
c) Total number of treatments in millions.
d) Total number of visits to the orthodontists’ offices in millions.
e) Percent of gastric and other ulcers treated with antacids.
f) Total number of retail prescriptions for oral antimicrobials (in millions).
g) Total number of oral antimicrobials prescribed by office based physicians (in millions).
Illustration 4

Table 4: Chronology of Advances in the Prevention and Treatment of Oral and Gastrointestinal Disorders with Relevance to Weight Gain.

<table>
<thead>
<tr>
<th>Decade</th>
<th>Advances</th>
</tr>
</thead>
<tbody>
<tr>
<td>1920s-1930s</td>
<td>Advances in sewage disposal, water treatment, food inspection and public health educations bring down the incidence of infectious diarrhea. (107,104).</td>
</tr>
<tr>
<td>1940s</td>
<td>Successful treatment of dentine sensitivity with fluoride paste is reported for the first time in 1943. (77). Water fluoridation begins as a pilot project in Grand Rapids, Michigan in 1945. (113). Widespread use of DDT reduces the population of houseflies, a common vector of food borne diarrhea (114).</td>
</tr>
<tr>
<td>1970s</td>
<td>Ensure, the first lactose free medical nutritional product is introduced in 1973. (117). Cimetidine, the first H2 receptor antagonist is introduced in 1976. (118).</td>
</tr>
<tr>
<td>1980s</td>
<td>FDA approves the first proton pump inhibitor, Omeprazole in 1989. (119).</td>
</tr>
</tbody>
</table>
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