Obesity Research and Resources

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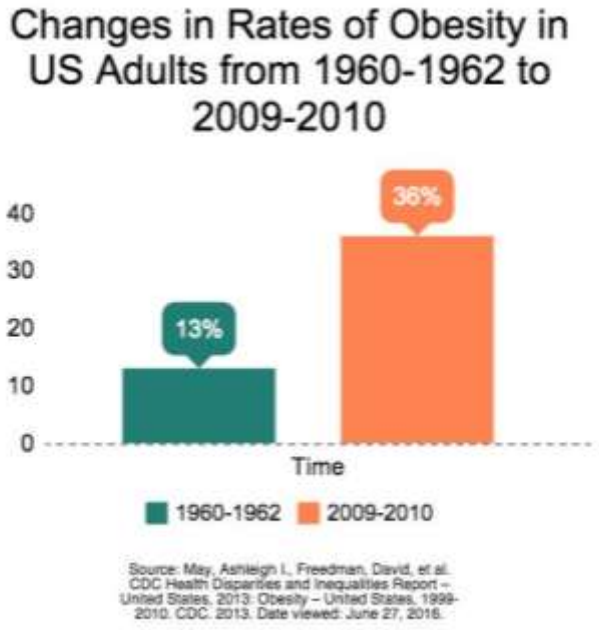
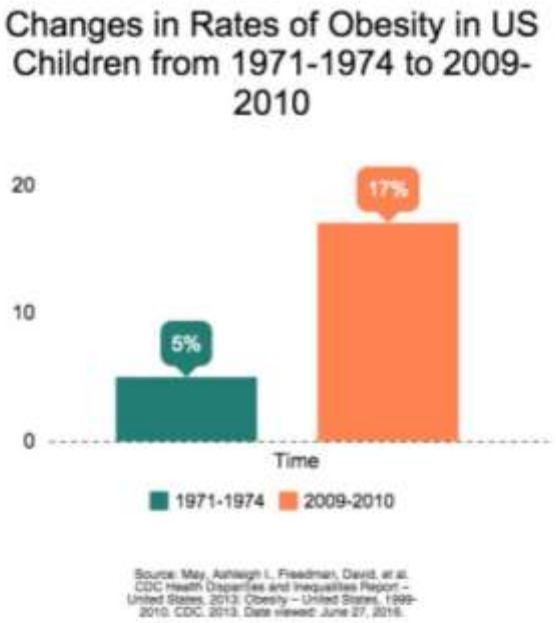
Obesity – Current Condition

In the United States and around the world obesity and obesity-related diseases are of considerable concern. The body mass index (BMI), in which an individual's weight is divided by their height squared, is a typical method to measure obesity. A score of 30.0 or greater is considered obese.[1] While this test is not fail proof, it offers a quick and cheap option to screen for obesity and follow up with more comprehensive diagnostic tests.



Obese individuals are at risk for a variety of health problems including[2]:

- Type 2 diabetes
- Cardiovascular Disease (CVD)
- Stroke
- High blood pressure
- Arthritis
- Breathing problems
- Some cancers
- Early puberty[3]
- Metabolic syndrome[4]



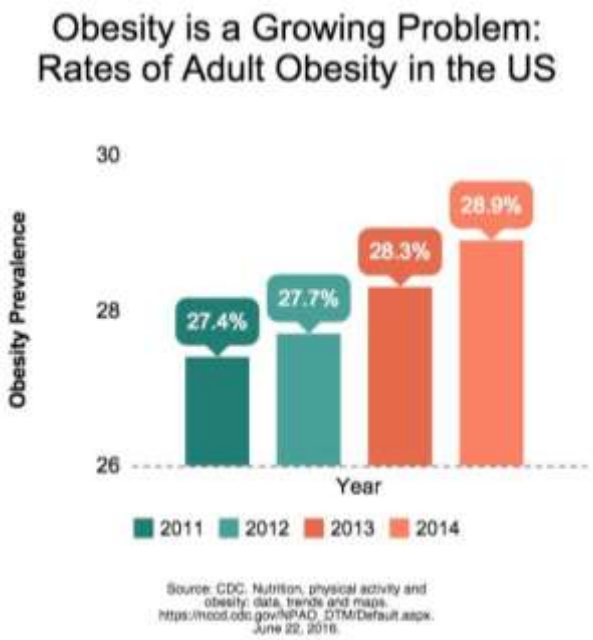
Population surveys show that the prevalence of obesity is rising. A report from the Centers for Disease Control and Prevention (CDC) found that since 1960, the prevalence of adult obesity in the United States has

nearly tripled, from 13% in 1960–1962 to 36% during 2009–2010.[5]

Childhood obesity is also a major concern. The same CDC report found that since 1970, the prevalence of obesity has more than tripled among children, from 5% in 1971–1974 to 17% in 2009–2010.[5]

The problems of obesity are also seen globally, with estimates from the World Health Organization (WHO) in 2014 finding that more than 1.9 billion adults were overweight and of those, 600 million were obese. The same WHO estimates found the worldwide prevalence of obesity more than doubled between 1980 and 2014.[6]
*Above statistics are from the Centers for Disease Control and Prevention's (CDC) Data, Trends, and Maps of Obesity[7]

The growing prevalence of obesity has increased momentum to study this disease, its impacts, and how to work towards prevention. Initial findings focused on lifestyle changes: eating more nutrient-dense foods and less energy-dense foods, in combination with more exercise. However, more recently studies indicate that there are also genetic and additional environmental risk factors for obesity. These studies have been growing in strength, and there is a rising need for more obesity-related research.



Environmental Contributors

Lifestyle Factors

Diet and exercise demonstrate a strong association with obesity. An unhealthy diet of energy-dense foods along with sedentary behavior is linked to an increased risk for obesity.[6] U.S. society has promoted an "obesogenic environment" that fosters easy and cheap access to unhealthy foods combined with the changes in the built environment that require greater car use, more sitting while working and relaxing, and overall less physical activity.[8] In addition, our communities have set up an environment in which walking and other active transportation is difficult.

Studies also relate sleep loss and stress to obesity. Sleep loss and chronic stress can lead to metabolic dysfunction through changes in the Hypothalamic-Pituitary-Adrenal (HPA) axis[9] and increased glucocorticoid[10] release. Changes in the HPA axis alters an individual's glucose storage, increases fat storage, and increases glucocorticoid stress hormones. Glucocorticoid excess causes unwanted inflammation as well as alters the release of glucagon, insulin, and adiponectin hormones, all which increase fat deposits.[11] The strong association between individuals working night shifts and increased body weight indicates the importance of sleep to prevent obesity. One study found that women who reported

eight or more night shifts per month have a 3.9 times increased risk for obesity.[12] Lack of sleep has been associated with increased desire for calorie-high foods[13] as well as low sleep quality,[14] both of which are significantly associated with obesity.

Toxic Chemical Exposures

Less understood but increasingly studied are the impacts of toxic chemicals on obesity. Chemicals that promote fat development are referred to as **obesogens**. According to the National Institute of Environment Health Sciences (NIEHS), these exposures may not cause obesity directly, but can increase an individual's susceptibility for gaining weight, especially when exposure occurs during development.[15]

The following table lists some of the most studied chemicals with a suspected link to obesity. Many chemicals have only been tested in the lab on rodents, and human evidence is still lacking. Additional problematic chemicals may be missing from this list because they have not been studied yet.

Possible Obesogenic Chemicals

Information below comes from following source unless otherwise indicated: Heindel, Jerrold J., Newbold, Retha, et al. Endocrine disruptors and obesity. *Nature Reviews*. November 2015; 11(11): 653-661.[16] The "strong", "good", and "limited" categories are taken from the CHE Toxic and Disease Database structure.[17]

Chemical	**Sources**	**Effects**	**Notes**
DDE (strong)[18]	DDT is a pesticide used to kill mosquitos; the body metabolizes it into DDE	Prenatal DDE is associated with increased infant growth	
Air Pollution (strong)	Incomplete combustion of fuels (diesel exhaust and biomass)	Increases fat and inflammation in adolescents whose mothers were exposed	Both lab and human studies
Cigarette Smoke (strong)	First-hand and Second-hand	Smoking while pregnant is strongly associated with childhood obesity	Both lab and human studies
Flame retardants (good)	Chemicals applied to furniture, electronics, children's clothing,	Increased rate of fat accumulation	One study found increased BMI of 7 year old boys whose mothers were exposed during

	foam, and insulation[19]		pregnancy[20]
Pesticides (good)[21]	Chemicals used to control weeds, insects, and other unwanted organisms; used in homes as well as in agriculture	Cause abnormalities in lipid metabolism; promote obesity in response to increased dietary fat	

The following chemicals are found to have limited association with obesity:

- Tributyltin
  - Fungicide in paints and polyvinyl chlorides (PVC) piping
  - Lack of human studies
- Bisphenol A (BPA)
  - Plastics and epoxy resins
  - Studied extensively in humans and labs with conflicting results
- Phthalates
  - Plasticizers, adhesives, personal care products; make consumer products softer
  - Associations depend on type of phthalate and age of exposure
- Polychlorinated biphenyls (PCBs)[22]
  - Coolants fluids
  - Mixed findings in both lab and human studies; strong association with diabetes[23]
- Perfluorooctanoic acid (PFOA) and perfluorooctanoate sulphonate (PFOS)
  - Components of lubricants, nonstick coatings, and stain-resistant compounds
  - Tests are inconclusive on humans and in labs
- Diethylstilbestrol (DES)
  - A synthetic estrogen prescribed to pregnant women to prevent miscarriages[24]
  - Studies are predominately from lab[25]

Genetics and Obesity

The genetic contributions of obesity are still largely understudied, leaving much unknown regarding how they work and their effects. The general agreement is that obesity may result from complex interactions between multiple genes and the environment. This means that one gene is rarely the cause for obesity. [26] Genetic components of obesity may result in different responses to the same environment, some protective and some harmful. Types of genetic factors include variations that influence behaviors, such as strength of appetite or energy expenditure, or that influence the metabolism, such as diminished capacity to use

fat or increased tendency to store fat.[25] There is still debate regarding the usefulness of genetic screening for obesity, since we cannot change our genes.

Epigenetics of Obesity

Epigenetics is a set of mechanisms that can turn on or off particular genes or sections of chromosomes. These changes are maintained as the cell divides and replicates.[27] As the fetus develops *in utero*, nutrients from the mother's diet can influence which genes are turned on and off. This process explains why pregnant women encountering a famine during early gestation have babies with higher risks of developing obesity later in life. When the mother is malnourished, genes involved in the baby's appetite regulators, pancreatic development, and obesity-associated genes can be affected. Maternal obesity itself also increases the risk of developing insulin resistance and has been associated with an increased risk of large gestational weight, fat accumulation, and type 2 diabetes in offspring.[28]

Endocrine disrupting chemicals are thought to interfere with fetal programming by affecting gene regulation and gene expression (epigenetics) during pregnancy. When pregnant mothers are exposed to EDCs, they can enter the bloodstream and alter the expression of genes in the developing fetus. The environmental exposures that the fetus encounters *in utero* have the chance of becoming persistent alterations in the individual's epigenome, leading to increased risk of obesity across multiple generations.[29] The idea of epigenetics and its link to obesity remains an active area of research.

Mechanisms of Obesity

This area of study is still under heavy research and many aspects remain unknown. What is below represents the basic understanding of current knowledge and studies.

Obesity in a Nutshell

The main idea for obesity is that it is a chronic state of inflammation and this inflammation causes many systems to act irregularly. The endocrine system of obese individuals tends to produce excess estrogen and leptin due the presence of excess fat cells. Individuals who experience obesity also tend to have dysfunctional glucose and lipid metabolism, insulin resistance, metabolic syndrome, and/or high levels of fat particles in their blood.[25] The excess fat cells from obesity are likely to induce insulin resistance, resulting in type 2 diabetes, which is why obesity is a risk factor for type 2 diabetes.[23]

The way inflammation occurs during obesity is by promoting the formation of visceral adipose tissue. This type of tissue surrounds the abdominal organs and increases the risks of impaired glucose and lipid metabolism, insulin resistance, cardiovascular disease, hypertension, and some cancers. The presence of visceral adipose tissue results in further inflammation due to activation of pro-inflammatory antibodies.[30] This cycle maintains chronic inflammation and potentially leads to insulin resistance.

Endocrine Disrupting Chemicals and Hormones Related to Obesity

Many of the chemicals discussed in the previous section were part of a group called endocrine disrupting chemicals (EDCs). These chemicals interfere with the human body's hormones disrupting their process and effects. One way obesogenic EDCs are thought to inappropriately stimulate fat storage and negatively impact the metabolism is by disrupting the hormones involved in appetite control.[31] Leptin from the adipose tissues, insulin from the pancreas, ghrelin from the stomach, and additional hormones from the gut are key players affecting appetite.[32] These various hormones are vulnerable to EDC's disrupting influence and explain one mechanism between toxic chemicals and obesity.

Vulnerabilities in Obesity

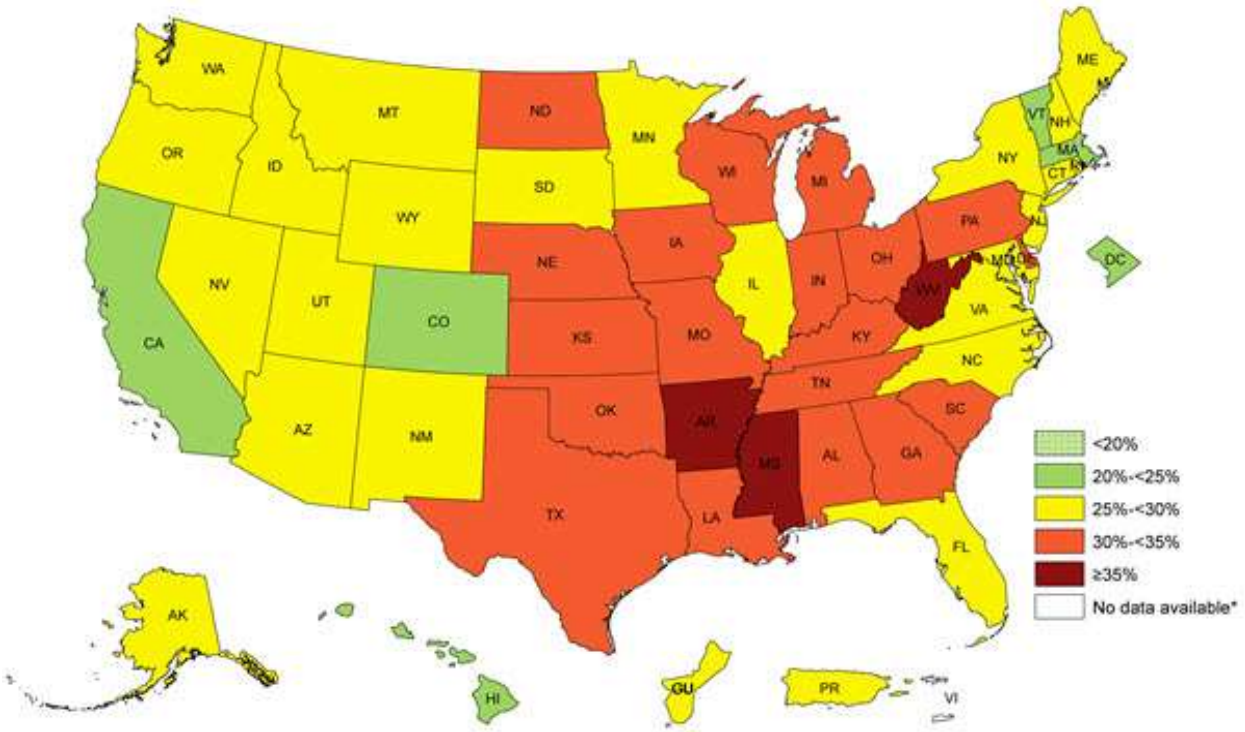
Vulnerable Windows in Human Development

While still under review, it is suspected that the most vulnerable period for obesity risk is during times of hormonal change. Early development is one of the most crucial phases of hormonal change, sparking closer examination of metabolic programming. Exposures surrounding pregnancy can have lifelong impacts on the fetus. It is thought that exposure to obesogenic toxicants during the development of metabolic tissues, such as the adipose tissues, the liver, and the pancreas, can lead to an increased susceptibility to childhood and adult obesity. Similarly, laboratory studies have shown that nutritional factors experienced during development can affect obesity risks later in life. For example, if pregnant women experience a famine during early gestation, their babies are more likely to develop obesity, raised lipid levels, and coronary heart disease in mid-life.[33]

Vulnerable Populations in Obesity

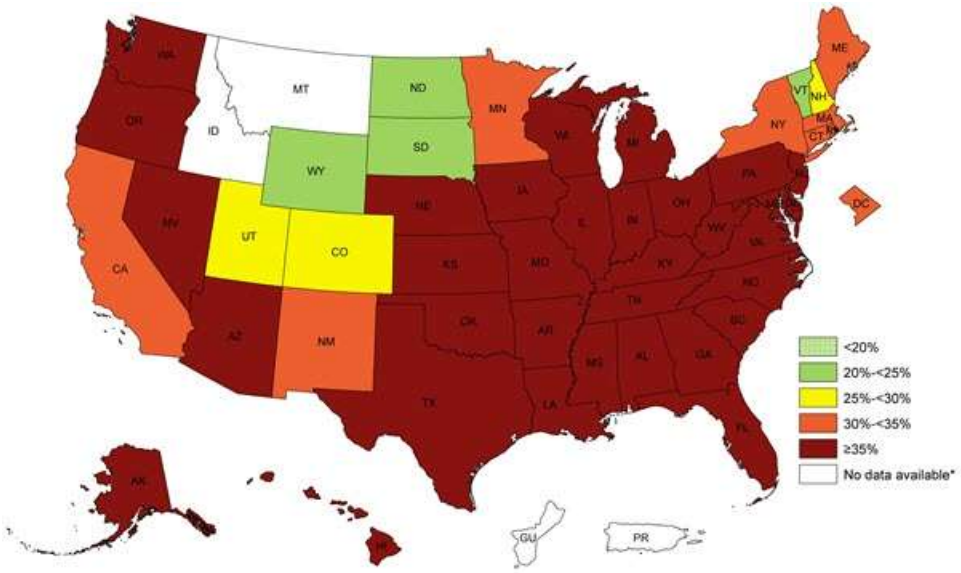
The prevalence of obesity throughout the world is widespread, however, some groups are affected more than others. One key example of this is the regional differences across the United States. As seen through the map below, the Midwest (30.7%) and the South (30.6%) experience a disproportionate amount of obesity as compared to the Northeast (27.3%) and the West (25.7%).[34]

Prevalence estimates for adult obesity, 2014[34]

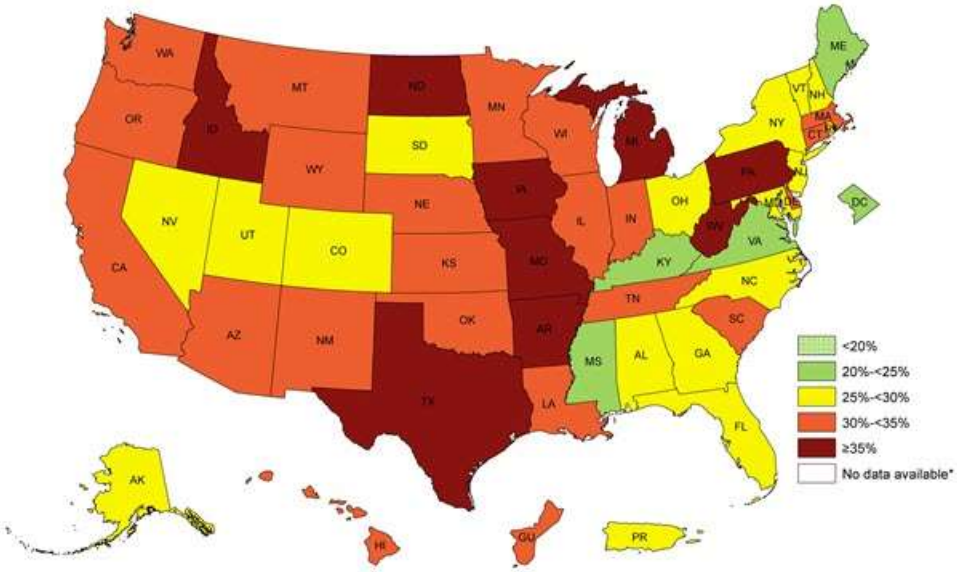


In addition to regional disparities, there are also ethnic disparities, with African Americans and Mexican-Americans experiencing a disproportionate amount of obesity in the United States. A report from the Centers for Disease Control and Prevention (CDC) found that large racial differences have remained consistent since their survey in 1988-1994 and that these differences persist even after controlling for differences in family income.[35] The maps below offer a visual representation of the ethnic disproportionality for obesity in the United States.

Prevalence of Self-Reported Obesity Among Non-Hispanic Black Adults, 2012-2014[36]



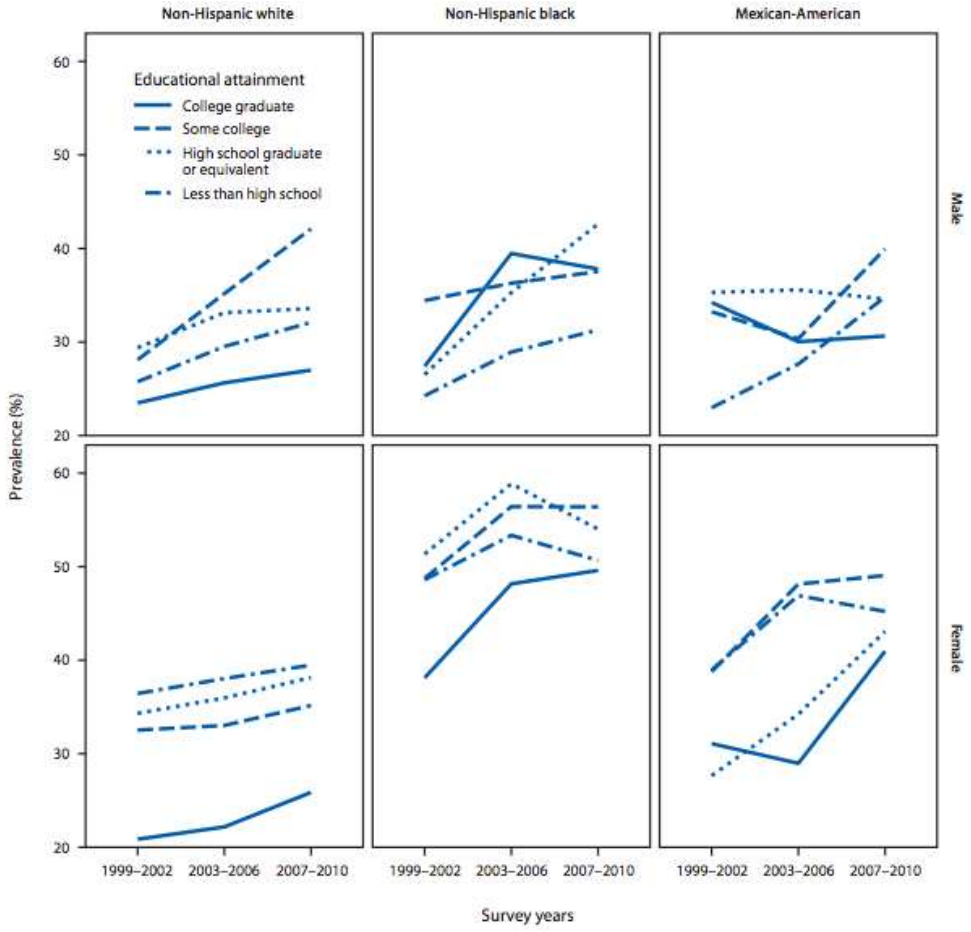
Prevalence of Self-Reported Obesity Among Hispanic Adults, 2012-2014[37]



Socioeconomic status and educational attainment are also determinants of obesity risk. The same 2011 CDC report stated that among the white population there is an inverse association between a family's income and their obesity risk.[35] In other words, the greater the income, the smaller prevalence of obesity and the smaller the income, the greater prevalence of obesity. This could be due to the higher expense of fresh fruit and vegetables, low cost of energy-dense and nutrient-poor foods, presence of food deserts within low-income areas, as well as lack of a built environment encouraging physical activity. However, the CDC study did not find a similar association between income and obesity among African Americans and Mexican-Americans.

An inverse association was also found for educational attainment and obesity among both men and women.[5]

Prevalence of obesity among adults aged ≥23 years, by educational attainment — National Health and Nutrition Examination Survey, United States, 1999–2010[5]



Medical Costs of Obesity as a Percentage of All Medical Costs

Obesity-Related Costs (21%)
All Other Health Expenditures (79%)

Source: Cawley, John, Meyerhoefer, Chad. The medical care costs of obesity: An instrumental variables approach. Journal of Health Economics. October 2011; 31: 219-230.

The Path Forward

Economic Concerns

If the severe health risks of obesity are not enough to propel society forward towards

preventing the disease, the economic burden of obesity is another driving factor. Studies estimating costs of obesity-related illness in the United States are in the billions. Each approach has produced variable estimates, one from 2008 measuring the cost at $147 billion per year[38] and another in 2005 finding the costs to be $190.2 billion per year, which was 20.6% of U.S. national health expenditures for that year.[39]

These estimates do not include the wide range of indirect obesity costs, such as work absences, decreased productivity while at work, and premature mortality and disability.[40] An economic review of obesity discovered that obesity has overtaken cigarette smoking as the most costly and detrimental preventive disease.[25]

Food Industry Regulations

The main goal when writing food industry regulations is to make the healthy choice the easy choice. This means making healthy foods more accessible and providing individuals with all the information they need to choose the healthier option. Workplaces and schools can offer healthy and sustainable food choices (such as fruits, vegetables, and whole grain options), eliminate industrially produced trans fats, and label menu items to allow individuals to make informed eating choices.[41] Due to the presence of food deserts, incentives to supermarkets and farmers' markets would benefit underserved areas and provide greater access to healthy food options. Nutritional information and caloric content in restaurant and fast food menus can also be required to allow individuals to make more informed decisions about what they are eating. States and communities can help with the promotion of healthier foods to combat marketing from energy-dense food companies.[42] Finally, the United States can change its policy from incentivizing production of sweets, fats, and meats, and instead subsidize healthier options like fruits and vegetables.[43]

Ethical Concerns

Obesity is a complex issue that is intertwined within multiple sectors of society. To best prevent the development of obesity and to support those who are obese, community responsibility should be targeted instead of individual choice. As seen through the discussion of the obesogeneic environment, U.S. policy subsidizing unhealthy foods, and many other policies, society is pushing its members towards obese lifestyles. This illustrates a system that incorporates less individual choice and more communal decision-making. In order to have the largest impact on obesity, community action is necessary to provide healthy food options, encourage physical activity, educate the public, and regulate the foods and chemicals that are associated with obesity.

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