

Factors Leading to Overweight and Obesity Point to Complex Combination of Forces Negatively Impacting the Health of Alaskans and the U.S. Population

***Abstract:** The conditions of being obese and overweight are serious and multi-dimensional health problems facing Alaska and the United States. The so-called “obesity epidemic,” and its negatives consequences, are seen in chronic disease outcomes and economic costs estimated at \$147 billion annually.¹ Though Alaska has distinct population and geographic differences from the country, residents of Alaska and the 49 other states face potential exposures to an interplay of some of the primary contributing factors to obesity: high caloric intake and poor nutrition, sedentary lifestyles, social and environmental conditions, hormonal and metabolic mechanisms, and some predisposing genetic factors. Strategies designed to reduce the prevalence of both conditions are driven by current evidence-base research that seeks reverse trends that can be controlled by interventions.*

Introduction: The case opens with Mrs. Rose expressing concern about health conditions tied to weight and related health issues in three generations of her family. She has good reason to be concerned, given obesity’s links to chronic diseases such as type 2 diabetes and cardiovascular disease. Her conversations with her doctor and a public health worker reveal multiple influences interacting with each other, and together leading to what health experts are calling an obesity epidemic, in small cities like Fairbanks and across the country. Before the predisposing factors can be distilled, this paper will first summarize characteristics of obesity and overweight and their prevalence in the United States and Alaska.

Obesity is “a condition of abnormal or excess fat accumulation in adipose tissue, to the extent that health may be impaired.”³ The U.S. Centers for Disease Control and Prevention (CDC) defines the terms obesity and overweight as “ranges of weight that are greater than what is generally considered healthy for a given height.” The terms encompass weight ranges that have been shown to increase the chance of some diseases and health problems.² For an adult, overweight is defined as a body mass index (BMI) of 25-29.9, while obese describes an adult with a BMI of 30 or more. However, other measurements of obesity exist, including waist-to-hip ratio (WHR), waist circumference (WC), and waist-to-height ration (WHtR).³ Both conditions increase the risk for health problems not mentioned already—respiratory diseases, kidney diseases, musculoskeletal disorders, mental health problems—all of which can reduce the quality of life, lead to early death, and substantially increase health care costs.³

Prevalence of Obesity in the USA: The prevalence of obesity in the United States has been rising nationally since 1976, and dramatically since 1991 (see appendix 1). The CDC’s National Health and Nutrition Examination Survey found that 32.8% of adults were obese in 2007-2008, and 68% of adults were either overweight or obese combined.⁴ The prevalence of obesity and overweight is impacting races differently also, with minorities experiencing a higher prevalence of obesity.

Table 1: Prevalence in adults of obesity and overweight, United States, 2007-2008.⁴

Categories/Age	All	Non-Hispanic White	Non-Hispanic Black	All Hispanics	Mexican American
BMI \geq 30/ Age \geq 20	33.8%	32.4%	44.1%	38.7%	40.4%
BMI \geq 25/ Age \geq 20	68%	66.7%	73.8%	77.9%	78.8%

Among adult American Indians/Native Alaskans in 2009, 34% were obese, and 69.8% were both obese and overweight.⁵

Obesity in Alaska: Despite having world-class outdoor recreational activities readily accessible to all residents and having a reputation for physical hardiness glorified in multiple reality TV shows, Alaska is more akin to Appalachia or the Southeast in terms of weight issues impacting its population. In 2009, Alaska ranked 22nd heaviest nationwide, according to the CDC, with one newspaper saying it was the “fattest this side of Texas.”⁶ The state’s Department of Health and Social Services reports that obesity is a serious health concern in Alaska, with common pathways to obesity clearly identified.^{7,8} The state’s Obesity Prevention and Control Program reports⁷:

- 65% of Alaskan adults are overweight or obese,
- American Indian/Alaska Native adults are significantly more likely to be obese (31% vs. 25%) and above a normal weight — that is, overweight or obese — (69% vs. 64%) compared to white adults,
- 26% of Alaskan high school students are overweight or obese, with 12% classified as obese,
- 26% of Alaskan adults do not meet the physical activity recommendations,
- 26% of adults watch more than 3 hours of TV a day,
- 1 in 5 Alaskans do not engage in physical activity during spare time, with lowest income groups (<\$15,000 annual income) having the least activity,
- 50% of high school students watch 3 or more hours of TV a day or spend time using a computer for non-academic purposes,
- 3 in 4 Alaskans do not eat the recommended 5 or more servings of fruit or vegetables daily.

Areas in Alaska hardest hit by both conditions are outside of the main cities—regions defined as “the Bush,” where Native Alaskans make up the majority of populations in more than 150 villages, towns, and cities.

Factors Leading to Obesity Up North: The State of Alaska's February 2010 report on overweight and obesity relies on data gathered from the CDC’s Behavioral Risk Factor Surveillance System (BRFSS) and Youth Risk Behavior Surveillance System (YRBSS) surveys of adults and youth. The report identifies the primary cause of both conditions as the imbalance between nutrition and physical activity.⁷ That imbalance, according to the report, is shaped by: personal preferences, norms (family, cultural, community), nutritional knowledge, food availability from multiple sources (stores, schools, work, restaurants).⁷ The principle predisposing factors identified by the program are: physical inactivity, poor nutrition, inadequate consumption of fruits and vegetables, excessive television and computer usage time, excessive

consumption of sweetened beverages, and a nutritional environmental at stores and restaurants that contributes to a poor diet.⁷ A full 95% of Alaskan adults state that individuals and parents have “some or a lot of responsibility for addressing obesity.”⁷

What Is the Best Evidence:

Literature on the causes of the obesity epidemic point to causal factors identified in multiple studies. Changes in nutrition are identified as a major factor, in the United States and globally. These shifts involve higher energy intake, greater consumption of fats, more added sugars in foods, greater saturated fat intake, increased consumption of industrially produced animal protein, less consumption of complex carbohydrates and dietary fiber, and reduced intake in fruits and vegetables.^{3,9}

Table 2, summary of evidence on factors that might promote and protect against obesity (Source: WHO).³

Strength of evidence	Decreased risk	Increased risk
Convincing	Regular physical activity	Sedentary lifestyle
Probable	High dietary intake of fiber	High intake of energy-dense foods
	Home and school environments that support healthy food choices for children	Adverse socioeconomic conditions in developed countries
Possible	Breastfeeding	Large portion sizes
	Low glycemic index foods	
Insufficient	Increased eating frequency	High proportion of food prepared outside the home (developed countries)
		Rigid restraint/periodic disinhibition eating patterns
		Alcohol

Other factors have been identified as having a probable cause in obesity, in addition to insufficient physical activity due to work patterns, lack of exercise, and more time spent in front of screens and televisions.³ These include:

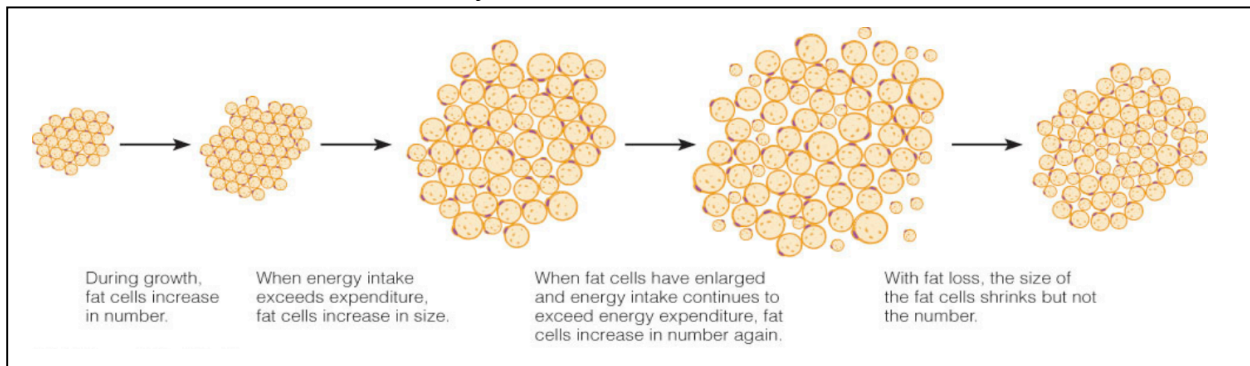
- **Social inequality:** Inequitable access to healthy foods as determined by socioeconomic factors could influence diet and health. Thus, energy dense but nutrient poor foods often become the best way to provide daily calories by lower-income people. The food market environment plays a key role here.³
- **Prenatal/post-natal health:** Adverse environments during *in utero* or postnatal periods may be a cause of obesity. Exposure to elevated or excess nutrients before birth is linked with an increase risk of obesity and metabolic disorders later in life.³

Cellular and Metabolic Pathways: Obesity and overweight also have to be understood in the context the body’s development and metabolism of fat. When humans consume excess energy, it is stored in fat cells of adipose tissues. During late childhood and puberty, the number of fat cells increases at its greatest rate. When energy levels rise, these cells enlarge, stimulating cell proliferation. However, when energy levels fall, the size of cells fall, but not their number. Thus, children and adolescents who experience hyperelastic obesity (due to number of fat cells) will carry additional risks later in life, as those with extra fat cells tend to gain weight rapidly (see table 3).¹⁰ In addition, researchers have determined that the body will self-adjust its metabolism to restore original weight, in a process described as set point theory, with energy expenditures increasing with gains and decreasing with weight loss.¹⁰ Thus those who have become

overweight will be fighting their body's metabolism to return to a pre-overweight status if they try to lose pounds.

The absence of physical activity, a problem nationally and in Alaska, also prevents the body from benefiting the speeding up of metabolism, which contributes to energy expenditure. Metabolism will remain slightly higher for several hours after prolonged exercise. Over the long term, people who practice daily vigorous activities develop more lean tissue, and metabolic rates rise, which supports continued weight loss and maintenance.¹⁰ In addition, not all people are equally predisposed to physical activity. Research has found that obese prone persons have a higher proportion of fast-twitch muscle fibers, which oxidize less lipids during exercise, and may generate feelings of fatigue easier, all which may predispose some people to be less active and gain weight.¹¹

Table 3: Fat cells can increase size by 20-fold and numbers several thousand-fold.¹⁰



Genetic Factors Genetic influences have also been identified in weight gain. Genetics research has explored the possibility of the “thrifty genotype” hypothesis, which suggests the rise in obesity is tied to contemporary environmental factors being mismatched with “energy-thrifty” genes that arose through evolution when food sources were scarce.¹² The CDC's discussion of this research notes several cases of extreme obesity due to mutations of single genes have been found, but that these account for a small fraction of worldwide cases. Research on mutations in a single gene (Melanocortin 4-receptor gene, linked to feeding behavior) show it has been strongly linked with about 5% of cases in some populations. As of October 2005, according to the CDC, single mutations in 11 genes were strongly implicated in 176 cases of obesity worldwide.¹²

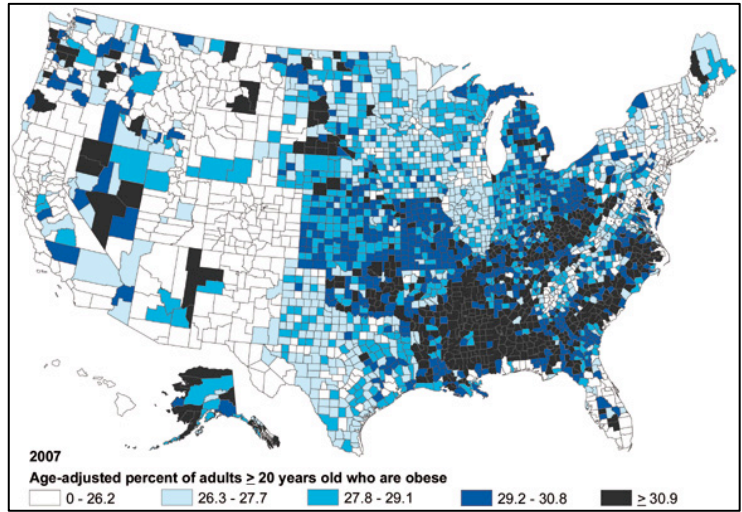
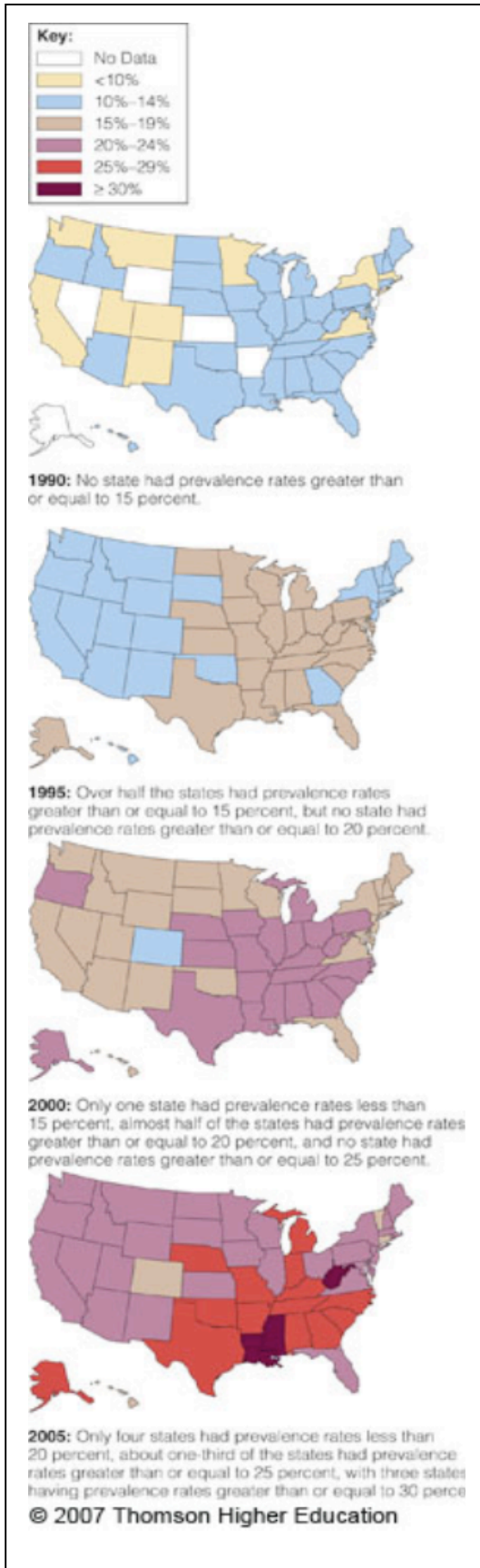
Hormones and Proteins: Researchers have identified the so-called obesity gene, called *ob*, which is expressed in adipose tissue and codes the protein leptin, which acts as a hormone in the hypothalamus and helps to suppress hunger and boost energy expenditures. Research on mice has shown those with a defective *ob* gene do not produce leptin and can weigh 3 times more as normal mice and have 5 times the body fat.¹⁰ Leptin also interacts with another protein in the hypothalamus called ghrelin, which is secreted in the stomach and serves as a regulator of energy balance by stimulating appetites and boosting efficient energy storage. It triggers the body's desire to eat, and blood levels of ghrelin normally rise before and fall after a meal. However, ghrelin levels do not appear to decline as much after a meal in obese persons or those with binge eating disorders as they do for lean people.¹⁰ It also has been suggested that ghrelin's role is to

maximize fat stores during famine, a biological advantage in a pre-industrial time but a disadvantage in an era of sedentary lifestyles and calorie-rich diets.

Back to the Case/Questions: Mrs. Rose's efforts to understand the factors leading to weight gain in her family uncovers multiple paths that converge on her, her children, and about two-thirds of those living in the United States. While genetic factors likely are playing a role in her household's experience, strategies seeking to address problems associated with obesity and overweight in Alaska are aimed at reversing the effects of other predisposing factors. The state's strategy promotes less television watching, diets with more fruits and vegetables, less consumption of sugary drinks like the soda served by Mrs. Rose, and reduced intake of high calorie foods.⁷ It is not clear how well the state's comprehensive strategy involving schools, communities, and state agencies can effectively put these ideas into action.

1. Alaska is among the most conservative states in the country, and healthy activities there are frequently associated with a political agenda of those left of center. To what degree have "healthy activities/diets" been branded as political?
2. Can any obesity intervention work when the dominant political culture and many media streams vehemently malign government intervention with extremist political discourse and vigorously promote an unregulated private sector that encourages unhealthy built environments and lifestyles (i.e., is the best intervention political change)?

Appendix 1: Rise in overweight and obesity in the United States (note that Alaska aligns with the South).¹⁰



Obesity rates by county, 2007 (Source CDC)

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Key Reference:

For information about Alaska's strategy, I would recommend consulting the state's Obesity and Prevention Control's report from 2010: Fenaughty A, Fink K, Peck D, Wells R, Utermohle C, Peterson E. *The burden of overweight & obese in Alaska*. Alaska Department of Health and Social Services. February 2010; http://www.hss.state.ak.us/dph/chronic/obesity/pubs/ObesityBurdenReport_2010.pdf.