CHANGING MEANINGS OF FAT:
FAT, OBESITY, EPIDEMICS, AND AMERICA’S CHILDREN

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Elise Paradis

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Francisco Ramirez, Primary Adviser

I certify that I have read this dissertation and that, in my opinion, it is fully adequate in scope and quality as a dissertation for the degree of Doctor of Philosophy.

Donald Barr

I certify that I have read this dissertation and that, in my opinion, it is fully adequate in scope and quality as a dissertation for the degree of Doctor of Philosophy.

John Meyer

Approved for the Stanford University Committee on Graduate Studies.

Patricia J. Gumport, Vice Provost Graduate Education

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Abstract

This dissertation falls within a tradition that investigates the making of health-related problems into social problems. Using literature reviews, document analysis, and qualitative and quantitative coding of medical publications from 1950 to 2010, I argue that both our increasingly individualistic culture and our collective faith in science fuel the current fear of obesity and lead to the expansion of the medical discourse on fat.

In Part I, I review the main medical research paradigm on obesity, which argues that fat is bad for your health, before turning to the critique of this paradigm, and show how both sides of the debate use science to justify their stance. I then combine both views to identify which educational strategies are most likely to be implemented, and efficient. The importance of stigma in the health and well-being of obese people appears to be critical to this effort.

Part II contributes a timeline for distinct but overlapping conceptualizations of bodily fat in the medical literature, and shows the massive and recent increase in medical interest in obesity. From merely an individual trait, fatness has become a medical problem (obesity), a social problem and an epidemic, and has culminated in recent years into a focus on children: the so-called epidemic of childhood obesity. This longitudinal approach to the medical literature at both the aggregate level (in the PubMed database) and in the most cited articles on obesity highlights the historical contingency of our cultural and medical obsession with fat, meanwhile identifying the role schools are expected to play.
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# Table of Contents

List of Illustrations xiv

List of Tables xv

Introduction: The Changing Meanings of Fat 1

Context 4
Conceptual Framework 10
Methods 12
Dissertation Overview 14

Part I: To Be, or Not to Be, Fat? The Complex Nature of Obesity 17

Chapter 1: To Be, Fat. The Health Effects of Obesity. 19

Methods 21

1.1 Overnutrition and Its Physiological Impacts 24

1.1.1 Body Fat as Active Tissue 24
1.1.2 Extra Calories 26
1.1.3 Syndrome X: The Metabolic Syndrome of Insulin Resistance 27

1.2 Mortality 28

1.2.1 Comparing Mortality Risks 28
1.2.2 Annual Death Toll 29
1.2.3 Years of Life Lost 30

1.3 Arthritis 31

1.4 Cancer 31

1.5 Cardiovascular Disease (CVD) 32

1.6 Diabetes and Metabolic Syndrome 33

1.7 Gestational Diabetes Mellitus and Fetal Health 35

1.7.1 Maternal Obesity and Gestational Diabetes Mellitus 35
1.7.2 Impact of GDM on Fetal Health and Future Health Outcomes 36

1.8 Mental Health Problems 37

1.9 The Economic Costs of Obesity 38

Conclusion 39

Chapter 2: To Get, and Be, Fat. Obesity in the U.S. 41

2.1 Weight Gain in the United States, 1960-2010 41

2.1.1 Adults 42
2.1.2 Children 42

2.2 Current United States’ Population Weights 46

2.2.1 Adults 46
2.2.2 Children 49

Conclusion 50

Chapter 3: Not to Be Fat. Obesity Research as Social Enterprise. 51

3.1 Skeletons in the Medical Closet – The Fat Scholar Critique 52

3.1.1 What Is the Obesity Epidemic? 54
3.1.2 Fat, Mortality and Morbidity 55
3.1.3 The Data on Weight Loss 63
3.2 Stigmatization, Discrimination, and Barriers to Care

3.2.1 Fat Discrimination in Everyday Life

3.2.2 Fat Discrimination in Health Care

3.3 Why obesity is not the new smoking

Conclusion

Chapter 4: Obesity, Its Models, Research and Policy Implications

4.1 The Causes of Obesity: Energy Imbalance and the Toxic Environment

4.1.1 Food Intake Trends

4.1.2 The Toxic Environment

4.2 Causal Models for Obesity and its Consequences

4.3 Strategies to Maximize Health

Part II: The Changing Meanings of Fat

Chapter 5: The Medicalization and Healthicization of Fat.

5.1 Medicalization and Healthicization

5.2 The Medicalization and Healthicization of Fat

Further Feminist Considerations

Conclusion: Health, for Better and for Worse

Chapter 6: Expanding Waistlines, Expansive Discourse: Obesity in the Medical Literature

6.1 The Growth and Pervasiveness of Obesity Research over Time

6.1.1 Obesity Journals

6.1.2 Obesity as a Topic of Scientific Investigation

6.2 Obesity as a Public Health Issue

6.3 The Economic Costs of Obesity

6.4 The Obesity Epidemic in Historical and Comparative Perspective

Conclusion: Growth, Healthicization, and an Epidemic of Epidemics

Part III: America’s Children

Chapter 7: Fat Kids and The Rise of Obesity as an Educational Problem

7.1 Fat Lessons, Fat Kids: Obesity, Epidemics, Education and Schools in PubMed

7.1.1 Obesity, Education and Schools

7.1.2 Childhood Obesity, Education and Schools

7.1.3 Obesity Epidemic, Childhood Obesity Epidemic, Education and Schools

7.2 What’s The Fuss All About? The Most Cited Articles on Childhood Obesity

7.4 The U.S. Government and the Role of Schools

7.4.1 Surgeon General’s Call to Action, 2001

7.4.2 Surgeon General’s Vision for a Healthy and Fit Nation, 2010

7.4.3 The White House’s Let’s Move! Program, 2009

7.4.4 H.R. 5504: The Child Nutrition Act

7.5 Singapore

Conclusion

Chapter 8: Conclusion and Future Research

Appendix I – BMI Table

Appendix II – Fifty Most-Cited Articles on Obesity
Appendix III – Important Dates in the Medicalization and Healthicization of Fat 172
Appendix IV – Articles on Childhood Obesity Coded for Section 7.2. 174
Appendix V – Coding Scheme, Most Cited Articles on Childhood Obesity and the Childhood Obesity Epidemic, ISI Web of Knowledge 177
Appendix VII – Supporters of Bill H.R. 5504 – Improving Nutrition for America’s Children Act 180
References 181
List of Illustrations

0.1 Normalized Trends, Obesity as Medical Issue and as Discourse, 1985-2009 ........................................................................................................ 7
2.1 Overweight and Obesity Rates, NHANES, 1960-2006 ................................................................. 43
2.2 Diabetes and Obesity in the U.S., 2007 .......................................................................................... 44
3.1 Percentage of Fit and Unfit Individuals in ACLS Sample, by BMI .................................. 61
3.2 Percentage of ACLS Subjects by BMI and Fitness Quintile ......................................................... 62
4.1 Calories Available per Person per Day, 1909-1998 ................................................................. 82
4.3 Ernsberger’s (2009) Model of the Relationship Between SES, Adiposity, and Adverse Health Outcomes ........................................................................ 87
6.1 Obesity In, and As Proportion of, PubMed, 1950-2009 .......................................................... 117
6.2 Obesity and Cancer, Diabetes, Cardiovascular Diseases or Childhood as Proportion of PubMed, 1990-2009 ................................................................. 118
6.5 Epidemics Discourse in Titles and Abstracts as Proportion of PubMed, 1990-2009 ........................................................................................................ 126
7.1 Obesity and Child- and Education-Related Keywords in PubMed, 1980-2009 ................................................................. 138
7.2 Obesity and Child- and Education-Related Keywords as Proportion of PubMed, 1980-2009 ........................................................................ 139
7.3 Ratio of Publications on Obesity Epidemic and Associated Keywords to Total PubMed by decade, 1980-2009 ................................................................. 140
## List of Tables

2.1 NHANES Data on Percentage of Obese Adults and Youth among US Population, and Growth Rates over Time ................................................................. 45

2.2 Median BMI and Percentage of Population by BMI, Gender and Age Group, 2007-8 ........................................................................................................ 47

3.1 Percentage of U.S. adults who use specific weight control practices by body mass index, Behavioral Risk Survey System, 2000 ........................................ 68

4.1 Part I Findings and Associated Health-Enhancing Strategies ........................................... 90

6.1 Year of Foundation and Names of Different Obesity-Related Journals ............. 115

6.2 PubMed Database Publications on Obesity and Related Keywords (Title and Abstract), Frequencies and Growth Rate Over Time, 1989-2009 ......... 116

6.3 PubMed Database Publications on Obesity and Related Keywords (Title and Abstract), Ratio to PubMed Total and Growth Rate Over Time, 1989-2009........................................................................................................ 116

7.1 Characteristic of Chosen Subsets of 34 Most Cited Articles, Childhood Obesity and Childhood Obesity Epidemic, ISI Web of Knowledge ............. 145

Appendix I: BMI Table........................................................................................................ 167
Introduction: The Changing Meanings of Fat

Over the past century, our culture’s interest in fat has escalated dramatically. Be it nutritional fat, fat as a public health problem, fat as a financial burden, fat as a biological or hereditary trait, or fat as a social construction and cultural obsession, scholars from all disciplines have participated in defining what fat is, what it means, and why and how it matters.

Why did we come to care so much about fat – our own, but also others’? Why has fat come under close medical scrutiny? In this dissertation, I argue that the rise of individualism and the growing faith in science are integral to our current cultural and medical obsession with fat. These two broad sociological forces combined to transform fat from merely an individual trait into a medical problem: obesity. In the 18th century, medical doctors turned their eyes to fat bodies. Since, fat first became a medical problem, before becoming a public health problem in need of state intervention needed to be mobilized.

The replacement of the word “fat” by the omnipresent medical terms “overweight” and “obesity” in everyday language testifies to the medicalization of fat. Both the use of “epidemic” to describe current rates of obesity and the mobilization of a violent imagery such as “the war on obesity” to describe efforts to manage the fat of the land builds highlight the role scientific authority played in turning what used to be merely an individual trait into a significant social problem.

While in the United States the federal government has not (yet?) regulated the perceived contributors to the so-called obesity epidemic (such as food production or consumption), some of its agencies have called attention to the issue, including the Surgeon General, the Department of Health and Human Services, the National Research
Council and the National Institutes of Health. Inter-governmental organizations such as the World Health Organization, non-for-profit organizations such as the American Heart Association and the American Diabetes Association, and most news outlets have either followed suit or participated in the definition of the problem, basing their support on scientific evidence.

Amid a growing medicalization of fat and public interventions to address obesity, both the literature on obesity’s adverse health effects and the public health campaigns to fight obesity have come under fire. Are obesity’s health hazards fact or fiction? Is the current “war” a response to a genuine medical emergency, or moral panic? In answering these questions, both sides of the debate – those who wage the war on obesity and those who try to undermine or transform the current attempts at regulating the “toxic environment” or fat bodies – have turned to science for support. As noted by Ian Hacking (1999), even among the most vocal social constructionists, those who would choose a herbalist over a medical doctor in fixing a broken arm are hard to find. Similarly, very few people are making pre-modern claims for or against “fat”: those who pray their pounds away and those who reject altogether the authority of science in matters of the flesh are few and far between, and more a curiosity than a mass phenomenon.

Our current explanations of obesity are too often simplistic and reductionist: we believe one’s weight to be nothing more the function of individual will and control of inputs (food) and outputs (energy expenditure). What a sociology of fat highlights are the important constraints on individual choice – both the “toxic environment” and a stratification of body weight by gender, sexuality, race, income, educational attainment –, the socio-historical contingency of the medical concern with fat, and the role that
individualistic beliefs and medicine play in the expansion of waistlines, the clash of discourses, and the mobilization to define what fat really is about.

The reasons to study fat sociologically will be clear to any Westerner who has paid attention to the news over the past five years. Obesity talk is everywhere: seen across media forms and genres. Beyond its urgency as a substantive area of inquiry, however, fat raises issues of scientific expertise, medicalization, gender, stratification, social mobilization, etc.

In this dissertation, I address three different elements in the literature on obesity. In Part I: To Be, or Not to Be, Fat? The Complex Nature of Obesity, I provide an overview of the two main paradigms in the study of fat: mainstream medical and epidemiological literatures that claim that obesity is a disease, and their critiques who argue that fat is a social construction and that most of the current fear of fat is an artifact of fat-phobia. I asked: Is obesity as a medical problem a fact, or a fiction? Based on the answer, I discuss potential interventions to maximize health and wellbeing in the American population.

In Part II: Changing Meanings of Fat, I show the evolution of the medical and public health discourse on fat over time, and argue that individualism and medicalization, as well as the rise of public health and of education as a means to solve social problems, explains this evolution. I asked: What processes turned fat into such a prominent medical and public health problem? How has the framing of bodily fat changed over time in the medical literature?

In Part III: America’s Children, I show the rise of education and schools in the medical discourse on obesity, investigate the empirical basis for assigning anti-obesity interventions to schools, and compare American and Singaporean school-based approaches.
Context

Medicine, one of American society’s central institutions, has had a major influence in the way contemporaries understand the connection between adiposity – bodily fat – and health. In this dissertation, I make a careful distinction between the medical phenomenon of fat / adiposity / obesity and the medical discourse on fat / adiposity / obesity. The first pertains to a bodily disposition, a condition of individual people. The second reflects the how fat, which was merely a bodily trait during most of human history, has become medicalized: it became perceived as within the purview of medical science, medical attention, and medical care. From 1960 to 2007, the proportion of Americans who are obese grew from 13.3% to 33.8%, a 154% growth. Meanwhile, the proportion of articles with obesity in their titles or abstracts in the medical literature has grown from 0.097% to 1.172%, a 1,100% growth. The growth in discourse was more than seven times greater than the growth in weights.

Functionalist accounts of the role of medicine in society would predict that the medical discourse on fat emerged as a direct response to two things: a growing knowledge on its causal link to disease, and increasing weights in the population. In this dissertation, I take a more skeptical look at the medical discourse, inspired by the institutionalist idea that discourses are often loosely coupled with the reality that they supposedly describe (Meyer and Rowan 1977).

The first part of the functional argument noted above, about the growing knowledge base on the pathological aspects of bodily fat, is addressed in Part I, in which I ask: Is obesity a “real” medical problem, or merely a social construction, what some authors have called a “moral panic” (Boero 2007; Campos et al. 2006a; Saguy and Almeling 2008). That increasing weights in the population may be the sole factor behind
the immense growth seen in the medical discourse over the past decade is unlikely. While it is impossible to totally deflect direct causal claims, Figure 0.1, below, suggests the extent to which these two phenomena (weight gain and discourse on weight gain) may be loosely coupled.

Figure 0.1 compares the growth in the medical discourse on obesity, childhood obesity, and obesity epidemics found in the PubMed database with the weight gain of the American population, using data normalized with baseline years 1985-91 equal 100. Normalized data on the number of articles that have “obesity,” “childhood AND obesity,” “obesity AND epidemic,” or “childhood AND obesity AND epidemic” in their title or abstract as a proportion of the total PubMed database, are compared with normalized obesity rates (among adults, and among children aged 6-11) and with the total number of publications in PubMed. On Panel A, we see how insignificant the increase in obesity rates has been since 1985 compared to the growth in discourse: the lines are barely distinguishable from the horizontal axis, dwarfed by the discourse on obesity.

Panel B excludes the two curves with the most important growth (obesity epidemic and childhood obesity epidemic in titles and abstracts) in order to allow for comparisons among lower-growth curves (note different y axes and scales). Again, on Panel B, we see discourse flying high above actual weight trends: the growth of obesity and childhood obesity publications in PubMed clearly surpasses the growth of obesity in the American population.

Admittedly, one should not expect observed frequencies of a medical phenomenon to map perfectly onto the medical discourse about this phenomenon. One would think, for example, that in the case of epidemics there should be a time lag

1 Data were normalized for 1985-9 because this five-year interval contains the first time point with non-zero frequencies for all observed variables.
between the discovery of a medical problem and its appearance in the medical literature.

Still, the magnitude of the gap, and the very large growth of obesity as a proportion of the total medical literature command sociological attention.
Fig. 0.1: Normalized Trends,\(^1\) Obesity as Medical Issue and as Discourse, 1985-2009.

\(^1\) Data was normalized for 1985-9 = 100.
Health has not always been the cultural imperative that it is today. I argue that the rise of science as a cultural authority and a growing cult of the individual have both participated in the rise of medicine, defining in turn our current preoccupations with health and with fat. It is likely that there have been fat people throughout most of history, yet it is only recently that it has been so deeply pathologized that being fat is seen as equivalent to being unhealthy: as the sign of disease. The fear of fat as a health hazard cannot be decoupled from the rise of health as a social value.

As observed by Starr (1982), Americans are singularly concerned with their individual wellbeing, and have been for quite a while. Indeed, he mentions de Tocqueville, who noted in the early 19th century the peculiar concern of Americans with health. Since, he writes, the United States has been swept by a series of popular movements concerned with improving health variously through diet, exercise, moral purity, positive thinking, and religious faith. Today, were a revived de Tocqueville to observe Americans jogging in parks, shopping in health food stores, talking psychobabble, and reading endless guides to keeping fit, eating right, and staying healthy, he would probably conclude that, if anything, the obsession is now more pronounced (Starr 1982, p. 7).

Starr is not alone in noting the rise of health in the American imagination. Many feminists have noted how De Toqueville would also notice that the bodily qualifiers “fat” or “portly” have been replaced by the omnipresent medical terms “overweight” and “obesity,” and that being overweight or obese (fat!) is now seen as a major health hazard (Rothblum and Solovay 2009).

Until very recently, as we will see, the medical cost of fat was limited to the individual. Then, in the early 1990s, economists started putting a tab to obesity (39.3 billion dollars in 1986, according to Colditz, 1992), and emphasized that obesity-related costs would be shared by all tax payers, fat and thin (see Chapters 1 and 6). In 1993,
obesity was first described as an “epidemic” in the popular press, and it had found its way into the medical press in a comment on an article about “excess body weight” (see Chapter 6). In 2007, Christakis and Fowler told us that fat runs in social networks, suggesting that fat is contagious and spreads to those who socialize with fat people (Christakis and Fowler 2007). Once discursively established as a poison for the individual and for society, fat and its purported causes may be on their way to be regulated, just like asbestos and cigarettes were in the past.

As early as 1992, before the emergence of the obesity epidemics discourse, Singapore had already started the promotion of its “Fit and Trim” program, targeted to children and implemented in schools (see Chapter 7). In contrast, the federal government in the United States has not (yet?) regulated fat bodies and the perceived contributors to the so-called obesity epidemic. Regardless, some of its agencies have called attention to the issue, including the Surgeon General, the Department of Health and Human Services, the National Research Council and the National Institutes of Health. Non-for-profit organizations such as the American Heart Association and the American Diabetes Association, have either followed suit or participated in the definition of the problem, basing their support on scientific evidence, while at the global level the World Health Organization (WHO) has been “managing and preventing the obesity epidemic” since 1998 (World Health Organization 2004 [1999]).

My dissertation does not seek to address whether obesity has health consequences, even though the research I review here will speak to this effect. Nor does it try to find a cure for obesity or the obesity epidemic. Rather, this dissertation is descriptive and ironic, in the sense put forward by Ian Hacking in *The Social Construction of What?* An ironic stance in the study of a specific object X (here fat / obesity) is “the
recognition that X is highly contingent, the product of social history and forces, and yet something we cannot, in our present lives, avoid treating as part of the universe in which we interact with other people, the material world, and ourselves” (Hacking, 1999, p. 20).

In short, I believe three things: that fat people really exist; that fat is a biological phenomenon with some physiological consequences; and that most of the concern over body weight is historically contingent and arises from the medicalization of fat. Evaluating the medical and the medicalized aspects of fat is my purpose in this dissertation.

**Conceptual Framework**

My conceptual framework is rooted in the form of institutional theory that DiMaggio and Powell (1991) have labeled “new” institutionalism. Neo institutionalism is concerned with the role that cultural scripts play in defining actors’ behavior and action, through the definition of cognitive patterns. Science, and medicine, are seen as institutions with legitimating power: the ability to produce and claim knowledge as valid, and to determine the course of action of societies and actors within them.

In the introduction to their edited volume, Drori, Meyer, Ramirez and Schofer (2003) discuss the world polity perspective on the role of science, a perspective that stresses the authoritative nature of science, and evaluates the impact of science on collective assumptions, state formation and law, among other things. A few assumptions about the role of science and science education undergird my dissertation.

First, science has cultural authority. Because the scientific method is supposed to transform a previously chaotic world into an orderly one, and is clamored as the privileged way to get to unbiased truth, when scientists speak, all listen. A clear
manifestation of this cultural authority is seen in the almost undisputed view that science and scientists are legitimate informants for individual, organizational and policy decisions.

Second, science is a set of ideas and assumptions about the rationality of the natural and social world, and, as such, is a cognitive cultural model – a world view. Most scientific endeavors assume that there is a world out there (be it natural or social) that behaves according to rational laws, and can be fruitfully studied for insights into how things “really” are. Today, most individuals see and understand the world the way scientists do: the world makes rational sense and can be studied as such.

Third, science is seen as a legitimating force behind choices and actions. Rational individuals, organizations or societies turn to science to inform decisions and use science to validate decisions, performance, outcomes; irrational ones either reject science altogether, or do not “get” science (Ramirez, 2006). This conceptualization of rationality motivates many social and behavioral sciences, most of whom work within an atomistic, rationalistic framework where will is mostly unconstrained, and decision-making processes are assumed to be decipherable based on rationalistic grounds.

Fourth, science education is seen as the cornerstone in the education of rational, empowered and agentic citizens. The growth of science education curricula and science education organizations over time has been documented widely (Benavot et al. 1991) It is a fundamental cultural belief that once youth is educated in the sciences, they will as adults act more rationally and better understand their own, and their organizations’ and societies’ needs and interests. In most societies, this socialization into a rational way of thinking is assumed to be mostly the province of the sciences and of mathematics, as opposed to, say religion or humanities (Benavot et al. 1991).
And fifth, science has become an authority across domains. From atoms structure to sexual health to the functioning of markets and to the evaluation of competence, we turn to science to find answers. If pre-modern cultures turned to God or gods and goddesses, modern societies address every issue through science. In most instances, scientific arguments trump other types of arguments, regardless of the field.

Science, so understood, is clearly a potential (and legitimate!) instrument of power and of social control. My dissertation attempts to be descriptive of the science on fat / obesity, describing the rising tide of medical research on obesity over time, its nature and its direction, withholding my ethical judgment in favor of a historical, sociological explanation of the factors which made this tide happen.

**Methods**

Because this dissertation is concerned with the evolution and contents of the discourse on obesity in medical research over time, its empirical focus is on academic publications, particularly on research articles. Academic publications are conceptualized as proxies for the state of knowledge on a certain topic at a certain point in time. Thus, the longitudinal analysis of publications at the aggregate and individual levels is seen to yield insights into the evolution of ideas over time. The location of publications in the intellectual field is seen to depend on the research tradition (medical science, sociology, history, feminist studies, etc.), and to depend on the social context at a specific point in time.

In conducting document analyses, I relied most often on two important databases: PubMed and ISI Web of Knowledge. The MEDLINE / PubMed database is the most comprehensive online database for access to medical contents worldwide, covering 4,600 biomedical journals published in the United States and 70 other countries,
totaling 19 million citations. Coverage is worldwide, but most records are from English-language sources or have English abstracts. In my queries, I used key words as found in “titles and abstracts,” but also used them as “MeSH terms.” According to the National Center for Biotechnology Information (NCBI) website (NCBI, Medical Subject Headings, 2010), “MeSH is the U.S. National Library of Medicine's controlled vocabulary used for indexing articles for MEDLINE / PubMed. MeSH terminology provides a consistent way to retrieve information that may use different terminology for the same concepts.”

I queried PubMed and recorded the number of publications in five-year increments from 1950 to 1980, and annually from 1980 to 2009. To control for the overall expansion of medical research over time, I normalized my data by dividing the number of query-specific publications by the total number of publications recorded in the PubMed database for every time period (5-year or yearly increments, depending).

While I collected data and ran analyses starting in 1950, the discourse on obesity in the middle of the 20th century was of such a different magnitude that I often focus my discussion on the 1980-2009 or 1990-2009 time period in order to be able to detect the most meaningful variations. For example, while the total number of publications on obesity over the 1950-4 time period was 565 according to the MeSH term counts, it was almost 37,000 in 2005-9. Finally, 2009 data must be taken cautiously because of the recording system of PubMed, and a decline in frequencies of publications at that time point may be artifactual.

I also often queried the ISI Web of Knowledge database. ISI Web of Knowledge is an extensive database that includes the PubMed database, but also documents the number of citations received, and thus allows to sort articles on this criterion, a feature
that is not available in PubMed. Information about the number of citations received by individual articles is often used as an indication of whether or not the scientific community has acknowledged the value of a contribution. Citation information is an imperfect measure of scientific centrality, and of course, as knowledge production moves forward in time, certain sources fall out of favor or are proven wrong by new investigations. Nevertheless, it is often the best measure we have of the centrality of research when evaluating a body of literature that includes millions of publications.

Depending on the aspect of obesity I am investigating, I use either PubMed, ISI Web of Knowledge, or both. Each chapter answers a different research question, serves a different purpose, and thus uses a distinct set of methods. Greater detail is provided at the beginning of each chapter, for clarification.

**Dissertation Overview**

*Part I: To Be, or Not to Be, Fat? The Complex Nature of Obesity*, has four chapters. Chapter 1 discusses the medical evidence that shows how obesity is associated with a score of negative health outcomes, using the most-cited medical articles and textbooks on the topic. Chapter 2 reviews the data documenting the expanding waistlines of Americans over the 1960-2010 time period using National Health And Nutrition Examination Survey (NHANES) data collected by the Center for Disease Control and Prevention’s National Center for Health Statistics (CDC/NCHS), and puts these data in comparative perspective. Chapter 3 voices and evaluates recent and vocal critiques of the current medical paradigm that pathologizes fat, including the idea of the toxic environment and the Health at Every Size (HAES) movement. Chapter 4 briefly reviews the literature on the causes of weight gain and provides a model for its understanding as both a medical and a social problem. It also makes predictions as to where policy and educational
strategies should take us in the near future, if we are to better improve the health of populations.

Part II: Changing Meanings of Fat, has two chapters. Chapter 5 traces a broad portrait of the historical development of our culture’s interest in body fat, and its changing meanings from 1950 to today. In Chapter 6, using queries of the PubMed database from 1950 to 2009, I look at the expansion of the medical discourse on obesity in general and in comparison with other conditions.

The single chapter of Part III: America’s Children, Chapter 7, engages in greater depth what I see as the most recent discursive shift in the medical discourse on obesity: obesity in general, and childhood obesity in particular, as a social crisis in need of educational solutions. It delves deeper into the educational trends in health education, and discusses the current school reforms in historical and comparative perspective.
Part I: To Be, or Not to Be, Fat? The Complex Nature of Obesity
Chapter 1: To Be, Fat. The Health Effects of Obesity.

In this Chapter, I provide a review of the medical literature that connects obesity to mortality and six associated comorbidities: arthritis, cancer, cardiovascular disease (CVD), diabetes, gestational diabetes and fetal health, and mental health. I also briefly review the literature on the economic or financial costs of obesity.

For the purpose of this chapter, I use Body Mass Index (BMI), or weight in kilograms divided by squared height in meters, to define obesity. Underweight is defined as a BMI below 18.5 kg/m$^2$; healthy weight from $18.5 \leq \text{BMI} < 25$; overweight from $25 \leq \text{BMI} < 30$; obese from $30 \leq \text{BMI} < 40$; and morbidly obese BMI $\leq 40$ (WHO 1995 Technical Report Series 854, p. 312). In 1998, the WHO, in its report on obesity, urged that obesity be defined in terms of BMI rather than “simply as a condition of abnormal or excessive fat accumulation in adipose tissue, to the extent that health may be impaired” (WHO 1998 Report, p. 6), which was perceived to be too subjective. Authors of the WHO Report argue that BMI “provides the most useful, albeit crude, population-level measure of obesity” (ibid., p. 7), despite the fact that (1) it does not take into account fat distribution on the body, and (2) ideal BMIs may be lower for Asians (Rigby 2006) and higher for Blacks (Allison et al. 1999). A reproduction of a common BMI Table can be found in Appendix I.

The use of weight categories that control for height allows to compare weight status across individuals, of course, but also within and across populations. Fixed criteria help to identify groups that are at increased risk of disease within populations, and to

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2 Another, less common method is the use of waist circumference thresholds that denote increased and substantially increased risk of metabolic complications for men ($\geq 94$ cm, and $\geq 102$ cm, respectively) and women ($\geq 88$ cm). See Kopelman, P. G. (2000). Obesity as a medical problem. *Nature*, 404, 635-43.
provide a “firm basis” to evaluate (medical and public health) interventions (WHO 1998 Technical Report Series 894, p. 7).

Among youth, obesity is defined as a BMI above the 95\textsuperscript{th} percentile and overweight above the 85\textsuperscript{th} of the appropriate age- and sex-specific growth chart of the CDC. Because reference growth charts were built out of 2000 population data, the percentage of the youth population above a certain threshold will vary over time. The 95\textsuperscript{th} percentile in the 2000 data is exactly 5%, but as we will see in Chapter 2, there has been a growing percentage of youth above this 95\textsuperscript{th} percentile since 2000, and percentages were lower than 5% before 2000.

Historian Hillel Schwartz discusses, in his history of the Western obsession with weight, how the earliest studies linking weight to negative health outcomes were based on doctors’ personal observations and, later, insurance companies’ data. As he notes, the early analyses were fraught with many flaws at all of the data collection, design and interpretation stages of research (Schwartz 1986). More recent critics, including the WHO 1998 Report on the global epidemic of obesity and the NIH /NHLBI’s 1998 Clinical Guidelines on obesity and its treatment, have also pointed to flaws in the research on obesity, including the omission of factors, such as smoking and exercise, which dramatically and systematically influence disease incidence and mortality (Campos et al. 2006a).

Today, however, in the medical community, obesity is seen to be associated with a wide range of negative health outcomes (Rigby 2006), even though there remains a significant level of controversy, especially when it comes to the mitigating effect of cardiorespiratory fitness (Blair and LaMonte 2006). Medical science and public health research have come a long way since the late 19\textsuperscript{th} century, with increasing standards for
the scientific study of weight, better analytic techniques, and more refined data collection methods. Yet they are not immune from criticism, as we shall see in later chapters.

Methods

Because I am not trained in medicine or public health, I based this review on the ISI Web of Knowledge database, which I queried with a combination of “obesity,” mortality and the six health conditions cited above. I reviewed the 10 most cited articles that included keyword combinations in either their titles or as their topic. When needed, I followed the leads found in the identified top 20 articles for each factor to paint the most accurate portrait of its connection to obesity. I also made use of handbooks and textbooks on obesity, which provided useful literature reviews. More than 140 articles were reviewed, and only the pertinent ones cited here.

This approach allows me to describe the current research paradigm on obesity by focusing on what is taken as of proven value (and thus widely cited) in this field. I am presenting these data as though they were facts, since their high number of citations place them amongst obesity “facts,” or widely accepted truths (Latour & Woolgar, 1986 [1979]).

I report here on the medical consequences of obesity, rather than overweight, for two reasons: first, rates of obesity have been growing since the 1960s, while the rates of overweight haven’t been (see Chapter 2); second, the consequences associated with obesity are much clearer and better documented than the consequences of overweight, with some studies finding a protective effect of overweight.

In the medical literature, the impact of a risk factor – here obesity – on a specific outcome is evaluated using odds-ratios or relative risks. Odds ratios and relative risks greater than one (>1) indicate an increased likelihood of an event happening in the first
group; odds of one (1) indicate equal likelihood; and below one (<1), a decreased likelihood. Odds ratios are often reported with a confidence interval that determines statistical significance. The confidence interval (CI) is typically defined as 95% of the total probability distribution, and estimates that do not include 1 are said to be statistically significant at the 0.05 level.

In population studies, odds-ratios estimates are complicated by uncertainty of measurement, and by the fact that we rarely know the full causal chain that connects a factor to an end outcome. Odds ratios, while harder to interpret than relative risks, are often used in medical research because they are insensitive to sampling methods, and can be used with convenience samples, while risk ratios should be used with representative samples only.

Odds-ratios provide an estimate of the strength of the association between a binary variable and the probability of an event. In the case of obesity research, odds ratios are typically calculated across BMI categories, controlling with logistic regression variations within groups on confounding variables such as age, race, gender, smoking, etc. They are defined by Equation 1.1, below.

**Equation 1.1. Odds Ratio (OR)**

\[
\text{OddsRatio} = \frac{\frac{p_2}{1 - p_2}}{\frac{p_1}{1 - p_1}}
\]

Here, \(p_1\) is the probability of an event happening in group 1; \(p_2\) is the probability of the same event happening in group 2. \((1 - p_1)\) is the probability of the event not happening in group 1; \((1 - p_2)\) the probability of it not happening in group 2. By dividing the ratio of \(p_1\) to \((1 - p_1)\) by the ratio of \(p_1\) to \((1 - p_2)\), we find a measure of the impact group membership has on the probability of a certain outcome, what is called effect size.
One may, for example, measure the effect size of hand washing on the likelihood of catching the flu in a certain year. Group 1 here is people who wash their hands; group 2 people who don’t. If the probability of catching the flu for someone who washes their hands regularly is 1/1000, and the probability of someone who doesn’t is 1/200 (fictitious numbers), then the odds ratio associated with hands washing is:

Equation 1.2. Odds ratio, hand washing behavior on likelihood of catching a cold

\[
OR = \frac{\frac{0.001}{1-0.001}}{\frac{0.005}{1-0.005}}
\]

\[
= 0.199
\]

The odds of catching a cold are much lower when one washes their hands than when one doesn’t, and we can cautiously interpret that there is a strong association between hands washing and catching colds. If one had a confidence interval, one could evaluate whether this association is significant or not.

Relative risks are computed and interpreted differently. They are calculated as the ratio of the probability of an event happening in group 1 over the probability in group 2.

Equation 1.3. Relative Risk (RR)

\[
RelativeRisk = \frac{p_1}{p_2}
\]

Using our flu example, the relative risk would be:

Equation 1.4. Relative risk, hand washing behavior on likelihood of catching a cold

\[
RR = \frac{0.001}{0.005}
\]

\[
= 0.2
\]
Here, people who wash their hands are 20% less likely to catch a cold than those who don’t wash their hands.

While in this case the relative risk is very close to the odds ratio, it is not always the case. Higher probabilities or probabilities of different magnitudes can significantly change the results. For example, the discrepancy is high for probabilities \( p_1 = 0.1 \) and \( p_2 = 0.5 \).

*Equation 1.5. Comparing OR and RR, higher probabilities*

\[
OR = \frac{\frac{p_1}{1-p_1}}{\frac{p_2}{1-p_2}} = \frac{0.1}{0.5} = 0.111
\]

\[
RR = \frac{p_1}{p_2} = \frac{0.1}{0.5} = 0.2
\]

With this introduction to statistical thinking in the research I am reviewing here, I turn to the evidence that connects obesity with morbidity and the six factors identified above, as seen in the most-cited articles on the topic. But first, I look at some of the mechanisms whereby overnutrition, and its associated weight gain, are potentially unhealthy.

1.1 Overnutrition and Its Physiological Impacts

1.1.1 Body Fat as Active Tissue

One of the fundamental elements in the medical study of overweight and obesity is the conceptualization of adipose tissue as “an active endocrine and metabolic organ that can have far-reaching effects on the physiology of other tissues” (Calle and Kaaks
2004, p. 584), rather than as an amorphous extra tissue. Indeed, fat has endocrine, immune, and metabolic functions (Power and Schulkin 2009, p. 245). According to Ahima and Flier (2000), the scientific community has turned its gaze to the role played by fat in the synthesis of proteins after the discovery in the mid 1990s of leptin – a hormone that is critical in the regulation of energy intake and expenditure. Lau et al. (2005) note that:

Adipose tissue is no longer viewed as a passive repository for triacylglycerol storage and a source of free fatty acids (FFAs). As developing preadipocytes differentiate to become mature adipocytes, they acquire the ability to synthesize hundreds of proteins, many of which are released as enzymes, cytokines, growth factors, and hormones involved in overall energy homeostasis (Lau et al. 2005, p. H2032).

Adipokines, proteins secreted by adipose tissue, include leptin, resistin, tumor-necrosis factor α (TNF-α), and insulin-like growth factor 1, hormones that are critical in either or both energy metabolism and insulin resistance, as well as cellular multiplication and cancerous tumor growth (Calle and Kaaks 2004; Lau et al. 2005). Adipose tissue also secretes C-reactive protein, which may lead to arthritis, and sex hormones (estrogens, androgens, and progesterone), which are connected to certain cancers. Clearly, extra adipose tissue is not merely a repository of fat: it impacts the body’s internal equilibrium with its ability to secrete a range of molecules that in turn trigger further physiological reactions.

As we are reminded by Power and Schukkin (2009), not all fat is bad. The brain, for example, is a high-fat organ. Too much fat, however, transform the body’s internal homeostasis – its state of balance.
When someone eats, blood sugar (glycemia) rises as a consequence. To handle increased blood sugar, beta cells in the pancreas produce insulin, a function of which is to transport glucose to peripheral cells, fueling them with energy and enabling glucose to cross the cellular membrane. Once peripheral cells have had their energy needs met, leftover glucose is transformed into fatty acids through another function of insulin, and then stored as fat. Since the body has a limited need for energy, excess caloric intake by necessity leads to extra fat storage. Further, too much fat in the diet prevents its full transformation into energy, which leads to the accumulation of health-damaging lipid droplets in the body (Power and Schulkin 2009, p. 246).

Chronically increased blood sugar levels (hyperglycemia) can lead to beta cells being over-stimulated and damaged or destroyed, which can lead to glucose intolerance or impaired glucose tolerance (IGT) and Type 2 diabetes mellitus (non-insulin-dependent diabetes mellitus, NIDDM) (Ferrannini and Camastra 1998). This is especially so among people with genetic predispositions for its development (DeFronzo and Ferrannini 1991). Because extra caloric intake increases blood sugar levels, which lead in turn to hyperinsulinemia (as beta cells produce more insulin to mitigate the increase in blood sugar), insulin resistance – the shutting down of cells from insulin-stimulated glucose uptake – can occur. Insulin resistance, indeed, has indeed been seen to be inversely related to insulin production (Ferrannini and Camastra 1998). The more insulin one produces, the more likely are cells to become insulin resistant.

As noted by Reaven (1988), genetic variation in insulin production makes for a large variation in the consequences of hyperglycemia. Individuals who can produce high levels of insulin will have better glucose uptake, and glucose intolerance will be less
pronounced. As shown by Ferrannini (2002), insulin release increases with BMI for every level of insulin sensitivity, making obese people more hyperinsulinemic than normal-weight individuals with similar insulin sensitivity. Hyperinsulinemia has documented adverse health consequences, and co-occurs with a host of factors that partake in disease genesis, including insulin resistance (Lau et al. 2005), and Reaven’s Syndrome X.

Still: not all obese people are or will become diabetic. They are distinct phenomena.

1.1.3 Syndrome X: The Metabolic Syndrome of Insulin Resistance

Metabolic syndrome of insulin resistance, the new name of Syndrome X, was first discussed by Reaven in his 1988 Banting Lecture. It refers to a cluster of conditions that co-occurs with insulin resistance and is associated with increased risks of cardiovascular disease. The conditions identified by Reaven (1988) as the main indicators of metabolic syndrome are: (1) resistance to insulin-stimulated glucose uptake; (2) glucose intolerance; (3) hyperinsulinemia; (4) increased circulating blood levels of very-low-density lipoprotein triglyceride (bad cholesterol); (5) decreased circulating blood levels of high-density lipoprotein cholesterol (good cholesterol); (6) hypertension (Table 1, Reaven 1988, p. 1605).

Because adipose tissue, as we have seen above, secretes adipokines that increase the likelihood of insulin resistance, it is an important part in the etiology of metabolic syndrome. Insulin resistance is a precursor, but not the only cause, of NIDDM (Ferrannini 2002). Meanwhile, obesity is both a complicating factor of insulin resistance and strongly associated with the likelihood of developing NIDDM (Ferrannini and Camastra 1998), particularly through the release by adipose tissue of free fatty acids,
which are in healthy individual metabolized through the use of insulin. Insulin resistant individuals thus further their glucose intolerance.

In sum: increased caloric intake leads to increased blood sugar level and increased insulin production in response. With progressively impaired glucose tolerance, hyperglycemia and hyperinsulinemia can lead to insulin resistance. Extra adipose tissue, with its secretory potential, further changes bodily homeostasis when it comes to glucose metabolism, resulting in a whole host of potential adverse health consequences complicated by insulin resistance.

1.2 Mortality

The effects of obesity on mortality have been investigated using three main methods: estimates of relative risks or odds-ratios associated with different BMIs, estimates of extra annual deaths at the population level, and in terms of years of life lost at the individual level.

1.2.1 Comparing Mortality Risks

Must et al.’s (1992) investigated the long-term costs of adolescent overweight and obesity among participants in the Harvard Growth Study. They found that excess weight was associated with an increased risk of mortality from all causes and disease-specific mortality among men, but not among women. The relative risks among men were 1.8 (95% CI, 1.2 to 2.7; P = 0.004) for mortality from all causes and 2.3 (95% CI, 1.4 to 4.1; P = 0.002) for mortality from coronary heart disease.

Manson et al. (1995), looking at the Nurses’ Health Study data on 115,195 women in the US, found that a BMI greater than 32 was associated with a death odds ratio of 1.5 (95% CI, 1.3-1.7), compared to women with a BMI lower than 19. Both obese women
who smoked, and obese women who never smoked and had maintained a stable weight, had even greater odds ratios of death compared to similar women with lower weights.

An early literature review by Solomon and Manson (1997a) found that extra weight was associated with greater mortality throughout the life course. A recent article by Adams et al. (2006) investigates more closely the impact of overweight on mortality, and finds that an “excess body weight during midlife … is associated with an increased risk of death” (p. 763). The trend was much stronger among men who had never smoked. A very recent study by Cai et al. (2010), however, cautioned that while the obese had significantly shorter life spans than normal-weight individuals, obese people who had lost weight in middle age had the lowest chance of surviving to age 65, and the shortest life expectancies.

1.2.2 Annual Death Toll

Allison et al. (1999) estimated the annual death toll for overweight and obesity in the U.S. at 280,000 extra deaths, and Mokdad et al. (2004) estimated it at 414,000 deaths. Flegal et al. (2005) criticized these estimates on methodological grounds and provided new estimates. Compared with normal-weight, obesity was associated with 111,909 excess deaths (95% CI, 53,754-170,064), and underweight was associated with 33,746 excess deaths (95% CI, 15,726-51,766). Their estimates were in turn criticized harshly by Willet et al. (2005): the inclusion of the chronically ill by Flegal et al. in their analyses led them to write that “the relative and attributable risks reported by Flegal et al are indeed affected by artifact” (Willet et al. 2005, 51), particularly for the overweight category, which had lower incidences of death in Flegal et al.’s analyses than the normal-weight category. They then point to “more valid” analyses that have found higher mortality among the
overweight: Manson et al. (1995), Willet, Dietz and Colditz (1999a), and Calle et al. (1999). I review these articles here.

Willet, Dietz and Colditz (1999a) argue strongly for the correlation of BMI with mortality and morbidity. Their figures show the differential effects of BMI on risk of death among women and men, across age groups. Among women, increasing BMI was found to be associated with greater mortality risks until age 75, where BMIs in the 22-32 range are associated with lower mortality rates than BMIs below 22. Among men, increasing BMI is also associated with greater mortality, but the relationship isn’t linear for men above 75. Their figures also traces the increasing risks of different afflictions with BMI.

Calle et al. (1999) note that among “healthy people who had never smoked, the nadir of the curve for body-mass index and mortality was found at a body-mass index of 23.5 to 24.9 in men and 22.0 to 23.4 in women” (p. 1097; see also Table 3, p. 1102). “Ideal” weights, then, are very close to the upper limit of the normal weight range for men, and just below that for women, in disagreement with Flegal et al. (2005) but in line with Willet et al.’s (2005), Willet, Dietz, and Colditz’s (1999a) and Manson et al.’s (1995) article.

1.2.3 Years of Life Lost

Using years of life lost estimates by 1-unit increments in BMI, Fontaine et al. (2003) found that “obesity has a profound effect on life span” (p. 189). They also found that the optimal BMI ranges are approximately from 23 to 25 for whites, and from 23 to 30 for blacks. Starting at the upper range of these intervals, the curve of years of life lost is concave, and increasingly steep.
1.3 Arthritis

Mokdad et al. (2003) write that “overweight and obesity were significantly associated with diabetes, high blood pressure, high cholesterol, asthma, arthritis, and poor health status” (p. 76). An odds ratio of 4.41 (95% CI, 3.91-4.97) predicted the appearance of arthritis among morbidly obese adults, and 2.03 (95% CI, 1.92-2.14) among obese adults. Similarly, Symmons et al.’s (1997) matched pair design to investigate the risk factors associated with rheumatoid arthritis found an adjusted odds ratio of 2.84 (95% CI, 1.20-6.76) among subjects with a BMI over 30, compared to lighter subjects. The ratio was greater for women than men.

Felson et al. (1988), in their study of Framingham data, found an odds ratio for arthritis of 1.51 (95% CI, 1.14-1.98) for the highest weight quintile among men, compared to the lightest three quintiles. Among women, the fourth weight quintile had an odds ratio of 1.44 (95% CI, 1.11-1.86), and the highest quartiles one of 2.07 (95% CI, 1.67-2.55), compared to the lowest three quintiles. Obese women were twice more likely than normal weight women to develop arthritis. An earlier review by Felson (1996) of studies on NHANES data showed a 4 times greater likelihood of getting knee osteoarthritis for obese women, and 4.8 times for obese men, compared to similar women and men with BMIs under 25.

1.4 Cancer

In their article for *Nature Reviews, Cancer*, Calle and Kaaks (2004) summarize epidemiological evidence connecting obesity to cancer. They note that extra weight increases the probability of many cancers: for men and women with BMIs over 40, respectively, they found odds-ratios of 1.52 (95% CI, 1.13-2.05) and 1.62 (95% CI, 1.13
to 2.05) overall. They also suggest that as many as 14% of all deaths from cancer in men and 20% of those in women could be attributed to obesity.

Must et al. (1992) found that men who were overweight as adolescents had a relative risk of 9.1 (95% CI, 1.1-77.5) to die from colorectal cancer compared to men who weren’t, while Manson et al. (1995), looking at Nurses’ Health Study data on 115,195 women in the US, found a doubling of the risk of death from cancer among obese women.

A study of Danish data by Moller et al. (1994) found similar increases in cancer incidence among obese people. Compared to non-obese subjects, risk of cancer for men was 1.16 (95% CI,1.07-1.25), 1.17 for women (1.0-1.23), with overall odds ratio of 1.16 (95% CI,1.11-1.21). The greatest odd ratios were, for men, for cancers of the oesophagus, liver and pancreas, all at 1.9. For women, the cancers most associated with obesity were oesophagus, liver, uterus, and kidney cancers, with odds ratios of 1.9 or 2.0.

Wolk et al. (2001) found significantly increased risks for all cancers with high BMIs, with odds ratios of 1.25 (95% CI 1.14-1.36) among obese men and 1.37 (95% CI, 1.30-1.44) among obese women. Obese men were significantly more likely to develop small intestine, pancreas, renal parenchyma, and Hodgkin’s disease cancers. Obese women were more likely to develop colon, gallbladder, cervix, endometrium, ovary, renal parenchyma, brain, and connective tissue cancers.

1.5 Cardiovascular Disease (CVD)

Obesity is a major risk factor for and comorbidity of both coronary heart disease and atherosclerotic cardiovascular disease. Kim and Popkin (2006) summarize that obesity increases the risk of coronary heart disease and stroke by augmenting the likelihood of hypertension, dyslipidaemia, and diabetes, three important risk factors in the
etiology of these diseases. Atherosclerosis is exacerbated among people with diabetes, but also more likely in obese people, because they tend to have higher circulating blood levels of low-density fatty acids (including bad cholesterol).

Hubert et al. (1983) defined obesity as an independent risk factor for cardiovascular disease using Framingham data. They found that both men and women faced higher risks of angina and other coronary heart disease. Ten years later, Must et al.’s (1992) study of data from the Harvard Growth Study found relative risks for men who had been overweight as adolescents, compared to lean men, of 13.2 (95% CI, 1.06-18.0) for death from atherosclerotic cerebrovascular disease, and of 2.3 (95% CI, 1.4-4.1) for death from coronary heart disease. As stated earlier, relative risk among overweight adolescent women was not significant. A few years later, however, Manson et al.’s (1995) Nurses’ Health Study, cited by the WHO 1998 Report, found a 5.8 odds ratio for death from coronary heart disease among obese women, compared to women with BMIs below 22, and a trend p-value < 0.001. Willet et al. (1999b) similarly found relative risk ratios of 3.56 (95% CI, 2.96-4.29) for coronary heart disease among women with BMIs above 29, compared to women with a BMI of 21.

McGill et al. (2002), in their post-mortem study of young men who died of non-heart related causes, found that BMI was strongly and significantly associated with coronary atherosclerosis, in terms of lesions and fatty streaks.

1.6 Diabetes and Metabolic Syndrome

Historian Hillel Schwartz’ research shows the long history of diabetes’ association with obesity. In 1921, he writes,

Dr. Elliott P. Joslin of Boston, America’s authority on diabetes epidemiology and himself a man of 300 lbs, reported on his study of more than one thousand diabetics. Forty percent had been obese before the
onset of diabetes in middle age. … So closely tied were obesity and diabetes by 1921 that when Banting and Best isolated insulin, they prescribed it with a low-calorie, low-carbohydrate, low-fat diet that required the weighing of foods (Schwartz 1986, 173).

The first scientific publication on obesity’s connection to diabetes was made by H.J. John in 1929 in *Endocrinology* (Albu and Pi-Sunyer 2004). Since, the causal connection between obesity and diabetes has been accepted as fact. Two intervening conditions have been posited: insulin resistance (Kim and Popkin 2006), and metabolic syndrome (defined above).

Using NHANES III data (1988-1994), Ford, Giles, and Dietz (2002) evaluated that the age-adjusted prevalence of metabolic syndrome in the US adult population was 23.7%, with Mexican Americans having the highest rates at 31.9%. Overall, men and women had similar rates, but both African American and Mexican American women had a higher prevalence of metabolic syndrome than their co-ethnic men.

Among adolescents aged 12-19, Cook et al. (2003) estimated the prevalence of metabolic syndrome at 6.1% of males, and 2.1% of females, with 4.2% of that population overall in the NHANES III data; 28.7% of adolescents above the 95th percentile of BMI had metabolic syndrome, and the BMI trend was highly significant (p<0.001).

Mokdad et al.’s (2003) review of 2001 Behavioral Risk Factor Surveillance System data found a 4.1% incidence of diabetes among normal-weight subjects, and incidences of 7.3%, 14.9%, and 25.6% respectively among overweight, obese and morbidly obese subjects. Fully adjusted odds ratios (controlling for age, education, smoking, sex, and race

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3 They defined metabolic syndrome as having at least three of the five following markers: waist circumference greater than 102 cm in men and 88 cm in women; serum triglycerides level of at least 150 mg/dL; high-density lipoprotein cholesterol level of less than 40 mg/dL in men and 50 mg/dL in women; blood pressure of at least 130/85 mm Hg; or serum glucose level of at least 110 mg/dL (Ford, Giles and Dietz 2002, p. 356).

4 The definition of metabolic syndrome among youth is a modified version of the adult criterion. See Cook et al. 2003, p. 822, for details.
or ethnicity) were, compared to normal-weight subjects, 1.59 (95% CI, 1.46-1.73) for overweight, 3.44 (95% CI, 3.17-3.74) for obese, and 7.37 (95% CI, 6.39-8.50) for morbidly obese subjects, all ratios being statistically significant.

Must et al. (1999) report on the prevalence of Type 2 diabetes by BMI in weighted NHANES III data. They found Type 2 diabetes in 2.03% of normal-weight men, 4.93% of overweight men, 10.10% of obese class 1 men (BMI between 30 and 34.9), 12.30% of obese class 2 men (BMI between 35 and 39.9) and 10.65% of obese class 3 men (BMI above 40). Among women, percentages were of 2.38%, 7.12%, 7.24%, 13.16% and 19.89%.

Solomon et al. (1997a) report that mortality from NIDDM was five times greater among individuals who were 25% overweight, compared to lean individuals.

1.7 Gestational Diabetes Mellitus and Fetal Health

Maternal obesity has been connected to many health problems for both mother and offspring. I first look at the evidence connecting obesity with gestational diabetes mellitus (GDM), before turning to its impacts on the fetus’ future health outcomes.

1.7.1 Maternal Obesity and Gestational Diabetes Mellitus

Obesity is associated with a greater likelihood to develop GDM in future pregnancy and Type 2 diabetes later in life (Holemans et al. 2004). Sebire et al. (2001) reported an odds ratio of 3.6 (99% CI, 3.25-3.98) for gestational diabetes among women with BMIs above 30. Chu et al.’s (2007) meta-analysis of 20 studies on the association between maternal obesity and risk of gestational diabetes. They found unadjusted odds ratios of 2.14 (95% CI, 1.82- 2.53), 3.56 (3.05-4.21), and 8.56 (5.07-16.04) for overweight,
obese, and severely obese pregnant women, compared with normal weight pregnant women.

Solomon et al. (1997b) were among the first investigators of the predictors of GDM. Using Nurses Health Study data, they found that obesity was associated with a multivariate odds ratio of 2.90 (95% CI, 2.15-3.91) for developing GDM, an effect greater than that associated with a pregnancy after age 40, and current smoking.

1.7.2 Impact of GDM on Fetal Health and Future Health Outcomes

Silverman et al. (1995) studied the long-term effects of mothers’ diabetes on glucose tolerance among offspring over time and found that impaired glucose tolerance (IGT) was indeed related to maternal diabetes, and that excessive insulin secretion by the fetus in utero is a strong predictor of IGT later in childhood. Similarly, Plagemann et al. (1997) studied the offspring of mothers with insulin-dependent diabetes and gestational diabetes. They found that these infants were more likely to develop overweight and obesity during childhood, an effect compounded by high neonatal plasma insulin levels and high birth weight. Later research by Gillman et al. (2003), however, with adolescent weights questioned the causal effects of gestational diabetes on adolescent weight.

In a matched-pair study, Boney et al. (2005) found odds ratios of 2.19 (95% CI, 1.25-3.82) for large for gestational age versus appropriate for gestational age children, and of 1.81 (95% CI, 1.03-3.19) for maternal obesity, defined as a BMI above 27.3. They also found a significantly greater frequency of metabolic syndrome among youth that were large for gestational age and whose mothers had gestational diabetes mellitus (p (χ²) = 0.001). Among the offsprings of mothers with gestational diabetes, those who were large at birth were 3.6 times more likely to develop metabolic syndrome by age 11 (p = 0.004).
Anderson et al. (2005) also found increased risks of central nervous system birth defects in offspring of obese women, and in the offspring of mothers with GDM. The joint effects of maternal obesity and gestational diabetes were found to be multiplicative for spina bifida and holoprosencephaly, with adjusted odds ratios of 4.5 (95% CI, 1.5-13.0) and 6.5 (95% CI, 1.3-31.0).

1.8 Mental Health Problems

The effects of obesity on mental health aren’t well documented. While the stigma faced by obese people has been well documented (see Chapter 3), the actual causal mechanisms that connect obesity and mental health problems, or those that connect mental health problems with future obesity, aren’t fully understood yet.

In their literature review of clinical studies, McElroy et al. (2004) found that

(1) children and adolescents with major depressive disorder may be at increased risk for developing overweight; (2) patients with bipolar disorder may have elevated rates of overweight, obesity, and abdominal obesity; and (3) obese persons seeking weight-loss treatment may have elevated rates of depressive and bipolar disorders (p. 634).

They also found that “most overweight and obese persons in the community do not have mood disorders” (p. 634). Mostly, then, it seems that mental health issues may lead to obesity rather than the other way around, and that obese people seeking mental health support from professionals may be generally more distressed than the average obese person, just as a lean person seeking professional mental health support is more likely to be distressed than the average lean person.

McIntyre et al.’s (2006) epidemiological study of obesity among people aged 15 and over found that people with a lifetime history of mood disorders were more likely to be obese than people without them (19% vs. 15%, respectively; p < 0.001), and that this effect was mostly driven by women with a history of mood disorders (1.22, 95% CI, 1.03
to 1.46). Their results align with those of McElroy et al. (2004). Finally, they note how the use of antipsychotic medicine was also associated with obesity, thereby hinting at a potential weight-gain side effect of these medications.

In one of the few longitudinal studies connecting obesity to symptoms of depression, Needham et al. (2010) found that while depressive symptoms at baseline were associated with increased BMI among whites and increased waist circumference over time for men, whites, and people with more than 12 years of education, they did not find that a greater BMI or waist circumference led over time to greater depressive symptoms, confirming the findings of others above.

1.9 The Economic Costs of Obesity

Calculations of the financial or economic cost of obesity are really controversial. The first article I could find which directly makes population-wide estimates is Colditz’ (1992) “Economic Costs of Obesity.” He predicted, based on 1986 data, that combining the costs of NIDDM, cardiovascular disease, gall bladder disease, hypertension and breast and colon cancer, obesity “cost” $39.3 billion dollars, or 5.5% of the cost of illness in 1986. A later estimate by Wolf and Colditz (1998) included the above, but also excess work-lost days, restricted activity, bed days and physician visits attributable to obesity. Both direct costs (such as personal health care, hospital care, and medication), and indirect costs (such as lost output due to reduction or cessation of work productivity due to morbidity or mortality) were included in the $99.2 billion dollars estimate for 1995. Quesenberry, Caan and Jacobson (1998), using data from 17,118 members of an HMO, found that people with BMIs between 30.0 and 34.9 cost 25% more mean annual total costs were 25% more than normal-weight individuals, while people with BMIs above 35 cost 44% more.
Van Baal et al. (2008) simulated the lifetime healthcare costs of obese individuals, smokers and otherwise healthy individuals (defined as non-smoking and non-obese). They found that obese people were the most expensive annually only until age 56, when smokers became more expensive. Over their lifetime, smokers are the least costly, followed by the obese, while healthy-living people were the most costly. Of course, their estimation is limited in that they do not value human lives in themselves: a lost life does not add anything to the cost balance. Ad absurdum, what Van Baal et al. point to is that health cost calculations are a risky matter, and that putting value onto lives and health is a tricky business.

**Conclusion**

We have seen in this chapter the effects of obesity on one’s health, specifically for mortality, arthritis, cancer, cardiovascular disease, diabetes, gestational diabetes and fetal health, and mental health, as they are depicted in the literature. Based on this literature, obesity appears as a “real” health problem in the United States: it is associated with both mortality and morbidity. In the next chapter, I explore weight trends in the United States, from 1960 to 2010.
Chapter 2: To Get, and Be, Fat. Obesity in the U.S.

In this Chapter, I discuss the current weights of Americans and put them in perspective by showing weight gain in the United States from 1960 to 2010. I use the same cutoff points and definitions as in Chapter 1: Among adults, overweight, obesity and morbid obesity are defined respectively as $25 \leq \text{BMI} < 30$, $30 \leq \text{BMI} < 40$, and $\text{BMI} \leq 40$; among children and adolescents, overweight is defined as BMIs above the 85th percentile of age and gender appropriate 2000 growth charts (U.S. Center for Disease Control, 2010), and obesity at the 95th percentile.

The main source of weight data for the United States’ population is the National Health and Nutrition Examination Survey (NHANES), conducted at irregular intervals by the Center for Disease Control’s National Center for Health Statistics (CDC/NCHS) and collected since the 60s through probabilistic sampling and population adjustments. I present NHANES data published in medical journals or online.

2.1 Weight Gain in the United States, 1960-2010

The reality of weight gain in the U.S. has been contested, and dismissed as potentially insignificant. Campos (2004), for example, wrote:

Because the population’s weight follows a standard deviation – that is, a bell curve – placing the definitions for overweight and obesity near the populace’s median weight guarantees that an average weight increase of just a few pounds will suddenly throw millions of Americans into the overweight and obese categories. This is precisely what happened during the 1990s, when an average weight gain of 8 pounds among American adults produced a 61% increase in the obesity rate (Campos 2004, 122).

What is the scale of weight gain in the United States’ population over time?
2.1.1 Adults

NHANES data starts only in the 1960s, but the growth of obesity over the past 45 years is undeniable, as can be seen on Figure 2.1 below, borrowed from the Health USA Report of 2009 (National Center for Health Statistics 2010). Over time, the percentage of the population that is overweight hasn’t changed much, oscillating within a 2.1% interval. The growth of obesity (BMI of at least 30 kg/m²), however, is notable.

Table 2.1 below displays percentages of obesity among the U.S. adult population, based on NHANES data. In 1960-2, 13.3% of the U.S. population was obese, 15.1% in 1976-80, 23.3% in 1988-94, and tipped over the 30% mark by 1999, to reach a high of 34.1% in 2003-6 (p. 116). By 2007-8, 33.8% of the adult population was obese. The number of obese people grew by 20.5% over time, and this growth represents a 154.1% increase from 1960-2. In more visual terms, weight gain amounted to more than one fifth of adults aged 20 to 74 becoming obese over a 45 year time period, or for two obese adults in 1960-2, there are now 5.

2.1.2 Children

That weights among children aged 6 to 11 and 12-19 have also been on the rise can be seen on Figure 2.1 and Table 2.1. The earliest data was collected in 1963-5, at which point 4.2% of youth aged 6-11 were above the 85th percentile of BMI based on the CDC’s 2000 growth charts. Ten years later, percentages were similar, and the first data on youth aged 12-19 showed that 6.1 percent of them were above the 85th percentile. By 1988-1994, 11.3% of the younger youth were overweight, compared to 10.5% of the older youth. At the turn of the millennium, it reached 15.1% and 16%; by 2007-8, 19.6% of 6-11 year olds and 15.6% of 12-19 year olds were overweight.
Fig. 2.1: Overweight and Obesity Rates, NHANES Data, 1960-2006

NOTES: Estimates for adults are age-adjusted. For adults: overweight including obese is defined as a body mass index (BMI) greater than or equal to 25, overweight but not obese as a BMI greater than or equal to 25 but less than 30, and obese as a BMI greater than or equal to 30. For children: overweight is defined as a BMI at or above the sex- and age-specific 95th percentile BMI cut points from the 2000 CDC Growth Charts. United States. Obese is not defined for children. See data table for Figure 7.

SOURCES: CDC/NCHS, National Health Examination Survey and National Health and Nutrition Examination Survey.
Fig. 2.2: Diabetes and Obesity in the U.S.  
CDC / NHANES 2007

Diabetes

Obesity
Table 2.1: NHANES Data on Percentage of Obese Adults and Youth among US Population, and Growth Rates over Time

<table>
<thead>
<tr>
<th></th>
<th>Adults</th>
<th>%</th>
<th>Youth</th>
<th>%</th>
<th>6-11 yo (%)</th>
<th>%</th>
<th>12-19 yo (%)</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>NHANES Years</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1960-2</td>
<td></td>
<td>13.3</td>
<td>(1)</td>
<td>1963-65</td>
<td>4.2</td>
<td>(1)</td>
<td>n/a</td>
<td></td>
</tr>
<tr>
<td>1971-4</td>
<td></td>
<td>14.6</td>
<td>(1)</td>
<td>1971-4</td>
<td>4.0</td>
<td>(1)</td>
<td>6.1</td>
<td>(1)</td>
</tr>
<tr>
<td>1976-80</td>
<td></td>
<td>15.1</td>
<td>(1)</td>
<td>1976-80</td>
<td>6.5</td>
<td>(1)</td>
<td>5.0</td>
<td>(1)</td>
</tr>
<tr>
<td>1988-1994</td>
<td></td>
<td>23.3</td>
<td>(1)</td>
<td>1988-1994</td>
<td>11.3</td>
<td>(1)</td>
<td>10.5</td>
<td>(1)</td>
</tr>
<tr>
<td>2003-6</td>
<td></td>
<td>34.1</td>
<td>(1)</td>
<td>2003-4</td>
<td>18.8</td>
<td>(1)</td>
<td>17.4</td>
<td>(1)</td>
</tr>
<tr>
<td>2007-8</td>
<td></td>
<td>33.8</td>
<td>(3)</td>
<td>2007-8</td>
<td>19.6</td>
<td>(2)</td>
<td>15.6</td>
<td>(2)</td>
</tr>
<tr>
<td><strong>Growth Rates</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1976-1988 growth</td>
<td></td>
<td>54.3</td>
<td></td>
<td>1976-1988 growth</td>
<td>73.8</td>
<td></td>
<td>110.0</td>
<td></td>
</tr>
<tr>
<td>1988-1999 growth</td>
<td></td>
<td>33.5</td>
<td></td>
<td>1988-1999 growth</td>
<td>33.6</td>
<td></td>
<td>52.4</td>
<td></td>
</tr>
<tr>
<td>1960-2007 growth</td>
<td></td>
<td>154.1</td>
<td></td>
<td>1963-2007 growth</td>
<td>366.7</td>
<td>n/a</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Annual growth, 1960-2007</td>
<td>3.3</td>
<td></td>
<td>Annual growth, 1963-2007</td>
<td>8.3</td>
<td>n/a</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Sources:**
(1) Health United States 2009 Data. Table for Figure 7, p. 116.
(2) Ogden et al. (2010), Table 2, p. 244.
(3) Flegal et al. (2010), Table 2, p. 236.

**Note:** Obesity in adults defined as BMI at or above 30. Among youth, it is defined at or above 85th percentile of CDC Growth Charts.
The growth rates of Table 2.1 show a distressing portrait of the rise of overweight and obesity among youth: among 6-11 year olds, annual growth has been of 8.3% over 44 years, totaling 366.7%; among 12-19 year olds, growth has been of 4.3% annually over the past 36 years, totaling 155.7%. Visually, this means that for approximately every obese youth aged 6-11 in 1963, there are now a little over 5; among 12-19 year olds, the ratio is of eight obese adolescents for every three obese adolescents in 1971. Minority youth were found to be more likely to gain weight between 1986 and 1998 (Strauss and Pollack 2001), and minorities and low income youth were also found more likely to gain weight between 1990 and 2006 (Wang and Beydoun 2007).

Statistical analyses on the NHANES data have shown the stabilization of weight among adults over the last decade (Flegal et al. 2010), with women’s weight stabilizing before men’s. They have also shown children’s weights stabilizing recently, except among the very heaviest boys (Ogden et al. 2010).

2.2 Current United States’ Population Weights

2.2.1 Adults

According to the CDC/NCHS’s most recent report, dated June 2010 and based on data from 2007-2008, “an estimated 34.2% of U.S. adults aged 20 years and over are overweight, 33.8% are obese, and 5.7% are extremely obese” (Ogden and Carroll 2010). Flegal et al. (2010) report on the median BMI of men and women for 1999-2000 and 2007-8, and on the percentages of the population in different BMI groups. I present a summary of the evidence they present in Table 2.2 below. This table makes it evident that median weights in the U.S. population are above the upper boundary of the healthy
weight category range, or in other words that a very large part of the population is overweight.

The summary table also shows gender differences. While there are many more men than women in the overweight category (72.3 vs. 64.1), there are many more women in the obese categories (10.7 vs. 17.8 at BMIs above 30). Among men, 4.2% of the population is morbidly obese; among women, it is 7.2%, a 1:1.7 ratio.

Flegal et al.’s (2010) paper also includes breakdowns by ethnicity. Among non-hispanic whites, and 31.9% and 33% are obese. Among non-hispanic blacks, percentages are 37.3% and 49.6% respectively; and among hispanics, 34.3% and 43.0%, respectively. Across ethnicities, then, women are more likely to be obese than men; and non-hispanic blacks and all hispanics have greater levels of overweight and obesity than non-hispanic whites.

### Table 2.2: Median BMI and Percentage of Population by BMI, Gender and Age Group, 2007-8

#### Median BMI (95% Confidence Interval)

<table>
<thead>
<tr>
<th>Age</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>20 to 39</td>
<td>26.6 (26.1-27.2)</td>
<td>26.5 (25.7-27.5)</td>
</tr>
<tr>
<td>40 to 59</td>
<td>28.3 (27.7-29.0)</td>
<td>27.7 (27.0-28.5)</td>
</tr>
<tr>
<td>60+</td>
<td>28.3 (27.9-28.7)</td>
<td>27.6 (26.9-28.3)</td>
</tr>
</tbody>
</table>

#### Percent of Population by BMI Range (95% Confidence Interval)

<table>
<thead>
<tr>
<th>BMI Range</th>
<th>Men</th>
<th>Women</th>
<th>Overall</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI &lt; 25</td>
<td>27.7 (25.9-29.6)</td>
<td>35.9 (33.1-38.7)</td>
<td>32.0 (30.2-33.7)</td>
</tr>
<tr>
<td>BMI ≥ 25</td>
<td>72.3 (70.4-74.1)</td>
<td>64.1 (61.3-66.9)</td>
<td>68 (66.3-69.8)</td>
</tr>
<tr>
<td>BMI ≥ 30</td>
<td>32.2 (29.5-35.0)</td>
<td>35.5 (33.2-37.7)</td>
<td>33.8 (31.6-36.0)</td>
</tr>
<tr>
<td>BMI ≥ 35</td>
<td>10.7 (9.1-12.3)</td>
<td>17.8 (15.8-19.8)</td>
<td>14.3 (12.7-15.8)</td>
</tr>
<tr>
<td>BMI ≥ 40</td>
<td>4.2 (3.3-5.1)</td>
<td>7.2 (6.1-8.4)</td>
<td>5.7 (4.9-6.6)</td>
</tr>
</tbody>
</table>

Sources: Flegal et al. (2010), and author's calculations.
The visualization techniques developed by the CDC / NCHS, and exemplified by Figure 2.2 below, have shown increasing rates of obesity and diabetes across the United States, as well as immense regional variation (CDC MMWR 2009, 58(45), p. 1261). In Figure 2.2, we see high percentages of diabetes in the South and East of the United States, as well as pockets in Oklahoma, Texas, Arizona, Montana, and both North and South Dakota. We see very high rates of obesity in the South, Alaska, many central States, and Nevada.

The most recent publications on the NHANES data does not include analyses of overweight and obesity by poverty level. The Health United States 2009 Report, however, does, using estimates from 2006-7. Table 72 (not shown) breaks down data by percent of poverty level, with three categories defined as below 100% of the poverty level, between 100 and 200% of the poverty level, and above 200% of the poverty level. Percentages of obesity among the populations of each category are: 35.9% below 100%; 36.7% between 100% and 200%, and 33.1% above 200%.
Because these numbers do not have confidence intervals in the Health USA 2009 Report, it is impossible to say if these estimates differ significantly across poverty levels. The only noticeable difference is between the above 200% category and the two others: there are unsurprisingly fewer obese people among the richest. Of note, however, is how people who earn just above 200% of the poverty level in the United States can’t really be called wealthy, especially in big cities; the class analyses thus presented in the literature may not provide the most accurate portrait of class-based inequalities in weight status, and will require further investigation.

2.2.2 Children

Obesity among infants is defined as weight for length above the 95th percentile, and as BMIs above the 95th percentile for their age for youth above 2 years of age, where percentiles based on CDC growth curves. Ogden et al.’s (2010) analyses of 2007-8 NHANES data showed that 9.5% of infants and toddlers were at or above the 95th percentile of weight for length, and that 16.9% of children and adolescents 2-19 were above the 95th percentile of BMI. They also found statistically significant differences by race / ethnic group. Compared to non-hispanic whites, non-hispanic black boys were 1.12 times more likely to be above the 95th percentile (not significant), and non-hispanic black girls were 1.70 times more likely (significant). Among hispanics, boys were 1.80 times more likely to be over the 97th percentile than whites (significant), and Hispanic girls 1.21 times more likely (although not significantly so). These ratios point to complex patterns of BMI among minority youth.
Conclusion

In sum, people have been getting heavier, and presumably fatter, although weight gain seems to have slowed down recently. While it may be true, as argued by Campos (2004), that having the higher cutoff point for the healthy BMI range at 25, so very close to the population median weight, may have made rates go up with a minor weight gain on average in the population, as noted by Flegal (2006), aggregate level statistics often mask the actual micro differences. Almost everyone in the U.S. population has been gaining weight, but it is the obese category that has gotten larger, probably by more than a few pounds. The general skewing of population BMI distributions at all age groups over the past 50 years has lead us with a very large population of large people: more than a third of the population at a BMI above 30.
Chapter 3: Not to Be Fat. Obesity Research as Social Enterprise.

In fat-obsessed cultures we are all ‘lipoliterates’ who ‘read’ fat for what we believe it tells us about a person. This includes not only their moral character but also their health (Graham 2005, p. 179).

In this chapter, I quickly review some of the main criticisms that are directed at the medical literature covered in Chapters 1 and 2. My goal is not to fatally undermine this literature, but rather to show its limitations and blind spots. I first review what I call the “fat scholar critique,” before turning to the literature on fat stigmatization.

Unlike in previous chapters, I often use “fat” rather than obese and overweight to designate large people. Many of the most vocal critiques of the current medical paradigm reject the “O” terms (overweight and obesity) explicitly as biased and prejudiced, and thus as participating in the pathologization of fat (Wann 2009). A group of self-proclaimed “fat scholars” and allies have argued along similar lines as Rhode (2010), who writes that “experts generally agree that weight is not simply a matter of willpower. Weight reflects a complex interaction of physiological, psychological, socioeconomic, and cultural factors” (p. 42).

Others, including Wann (2009), have argued that weight is just like height or other bodily characteristic: its distribution in the population will always follow a bell curve, such that there will always be both thin and fat people, just like there will always be short and tall people (p. ix). To be prescriptive when it comes to weight, then, she and others argue, is just as ridiculous as being prescriptive about height. Because I take this critique seriously, in this chapter and in the ones that follow, I use the language chosen by the authors I am reviewing.
The main insight from this critical literature is that medical research is embedded in a fat-phobic social context, and that this context matters in the design and interpretation of research on the health costs of body size and bodily fat.

3.1 Skeletons in the Medical Closet – The Fat Scholar Critique

There have been many critics of medical research when it comes to the study of overweight and obesity. A subset of these, inspired by a critical studies (and feminist) perspective, evaluates claims through their positioning in the knowledge-power structure. This literature, which is reviewed in Chapter 5, highlights how medical doctors and medical researchers are also participants in a broader culture that is fat-phobic and sexist, and benefit financially and scientifically from the study of fat in a specific historical moment where the culture at large and its constitutive institutions have declared a “war on fat.”

I focus here on the critiques that are addressed directly at the medical establishment, using their tools rather than those of historical analysis, sociological inquiry, or critical theory. Some of these critiques get published in medical journals, but they can almost be said to be “fringe” critics: they do not get the same number of citations as scientists working within the paradigm, and whose work I reviewed in Chapters 1 and 2. The reason? I argue in Part II that a long history of medical research has established a trajectory upon which medical research travels at high velocity, and thus has lots of inertia. So much so that a large proportion of the critiques of the current research paradigm also use science to justify their stance. The “counter paradigm” I describe here attempts to change the course of medical research and public policy, by providing an alternative story and suggesting a different course of action to intervene in public health.
In 2006, the *International Journal of Epidemiology (IJE)* organized a special edition focusing on overweight and obesity. Among the articles in this special edition, they published an article by an interdisciplinary team of scholars attempting to “debunk” the current medical paradigm that creates hype for the obesity epidemic and its health consequences. *IJE* also published seven different responses to the article by Paul Campos, Abigail Saguy, Paul Ersnberger, Eric Oliver and Glenn Gaesser. Of the seven articles, all but two were antagonistic. One of the supportive critiques came from fat activist Susie Orbach (2006), author of the feminist classic *Fat is a Feminist Issue* (Orbach 1997 [1978]), and from weight and fitness research specialist Steven N. Blair and La Monte (2006), specialists of exercise and health. Orbach wrote a piece titled “There is a public health crisis—it’s not fat on the body but fat in the mind and the fat of profits.” Blair and La Monte pushed the agenda of cardiorespiratory fitness and exercise as a potential mitigating factor in the interaction between BMI and ill health.

In their article, Campos et al. (2006) evaluate:

four central claims made by those who are calling for intensifying the war on fat: that obesity is an epidemic; that overweight and obesity are major contributors to mortality; that higher than average adiposity is pathological and a primary direct cause of disease; and that significant long-term weight loss is both medically beneficial and a practical goal (Campos et al. 2006, p. 55).

In what follows, I use the four claims above as an organizing principle to structure the fat scholar critique and evaluate its empirical grounding. My motivation to use the Campos et al. (2006) article as a starting point is twofold: First, *IJE* is the third most influential epidemiology journal according to ISI Web of Knowledge’s Article Influence Score. At 2.497, *IJE*’s ISI influence score doesn’t compare well with the *New England Journal of Medicine*’s 19.870 or the *Journal of the American Medical Association*’s 11.432. Yet in the epidemiology subfield, it is very close to the leader, the *American Journal of Epidemiology*,
whose score is 2.552. If *IJE* decided this article was worth publishing in their pages, along with a set of responses, it is probably because the editors felt the dialogue was worthwhile, and that the counter-paradigm deserved to reach a broad audience. Second, the authors of this article are prominent scholars within the field of fat studies, and among the most vocal critiques of the current paradigm. This article is thus a summary of the main attacks that this counter-paradigm has waged against the dominant paradigm that I described in Chapters 1 and 2.

3.1.1 *What Is the Obesity Epidemic?*

As we will see in Chapter 6 the use of the word “epidemic” to discuss obesity is a very recent phenomenon. Campos et al. (2006) take issue with this language, and claim that to see something as an epidemic should imply “an exponential pattern of growth” (p. 55). Similarly, Boero (2007) argues that the obesity epidemic should be seen as a “post-modern epidemic,” one where a phenomenon gets “cast in the language and moral panic of more ‘traditional’ epidemics” (p. 42). “What makes the ‘obesity epidemic’ unique,” she writes,

is that we are all at risk for obesity; what varies is our degree of risk. … Indeed, in an era of personal responsibility for health, one no longer need manifest any concrete symptoms to be considered at risk for any given disease (Boero 2007, p. 42).

Two of the seven response articles to Campos et al. (2006) addressed the meaning of “epidemic” in the case of obesity directly. Stevens, McClain, and Truesdale’s (2006) short response is to cite definitions of “epidemic” from both a general medical dictionary and an epidemiology textbook and note that they are not “confined to infectious diseases but rather rest on a health phenomenon being ‘clearly in excess of normal expectancy’” (p. 77).
Similarly, Katherine Flegal (2006), investigator at the NIH/CDC who was a lead author on many of the NHANES papers examined in Chapter 2, points her readers toward both lay and technical definitions of “epidemic.” First, from the *Dictionary of Epidemiology*, she reads “The occurrence in a community or region of cases of an illness … or other health related events clearly in excess of normal expectancy” (Flegal 2006, p. 72). Second, she reads from the *Webster Dictionary*, where among the uses of epidemic as an adjective one finds: “2. Widely prevalent (epidemic anxiety)” (ibidem, p. 72). Clearly, anxiety is not an infectious disease, and its spread would not follow an exponential growth curve.

That the use by the medical community of “epidemic” to talk about obesity would signify moral outrage is dismissed by the direct commentators on Campos et al. (2006) cited above. They all mobilize scientific (linguistic and medical) definitions, rather than address what the word evokes in lay people’s minds. I would like to argue that it is difficult to separate both. If we give benefit of the doubt to medical doctors when it comes to their own usage of the epidemic language (see Chapter 6 for a historical exploration of this issue), we nevertheless have to acknowledge the intellectual associations non-specialists may make when confronted with the idea of an “epidemic of obesity.” While to my knowledge nobody has evaluated this issue yet, it seems fair to think that ideas about very high rates of affliction, contagion, and danger would come up regularly.

### 3.1.2 Fat, Mortality and Morbidity

I have discussed in Chapters 1 and 2 the evidence medical researchers use to supports the claim that fat has mortality and morbidity costs, and will not return to it here. The critique raised by Campos et al. (2006) is based on findings from two articles:
the one by Flegal et al. (2005) covered in section 1.1.2 along with its critiques, and one by Durazo-Arvizu et al. (1998), which requires further consideration.

Durazo-Arvizu et al.’s *American Journal of Epidemiology* paper should have been explosive, for they contradict current beliefs. Indeed, in the abstract we read that based on their statistical analyses on NHANES I data,

The BMI of minimum mortality was 27.1 for black men (95% confidence interval (CI) 24.8-29.4), 26.8 for black women (95% CI 24.7-28.9), 24.8 for white men (95% CI 23.8-25.9), and 24.3 for white women (95% CI 23.3-25.4). Each confidence interval included the group average. Analyses conducted by smoking status and after exclusion of persons with baseline illness and persons who died during the first 4 years of follow-up led to virtually identical estimates. The authors determined the range of values over which risk of all-cause mortality would increase no more than 20% in comparison with the minimum. This interval was nine BMI units wide, and it included 70% of the population (Durazo-Arvizu et al. 1998, 739).

When the 30% of the population that falls outside the confidence interval defined by Durazo-Arvizu and colleagues is understood to include also underweight people, the percentage of obese people who are at risk of increased mortality appears to be smaller than the total percentage of obese people in the population (at 33.8% in 2007-8).

Strangely, the article didn’t get much attention, collecting only 70 citations in ISI Web of Knowledge, and 3 in PubMed. In comparison, the article by Allison et al. (1999) titled “Annual deaths attributable to obesity in the United States,” which listed 280,184 deaths, was cited 793 times in ISI Web of Knowledge, and 58 times in PubMed. The causes of the general overlooking of Durazo-Arvisu et al.’s article by the research community are unknown, but certainly puzzling.

Where the fat scholar critique of the connection between morbidity and mortality has a more unequivocal bite is with the role played by cardiorespiratory fitness. Counter-intuitively given the current policy emphasis on exercise (see Chapter 7), cardiorespiratory fitness remains understudied, and under-acknowledged in the literature on the health
effects of fat. Very few of the most-cited articles on obesity include an assessment of either (a) exercise as a strategy for weight loss, or (b) of cardiorespiratory fitness as a mitigator of the negative health effects of obesity. To investigate the place taken by cardiorespiratory fitness in the medical literature, I queried the ISI Web of Knowledge database and reviewed the titles and abstracts of the 50 most-cited articles with “obesity” in their title (average citation number = 1,298; s.d = 527.7; see full list of references in Appendix II).

None of these articles used the words “exercise,” “fitness,” “cardiorespiratory health,” or “activity” in their titles, and only three (6%) mentioned one of them in their abstract. The most cited of these three, by DeFronzo and Ferrannini (1991; 2,615 citations, rank 3 / 50), is a review of the evidence on insulin resistance. The only mention of physical activity notes that it enhances insulin sensitivity. The second most cited article, by Mokdad et al. (2001; 1,153 citations, rank 25 / 50)\(^5\), presents population-level data on what is called, in Table 4, “Specific Weight Control Practices by Selected Characteristics Among Persons Trying to Lose or Maintain Weight” based on Behavioral Risk Factor Surveillance System data from 2000, and in comparison with 1996 data. Data on these practices (dieting, eating fruits and vegetables, physical activity) are presented as percentages, but all based on self-reported practices, which are dubious measures of actual behavior and cardiorespiratory fitness.

At any rate, the Mokdad et al. (2001) paper is not investing causality, but rather inferring it. It is descriptive, notes the high prevalence of physical inactivity in the population (27%), but investigates neither of (a) or (b) above. Mokdad et al.’s last sentence notes the importance of physical activity: “To control these dual epidemics [of

\(^5\) See more analyses of the Mokdad et al. (2001) paper in Chapter 3 and Chapter 4.
diabetes and obesity], now is the time for implementing multicomponent (sic!) interventions for weight control, healthy eating, and physical activity” (p. 1199).

The third most cited article, Kopelman (2000; 1,070 citations, rank 32 /50), is a review article that makes the case that obesity is a medical problem of global import. Kopelman mentions physical activity in three occasions. First, in a section on energy expenditure, in which he notes that reduced exercise levels in populations worldwide is a major component in population-level weight gain, and notes increased risks of overweight and obesity for people with low levels of physical activity. Second, in a section on culture, where weight gain is noted after marriage and with gender parity. And third, in his conclusion, and the final sentence that recommends, beyond fundamental research in the biological causes of obesity,

the development and implementation of effective programmes that successfully encourage increased physical activity and healthy eating across populations … for the prevention of obesity and its associated diseases (Kopelman 2000, p. 642).

In sum, of the 50 most-cited articles on obesity, only three mention physical activity. Of these, none investigates two of the main potential of exercise as a weight loss strategy, or the mitigating effect of cardiorespiratory fitness on BMI. The benefits of exercise are taken for granted in two of these articles, and a mechanism for increased insulin sensitivity in the other; and the mitigating impact of cardiorespiratory fitness is not investigated.

There are exceptions to the rule, and they are certainly noted by fat scholars. A range of studies by Blair and associates suggest that cardiorespiratory fitness may be the main factor in defining poor health outcomes, such that sedentarity would be the main culprit (Blair and Brodney 1999; Blair and Church 2004; Blair and LaMonte 2006; Sui et al. 2007; Wilkinson and Blair 2004). The most recent of these studies, Sui et al. (2007),
was published in JAMA, and notes how the effect of waist circumference (as proxy for adiposity) on mortality among adults aged 60 and over disappeared after controlling for cardiorespiratory fitness, with p values shifting from p=0.02 before control to p=0.86 with control. Fitness also significantly predicted mortality risk across almost all risk factors.

Another article, by Katzmarzyk et al. (2005), suggests that once we control for cardiorespiratory fitness, the risk ratios of all-cause mortality for normal weight, overweight and obese individuals is not statistically significant.

During an informal interview, Steven Blair, co-author on the articles on exercise cited above, confirmed that very few obesity research projects control for fitness level, and how those who do use a self-reported measure of exercise habits as a control. According to his own calculations, self-reported exercise levels correlate at about r = 0.3 or 0.4 with actual cardiorespiratory fitness, as measured through treadmill tests, making the variable basically useless (interview with Steven N. Blair, 21 September 2010).

The question remains: Can one be fat and fit? What is the correlation between weight and cardiorespiratory fitness? It depends on how you look at it. Sui et al. (2007) defined “unfit” as the lowest 20% of the fitness distribution of the whole sample, as per a treadmill test. In defining fitness as the top four quintiles of measured fitness, which led to the distribution of fitness by BMI showed in Figure 3.1.

Figure 3.1. suggests that while there is an increasing number of “unfit” people with increasing BMI, and that there is more than 100% more unfit individuals with BMIs in the 30.0-34.9 BMI range than in the overweight range (10.0% vs. 23.1%), it is only at

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6 Sample sizes were very small among individuals with BMIs above 30 in the sample, reducing statistical power. When using other indicators such as waist circumference and percent body fat, which yielded larger subgroups, the trends were highly significant.
BMIs at or above 35 that the unfit surpasses the fit. The correlation is indeed very high, at about 0.93.

Figure 3.2 shows the full distribution of BMIs by fitness level. The three-dimensional graph shows two local maxima and two local minima. The absolute and first local maximum is found at the 1st quintile of fitness, BMI over 35 coordinates: 54.1 percent of first-quartile fitness individuals have BMIs at or above 35, suggesting that the heavier individuals in the sample were in majority unfit. The second local maximum is found at the 5th fitness quintile, lowest BMI category, suggesting that a large proportion, yet still a minority, of lean people score among the fittest on treadmill tests (41.3%). The local and absolute minimum is found in the fifth fitness quintile, highest BMI coordinate, where we find 1.6% of the sample, suggesting that there are some, but very few highly obese people who are really fit. The second local minimum is found at the first quintile, lowest BMI coordinate: 6.3% of “normal weight” individuals are unfit. The chi-square statistic for these data is 456.85 (df=12), and significant at p < 0.000001.
Fig. 3.1: Percentage of Fit and Unfit Individuals in ACLS Sample, by BMI
Figure 3.2: Percentage of ACLS subjects by BMI and Fitness Quintile
There are two take-home lessons from these numbers. First, we should pay greater attention to the health and exercise needs of larger people, meanwhile encouraging everyone, thin and fat, to exercise and care for their cardiorespiratory health. This will require public health messages that emphasize the joy of movement for its own sake and for its health-associated benefits, and public investment in spaces where people of large sizes can exercise safely. Some studies suggest that mixed-weight, gender-specific group interventions may work to instill good exercise habits and improve health markers (Flowers 1976; Robinson 2010). Second, while the association between BMI and cardiorespiratory fitness is very high, the existence of fat and fit individuals, as well as the dampening effect of low levels of cardiorespiratory fitness on mortality should reassure us that prescribing exercise is a really productive suggestion. Small improvements in cardiorespiratory health may lead to serious overall health benefits.

3.1.3 The Data on Weight Loss

The final element in Campos et al.’s (2006) critique chastises the medical establishment for promoting weight loss when no weight-loss method has been found to be universally effective at inducing permanent weight loss, and that most people who do lose weight re-gain it over time. On this point, the evidence is also in their favor.

According to Puhl and Heuer (2010) and Wadden, Brownell, and Foster (2002), there is consensus in the scientific community that weight loss, and weight maintenance after weight loss, are significant challenges for which there is absolutely no panacea. A meta-analysis by Dansinger et al. (2007) highlighted the poor methodology of most recent diet-based weight-loss studies, and the overall very small weight loss associated with these studies, and the typical re-gain.
The NIH (1998) Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults, Evidence Report result from a conference of experts in obesity research and treatment. In its section 3.B. titled “What treatments are effective?,” we learn that randomized controlled trials, participants lose a small percentage of their mass when given dietary advice (in the order of 2-10% when combined with other strategies, including exercise and behavioral therapy), but that they typically regain it. There is also no improvement of cardiorespiratory fitness among participants in weight-loss programs who do not exercise as a part of their weight loss strategy. Of those who exercise, there is improvement in cardiorespiratory fitness, small weight loss, small loss of abdominal fat, and slower regain.

Behavioral therapy has been shown to be effective in the short term, but inefficient without follow-up in the longer term. A combination of exercising, diet, and behavioral therapy was recommended to maximize the impact of treatments. No long-term effects of pharmacotherapy on weight have been documented, yet they are still recommended.

Earlier NIH consensus statements on surgery for severe obesity acknowledged the same difficulties: non-surgical strategies for weight loss among the morbidly obese have not proven successful (NIH 1978 and 1991 Consensus Statements). While in 1978 surgical procedures were found to have many undesirable and non-trivial side effects. By 1991, those were found by the NIH to be much less significant. As of 1998, the only clear weight loss results came with surgery, a draconian solution recommended to adults with BMIs greater or equal to 40, or for adults with BMIs over 35 with comorbidities (defined as other signs of ill health, such as diabetes, a history of cardiovascular disease, etc.). Comorbidities also showed improvement after surgery. Today, however, there is much
controversy about the long-term effects of surgery, and documented increases in completed suicides (Adams et al. 2007; Tindle et al. 2010).

Gaesser (2009), one of the co-authors of the Campos et al. (2006) piece, discusses the irony that weight gain has happened in the United States despite American’s increasing efforts to lose weight. He criticizes studies of “permanent weight loss” that define success as maintenance of weight loss for a one-year-long interval, while it is known that most people regain the lost weight over a period of one to three years. The database that comes under fire is the “National Weight Control Registry” (NWCR), a registry that is based on voluntary signup by people having lost at least 30 pounds and kept it off for a year, and which serves as a basis for the publication of papers on “permanent” weight loss. Of approximately 80 million U.S. adults who are currently trying to lose weight, Gaesser points out, the 5,000 members in the NWCR constitutes very far from a large or random sample of all dieting adults, pointing to the rarity of success stories, but also the ubiquitous failure of dieting.

A review article by Mann et al. (2007) comes to a similar conclusion. If there is clear evidence about dieting, they write, it is that (1) it rarely results in weight losses of more than 10 to 15% of initial body mass, barely changing the risk profile of the very obese, (2) the weight lost is regained. They cite Garner and Wooley, 1991, who wrote: “It is only the rate of weight regain, not the fact of weight regain, that appears open to debate” (Garner and Wooley, 1991, cited in Mann et al. 2007, p. 221).

Others have noted how weight cycling may be more dangerous health-wise than being overweight (Cai et al., 2010; Mann et al., 2007; Wannamethee et al., 2002) except among the very heaviest individuals (Blair et al., 1993). Dieting is also argued to participate in weight gain by slowing metabolic rate (Blackburn et al. 1989). Most studies
of obesity, then, confound two inter-related variables and may confuse the effects of one for the effects of the other: using weight as a proxy for the effects of dieting and weight-cycling, and vilifying fat rather than the unhealthy behaviors associated with it (Bacon 2006; Germov and Williams 1996; Saguy and Riley 2005). The lack of research in that area is often deplored (NIH 1998 Evidence Report; Puhl and Brownell 2006).

There is also very little research connecting weight loss to lower risks of mortality. Among studies that address this connection, the distinction between intentional and non-intentional weight loss is rarely made, such that it is unclear if there are substantial benefits to intentional weight loss, as non-intentional weight-loss is typically related to serious illness (NIH 1998 Evidence Report). Intentional weight loss studies are also plagued with the same methodological limitations as other studies: that they don’t control for cardiorespiratory fitness, and in many case do not control for intervention effect. Furthermore, a very recent study by Cai et al. (2010) has shown that while the obese had significantly shorter life spans than normal-weight individuals, obese people who had lost weight in middle age had the lowest chance of surviving to age 65, and the shortest life expectancies.

In this light, it appears that an emphasis on weight loss in public health is potentially hazardous to patients’ health, and may only increase their frustration and weight cycling. In its 1985 Consensus Statement on the Health Implications of Obesity, the NIH noted that “Obesity creates an enormous psychological burden. In fact, in terms of suffering, this burden may be the greatest adverse effect of obesity” (NIH, 1985). To focus on weight loss rather than on improving the health of all may only be adding to this psychological burden, and further stigmatize large people.
Finally, it is sometimes argued that the reason why obese people are obese is that they are not trying to lose or control their weight, and that they simply do not know that carrying extra weight leads to negative health outcomes. As early as 1994, the New York Times was publishing an article titled “Despite awareness of risk, more in U.S. are getting fat,” clearly noting the paradox (Burros 1994). Mokdad et al. (2001) provide a stronger illustration of the same point, using Behavioral Risk Survey System data from 2000 (see Table 3.1, below). First, among the obese, 65.7% mentioned trying to lose weight, compared with 20.8% who were trying to maintain their weight, and 13.5% who were neither trying to lose or to maintain weight. In other words, only about 1 in 7 obese individuals is not weight conscious, which makes the “out of control” and “ignorance” hypotheses unlikely. Another argument against these hypotheses is found in the prevalence of weight loss advice given to obese individuals during a routine checkup over the past year. Indeed, 42.8% of obese people recalled being advised to lose weight. While it is difficult to predict the direction in which the estimate is biased, given that this question was only asked of people trying to lose or maintain weight, it is clear that a large proportion of obese people are “made aware” of weight as a health issue during their annual medical exams.

In sum, the Campos et al. (2006) article that framed this section is a highly polemic piece that had the potential to generate dialogue among epidemiologists and other obesity researchers about the assumptions of medical research on obesity. The article itself got 35 citations in the ISI Web of Knowledge database, including 5 immediate citations from the seven critical, immediate responses published in IJE. The article, then, can’t be said to have sparked the wide debate it was expecting.
3.2 Stigmatization, Discrimination, and Barriers to Care

We have seen in Chapter 1 that there is no clear evidence that being obese leads to the development of mental health problems, but rather that mental health problems are sometimes one of the causes of weight gain, maybe as a side effect of medication. There is, however, a large literature documenting the effects that being overweight has on life chances, employment, marriage, etc. Very recently, Puhl and Heuer (2010), in their discussion of the public health implications of weight stigma, noted that it has not received enough attention in public health and medical circles, despite “5 decades of scientific research documenting weight stigma and its consequences for obese individuals” (p. 1020). As early as 1968, they write, the author of an article titled “The Stigma of Obesity” was amazed by the omission of obesity from “the writings of sociologists” and from the “literature on social deviance,” meanwhile “in our kind of
society, with its stress on affluence and upward mobility, being overweight is considered to be detrimental to health, a blemish to appearance, and a social disgrace” (Cahnman 1968, cited in Puhl and Heuer 2010, p. 1020). I review the literature on obesity and stigmatization and barriers to care briefly below.

3.2.1 Fat Discrimination in Everyday Life

While obesity has only recently been formally defined as a disease, it has been a stigmatized condition for almost a century (see Chapter 5). Symbolic interactionists like Erving Goffman (1986 [1963]) note the way illness or disability carry stigma and upset the way people generally interact. Susan Sontag (1990) argued convincingly that diseases are associated with meanings that impact how healthy people interact with affected people, and the way affected people perceive themselves and their condition, and thus how they manage their identity. Social psychological research has shown that people respond with feelings of anxiety in interactions with stigmatized people (Crocker, Major & Steele, 1998; Blascovich et al., 2001), a phenomenon they have called “stigma threat.” The consequences of this stigmatization have been documented by social scientists across research paradigms.

Rhode (2010) reviews evidence that points to weight stigmatization and discrimination. In nationally representative surveys, over 50% of women report they would prefer to be hit by a truck than be fat; two thirds would rather be mean or stupid. More than a third of obese individuals would risk death to lose just 10% of their weight (p. 6). There is more: sixty and forty percent of overweight women and men respectively report experiences of weight discrimination; weight is consistently associated with a significant income penalty; obese women are more likely to be poor, even after controlling for other characteristics such as education level and cognitive ability; obesity
matters on the dating and marriage markets; it skews people’s evaluation of competence, job performance and social skills; and overweight individuals are seen as less likeable, less well adjusted, and lacking self-control and self-discipline (pp. 24-8).

Wann’s (2009) review provides a similarly grim portrait of the cost of fat phobia to individuals of all weights. Among youth, she notes that girls as young as five fear weight gain; fat kids get teased more and may in turn tease or bully others; fat children are more depressed and isolated, and those who don’t think they are the right weight contemplate suicide more often. Most young women have a disordered relationship to food; and fat students are less likely to get encouraged to go to college (p. xix).

Puhl and Brownell (2001) note that negative stereotypes against obese people are already established in American children by age 8, that obese children have lower self-esteem, that about 90% of overweight children are ashamed of being fat and believe that others would stop teasing them if they lost weight. Normal weight youth get more financial support from their parents to go to college than overweight ones, suggesting (unconscious?) parental bias (p. 796).

A recent study of gender, obesity and education by Crosnoe (2007) showed gendered patterns of transition to college. Obese girls were left behind, entering college at lower rates than non-obese girls, especially in school contexts where obesity is uncommon. Obese boys, on the other hand, were unaffected. Potential mechanisms included greater internalizing symptoms, self-medication, and academic disengagement.

According to an analysis of the social networks of 17,557 adolescents aged 13 to 18 in the Add Health study, Strauss and Pollack (2003) found that while overweight adolescents (defined at BMI > 95th percentile) listed the same number of friends as non-overweight adolescents, they were significantly more likely to receive no friendship
nominations (OR 1.71, 95% CI 1.39-2.20), and received fewer nominations than normal-weight students. Participation in sports and school clubs mitigated that effect.

Among adults, weight discrimination is seen in the workplace in the distressing statistic that 93% of human resources professionals admit they would hire a normal-weight individual over a fat one, and fat applicants are often rejected for not fitting a corporate image; the fattest women earn one fourth less than normal-weight women; fat employees have lower rates of promotion, and can get fired simply for being fat (Wann 2009, pp. xix-xx). Obese workers also perceive consistent weight-based disparities, disparities that are confirmed by experimental and survey data (Puhl and Heuer 2009). It is also seen in lower marriage rates and higher levels of poverty (Ernsberger 2009; Gortmaker et al. 1993).

A recent study by Glass, Haas and Reiter (2010) evaluated the role of BMI in career attainment, and found that the main reason obese women lag behind leaner counterparts is that they tend to obtain fewer post-secondary degrees than leaner counterpart, indirectly supporting Crosnec’s findings.

### 3.2.2 Fat Discrimination in Health Care

When it comes to medical care, the bias against fat people is also well documented. Fontaine et al. (1998) investigated the impact of BMI in women’s use of health services using nationally-representative data. Controlling for age, race, income, education, smoking, and health insurance status, they found that women with BMIs above 35 were more likely to delay clinical breast examinations (OR 1.26, 95% CI 1.00-1.58), gynecologic examinations (1.39, 95% CI 1.15-1.69), and Pap smears (1.29; 95% CI 1.04-1.58), than women with BMIs below 25. Fontaine et al. concluded that “increased BMI is associated with decreased preventive health care services, which may exacerbate
or even account for some of the increased health risks of obesity” (p. 381), including cancers, which are documented to be more prevalent among the obese, and could be prevented or addressed if found earlier (p. 383). In her editorial comment on the Fontaine et al. (1998) paper, Yanovski (1998) notes how clinicians’ attitudes toward obese patients mirror those of the population in general, and that laissez-faire may have serious health consequences for the obese.

Later work has shown that it is not only laissez-faire that is leading to adverse health outcomes for fat patients. Puhl and Brownell (2001) review the documented effects of obesity in employment, education, and health care. They show the prevalence of stereotypes that attribute laziness, lack of self-control, and indulgence to obese patients among medical practitioners. When it comes to actual care, nurses admitted in large numbers that they would rather not have to care after or touch obese patients, and family physicians reported not feeling responsible for their obese patients’ weight loss, while feeling discouraged by perceived lack of compliance and motivation. On patients’ side, shame with weight, and ritual weigh-ins were connected to cancellation of appointments and to delays in seeking medical advice.

A study by Puhl and Brownell (2006) with 2,449 obese women, doctors were the second-most reported source of stigma out of more than 20 possibilities. 69% of respondents had perceived weight-based stigma in their doctor, and 52% had repeatedly so.

Andreyeva, Puhl and Brownell’s (2008) study of weight discrimination notes that the prevalence of weight/height discrimination has increased from 7% to 12% between 1995-6 and 2004-6 in a survey of 3,437 adults in the United States. An updated review of the literature on stigma by Puhl and Heuer (2009) notes that stereotypes about obese
people are common internationally among primary care physicians (p. 944). Primary-care doctors generally believe that obesity is a behavioral problem caused by inactivity and overeating. Experimental results with standardized patients with different BMIs and genders confirmed that physicians see heavier patients as less healthy, worse at self-care and less self-disciplined than their otherwise-identical leaner equivalents. Implicit Association Test studies showed a strong implicit antifat bias among clinicians and researchers specialized in obesity. Similar findings were found across categories of health professionals. When it comes to actual care, doctors feel unprepared to assist obese patients, who, in turn, get less time with their doctors and less health education during doctor visits.

3.3 Why obesity is not the new smoking

Obesity has been likened to smoking discursively recently. Five different arguments are typically made to compare them, some more problematic than others. First, both are perceived as serious public health issues: both were the subject of public health reports by the World Health Organization, and both were called global epidemics (WHO 1998, 2009). The strength of the association between smoking and ill health, including mortality, is of a much greater magnitude than the association between obesity and ill health, including mortality, however. As seen in Chapter 1, relative risks and odds ratios were mostly in the 1 to 7 range, and thus much smaller than the risks associated with smoking (Ross 2005).

Second, they have been stigmatized in recent decades, partly through public health campaigns (for fat stigmatization: Bayer 2008a, Bell et al. 2010b; for tobacco

7 Others, such as Escalera (2009), have noted the strong connection between racism and fat bias in implicit association tests.
stigmatization Burris 2008). Some believe that stigmatizing smokers and obese people will lead them to curb their destructive behavior (smoking and overeating / not exercising), such that a short-term discomfort is traded for the long-term benefit of smoking cessation or weight loss. This belief is highly problematic in the case of obese people. On the one hand, if there is evidence that smoking cessation has clear health benefits, as we have seen earlier there is (1) no proven method for permanent weight loss, (2) little data that proves that weight loss is beneficial for the obese, but (3) clear data that weight cycling is damaging for health. As such, stigmatizing obese people is likely to yield more harm than good.

On the other hand, there is substantial current debate about the value of stigmatization as a public health strategy to eradicate smoking. In a special edition of Social Science & Medicine on stigma and health, tobacco scholars got up in arms about the use of stigma in reducing incidence of tobacco use. In tracing a history of the public health discourse on stigma, Bayer (2008a) notes that in the case of HIV / AIDS, addressing stigma was seen as a key part of an effective tackling of the epidemic, a strategy that has been echoed by Bell et al.’s (2010b) discussion of substance abuse, minus tobacco (see p. 795). Yet for Bayer (2008a), stigmatizing public health policies may be inequitable in the near term. But if they work, they may represent a significant contribution to the wellbeing of the very people they burden and on those who might be dissuaded from engaging in behaviors that have profound implications for health on a population level (Bayer 2008a, p. 470).

Burris (2008) and Bell et al. (2010b) both take issue with this point. Burris (2008) argues that we not see stigma as a matter of degree, but rather as an absolute no-go public health strategy. Stigma not only discriminates against and dehumanizes the stigmatized, but it also make them “recognize [their] spoiled identity as a social reality” (p. 474), which
may lead to the internalization of prejudice and stereotype, and to ensuing social isolation and delays in seeking care. A cruel and arbitrary punishment according to Burris.

Bell et al. (2010b) are chiefly concerned with the reinforcement of inequalities that stigma brings about. They note how stigmatizing messages are more likely to alienate stigmatized smokers from their health care practitioners than to reduce their smoking behavior. In sum, the debate about the value of stigmatizing or shaming campaigns for tobacco should alert us to the role of stigma in “anti-obesity” campaigns. Fat scholars are united on this front: increasing stigma is only causing more harm than good, complicating health care provision, and limiting access.

Third, both obesity and smoking are seen as issues of lacking morality and self-regulation, and seen as unhealthy individual choices. There are significant problems with this parallel. In addressing the choice argument, I believe we have to distinguish between the initial decision to become obese or to start smoking, and the decision to continue to be obese or to continue smoking. It seems far-fetched to claim that in our fat-phobic culture, anyone would choose to be fat. If this really was indeed a choice, we would expect fewer people to be thin (Crossley 2006). Indeed, the statistics reviewed above show that kids very quickly internalize fat phobia, and fear weight gain – some do by age 3. It is thus unlikely that a kid would choose to be fat; yet there are fat kids. It is also unlikely that an adult would choose to be fat; yet there are fat adults. One may object that people do not really choose to be obese, but that they still choose an indolent, indulgent lifestyle of sloth and gluttony. Although there are indeed individuals who are “indulgent,” they are not universally fat; nor are lean individuals universally following the healthy living prescriptions we impose on the obese. Using weight as a proxy is condemning the former while giving a free pass on the latter.
In contrast, the valence of smoking varies by social location and has shifted over time. While throughout most of the 20th century smoking was a glamorous thing to do, starting in the 1980s it became less and less so. Yet, one may choose to start smoking: to be cool, to do as friends do, etc., and the fact that smoking rates have failed to decline among young people since 1990 (Sloan et al. 2003) seem to support this view. It may not be a fully rational choice, yet it is an event bounded in time and that can often be traced to a specific calculus.

When it comes to choosing to stay fat, the research reviewed above shows that losing weight permanently is no small feat. Fat people have been pressured into dieting for decades (see Chapter 5), yet if we trust epidemiological data, they have actually gotten fatter over time (see Chapter 2). As noted above, the stigmatization of fat people has increased, yet the number of obese people has also increased. Conversely, while stigma against smokers has been increasing (Bell et al. 2010a), there is evidence that one can stop smoking for good: smoking rates have declined substantially over the past decades, from 50% of men and 35% of women in 1964, to 19.6% of men and 16.6% of women in 2009.8

Fourth, tobacco has been likened to obesity through second-hand harm discourses of two kinds. A first line of argument notes the financial burden that everybody, lean or fat, pays for the health bills of obese individuals, is a first type of second-hand harm, affecting the “innocent,” lean, bystander, an argument put forth in the past with smoking. The jury is still out on this issue.

Another line of argument is found in the idea that obesity can be “socially contagious” and “spread” in family and friends networks. Christakis and Fowler (2007)

8 Sources: 1964 statistics found in Bayer 2008a, p. 466; 2009 statistics found on the CDC Behavioral Risk Factor Surveillance System website, http://www.cdc.gov/brfss/
found clusters of obese people spanning as many as three degrees of separation, suggesting that obese people tend to hang out together and thus influence each other. Their analyses of the weight of 12,067 individuals over time made them predict that having “alters” who become obese around you significantly increases your own chances of becoming obese, compared to a network where obesity is randomly distributed: a 57% increase when a friend becomes obese, a 171% chance when a close friend becomes obese, a 40% chance when a sibling becomes obese, and a 37% chance when your spouse becomes obese. The causal mechanisms they discuss are mostly psychosocial: changing norms about the acceptability of obesity, and ensuing changes in obesogenic behaviors.

In talking about obesity as a contagious disease, we face many problems, particularly in the definition of who is contagious and who is not, and in the design of solutions to this problem of contagion. In terms of identifying the contagious people in this epidemic, one must consider that not all obese people are obese because they overeat or are sedentary, and that some non-obese people overeat and are sedentary. Therefore, if the contagion agent is psychosocial, and body size is not a perfect indicator of norms and behaviors, it is difficult and unfair to blame obese people as a whole and exempt all the lean ones from scrutiny.

In terms of solutions, given that 34% of the American population is now obese and unlikely to lose any weight, it seems futile and counterproductive to “quarantine” or isolate them from others to avoid contagion, and also futile to stigmatize them, as stigma has health costs.

In sum, there are fundamental differences between obesity and smoking, fundamental differences between obese people and smokers, even as those two groups are often framed as reckless risk takers and pariahs who should be excluded from the
There have always been fat people, and will always be, while a world without smokers is possible. The health risks associated with smoking are clear, while the health risks associated with being fat often disappear once we control for even very low levels of cardiorespiratory fitness, as the literature reviewed above shows. Being fat is not a behavior, the way smoking is. And while weight is more malleable than height, pounds are still not easily shed and rarely shed forever, as the millions on Americans permanently on a diet and still gaining weight show (Campos et al. 2006; Gaesser 2009; Mokdad et al. 2001).

While there are indeed some similarities between smoking and obesity, they are generally superficial, and of limited help in defining the right course of action to take in addressing the consequences of obesity.

**Conclusion**

In this chapter, I have reviewed a literature that is critical of the way medical research is conducted, and that documents the significant consequences of weight stigma. The message sent to the American public is Manichean, despite the limitations of obesity research: fat is bad, lean is good; weight gain is bad, weight loss is good; fat people are ill, lean ones are healthy.

The alternative paradigm pushed by fat scholars is the Health at Every Size (HAES) framework, one where instead of focusing on weight loss, you focus on changing health-depleting behaviors: encouraging self-care in the form of intuitive eating (eating only until full, and only when hungry) and joyful movement, fighting isolation through

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9 In jurisdictions where health care is universal, such as Canada and the United Kingdom, these discussions are happening because of the shared costs argument. In the United States, still, the argument has surfaced, as insurance companies refuse health coverage for obese patients and lean consumers often angrily refuse to pay for their obese counterparts.
community building, and improving knowledge on nutritional contents and portion size. HAES tries to prevent iatrogenic harm, harm that is treatment related and greater than the problem for which treatment was prescribed, harm which seems all-too-likely in the case of weight loss prescriptions.

Given what we know (about the costs of weight cycling, about the health-depleting effects of stigma) and what we do not know (about the role of extra adipose tissue on health, net of prejudicial health care, stress, poverty, and cardiorespiratory fitness, for example), caution in health provision for fat patients is indicated.

Finally, the individualistic focus of almost all of the literature covered in Chapters 1, 2 and 3 alike is worth noting explicitly. Despite their divergences, most scholars of obesity see the individual as the privileged site for intervention, even as the blame is placed on the toxic environment, on socioeconomic factors, or in some cases, on the family and mothers (Boero 2009). System-wide solutions are regularly proposed (see, for example, Brownell, 1994; Kopelman, 2007; Nestle & Jacobson, 2000), but few are implemented in the very individualistic and non-interventionist context of the United States.

In the following chapter, I review models of obesity and discuss solutions that could be developed to address its consequences, however uncertain we are about them.
Chapter 4: Obesity, Its Models, Research and Policy Implications

4.1 The Causes of Obesity: Energy Imbalance and the Toxic Environment

At its simplest, the causal model of obesity has been described as the result of an imbalance in the equation between energy consumption and energy expenditure. To maintain the same weight, one has to have the same energy expenditure as energy consumption. Over time, energy imbalance causes the accumulation of fat, and 3,500 extra calories translate into one extra pound of body fat. Equation 4.1. below summarizes this model.

*Equation 4.1: Energy / Fat Equation*

\[
\text{Energy Consumption} - \text{Energy Expenditure} = \text{Caloric Overconsumption} \Rightarrow \text{Fat}
\]

As noted by Nestle and Jacobson (2000), however, the precise relationships between diet, activity, and weight gain is still unclear and under investigation, and there seems to be much individual variation in the blackbox of “genes, metabolism, behavior, environment and culture” (U.S. Department of Health and Human Services, Surgeon General Vision, 2010).

4.1.1 Food Intake Trends

One of the hypotheses for the rise of obesity is the rising availability and consumption of food. In terms of availability, economists at the United States Department of Agriculture have noted the rising food supply from 1909 to 1998, and shown that Americans turning away from vegetables while consuming increased levels of fruits and sugar, including corn syrup.

Figure 4.1, below, shows trends in food supply. Since the beginning of the 20th century, trends in total food supply have not been uniformly linear and positive. After
adjusting for waste and spoilage, caloric availability in the United States has increased from 2,220 calories per person per day in 1970 to 2,680 in 1997, a growth of 20.7% in less than 30 years, or 0.77% annually (USDA, 2000). These numbers would predict an average annual weight gain of close to 48 pounds per person, or 1,439 pounds over the time period. Obviously, these numbers are high above the mark.

Fig. 4.1: Calories Available per Person per Day, USDA Food Report (2000: 10).

Food Supply

The U.S. food supply provided 300 calories more a day per person in 1994 than in 1970. Calories from the food supply, adjusted for spoilage and waste, increased from 2,220 per person in 1970 per day to 2,980 in 1997.

Meanwhile, reported caloric consumption has not been shown to have increased dramatically. Analyses of food survey data by Wilkinson Enns, Goldman and Cook (1997) found that reported daily caloric intake in 1994-5 was of 1,633 kcal for women, and 2,470 kcal for women, representing respectively 78 and 91 percent of daily recommendations. The authors are perplexed that there would be so many obese people with such caloric intakes.

percent of boys age 6-11 watched 4 or more hours of television or videos daily (p. 64), noting that weight gains among youth may be attributable to sedentary behavior rather than overeating. Furthermore, they note that salty snacks intake (e.g. crackers, pretzels, chips) has increased, milk consumption decreased, and carbonated soft drinks consumption increased.

A review of the research on the differential metabolic effects of certain types of foods by Kessler (2009) has shown that foods high in sugar, salt and fat lead to overeating, finding which led him to start an offensive against the food industry whose trademarks are sugary, salty and fatty fare. Beyond calories, argues Kessler, the presence of certain substances and additions into our foodstuff matters.

In sum, while we know that food supply has grown since the 70s, the food intake research based on United States survey data shows little evidence of increased caloric intake, which hints to underreporting or unawareness of caloric intake by survey respondents. The American diet, however, turned away from fats and vegetables towards carbohydrates, a shift that some have argued is at the root of weight gain in the United States (Briefel and Johnson 2004; Taubes 2007).

4.1.2 The Toxic Environment

One key idea in both the popular press and in the public health literature on obesity is the idea of a toxic food environment (Davey 2004; Devlin et al. 2000; Horgen and Brownell 2002; Nestle and Jacobson 2000; Saguy and Riley 2005; Wadden et al. 2002). One of its first applications to food was made by Kelly Brownell in a 1994 New York Times op-ed. After denying that a “fat gene” is the culprit in the American “obesity epidemic” and noting our cultural tendency to place responsibility for obesity “on the
sufferer,” Brownell writes that “[e]nvironment is the real cause of obesity” and argues for taxes on unhealthy foods and bans on advertising to children (Brownell 1994).

The idea is now understood to refer to “unprecedented exposure to energy-dense, heavily advertised, inexpensive, and highly accessible foods” (Saguy and Riley 2005, p. 513), and is supported by a wide range of public health advocates. In this critique, the (global) food industry and its agriculture, grocery and restaurant components are the major culprits. Governments worldwide who refuse to legislate and provide subsidies to farmers are seen as accomplices. Their weapons? High fructose corn sirup, added salt, sugars and fats, supersize portions, advertising, and cheap unhealthy options (Campos, 2004; Gaesser, 2002 [1996]; Kessler, 2009; Nestle, 2002; Oliver, 2006; Pollan, 2008; Schlosser, 2001; Taubes, 2007).

Finally, the idea of “food deserts,” or places where it is impossible to buy affordable, healthy food, has been getting attention in the debate over the association between poverty and ill health, including obesity. Interestingly, the idea that healthy food is more expensive in poorer neighborhoods has been shown to be false in the UK (Cummins & Macintyre, 2002a, 2002b; Macintyre, Ellaway, & Cummins, 2002)), while it is supported as true in the United States with studies of Buffalo and Chicago, among others (Gallagher 2006; Gallagher 2010; Lee and Lim 2009).

To address these issues, some have argued for the international regulation of production and other factors leading to overnutrition (Chopra et al. 2002). Others argued for nation-wide interventions (Campos, 2004; Campos, Saguy, Ernsberger, Oliver, & Gaesser, 2006b; Horgen & Brownell, 1998, 2002; Nestle & Jacobson, 2000; Pollan, 2008; Wadden et al., 2002).
4.2 Causal Models for Obesity and its Consequences

Figure 4.1, reproduced from Kim and Popkin (2006), is the most detailed model I have found, although we will see that it is not complete. Kim and Popkin’s model combines, from left to right, (1) initial factors, (2) obesity, (3) intermediary conditions, and (4) chronic diseases. It identifies thirteen different pathways that connect elements of the model to one another (A1 through 3, B1 through 3, C1 through 3, and D1 through 4). Three types of initial factors are included (fetal / infant, dietary, and physical activity) and connected to (2), (3) and (4) above. Similarly, (2) is connected to (3) and (4), and (3) is connected to (4).

Genes and metabolism are noticeably missing from the Kim and Popkin model, and so are early childhood experiences and parental influences, which have been getting attention recently (see Chapter 7). The concept of the “toxic environment,” which has gained traction in recent years and is reviewed below, is also absent from this model. Still, with all its shortcomings, the model shows the immense complexity of the issue of overweight and obesity as it relates to different factors, and participates in the genesis of disease. Equation 4.1 above reduces Kim and Popkin’s causal model to pathways B2 and C1, and forgets pathway A3.

Two further critiques can be waged against Kim and Popkin’s model. First is one voiced by Campos et al. (2006b) in their critique of the Kim and Popkin model, write that while most obesity research investigates the role of obesity on chronic disease, directly (D3) or via intermediary conditions (D1 → Intermediary Conditions → D2), emphasis should be put elsewhere. Schematically, they express their view of obesity as

Equations 4.2: Comparison of Obesity Pathways

\[
\begin{align*}
A2 + B1 + C2 & \Rightarrow D1, \\
A1 + B3 + C3 + D4 & \Rightarrow D3,
\end{align*}
\]
where “$$\gg$$” indicates much greater than. In this framework, research and policy should focus on the direct or non-mediated role of the leftmost causes (1) on (3) intermediary conditions and (4) chronic disease, rather than on the role of obesity on (3) and (4). In light of the research reviewed in Chapter 1 and 3, the relative contributions of any or each of the pathways described by Kim and Popkin’s model remain unclear.

Fig. 4.2: Key Pathways Connecting Individual Factors with Obesity, Intermediary Conditions and Chronic Diseases (Kim and Popkin, 2006: 61).

Again, as argued in Chapter 3, the clearest pieces in the health puzzle are that physical activity and healthy eating are major contributors to health, and that extra adipose tissue influences intermediary conditions (D1), which in turn have been connected to chronic conditions (D2). That chronic diseases would be connected to one another (D4) is no surprise. An important note, however, is that D3 is a highly understudied relationship, yet the shortcut taken by many researchers: fat causes illness.
The second critique waged at the Kim and Popkin model highlights the missing complicating and mediating factors suggested in Chapters 2 and 3: cardiorespiratory fitness, weight cycling, discrimination, prejudicial care and poverty. An alternative model developed by Ernsberger (2009) unpacks relationships D1, D2 and D3 of Kim and Popkin’s model in light of the impoverishing effects of obesity, its social stressors, and prejudicial medical care. In this model, adiposity and social stigma are inseparable in their effects on health and illness, and may be part of the understudied D3 link.

Returning to Kim and Popkin’s model on Figure 4.1 above, it is easy to see how a toxic environment would impact the left-most factors, affecting the fetus through mothers’ blood sugar levels (see Chapter 1), influencing dietary choices (and / or lack thereof), and conditioning exercise / sedentary habits. Yet I want to further argue that the idea of the toxic environment could be expanded to encompass the cultural pressures exerted on obese people. Cultural schemas translate at the micro level into discrimination
and stigmatization in the case of bodily fat, in a way similar to that which has been argued to be at play in the case of gender or race (Ridgeway and Correll 2004). These schemas are also likely to exert pressure on fat people to lose weight in ways that can be extremely unhealthy. I believe that an expanded toxic environment concept would give proper weight to the processes described by Ernsberger’s model in the definition of health for fat people, and thus lead to a more complete contextualization of obesity, above and beyond the pathways posited by Kim and Popkin (2006), as much more than an individual problem: something more akin to race and gender than is typically understood.

The parallel has already been made for perceived discrimination by Puhl, Andreyeva and Brownell (2008), who found that weight or height discrimination was the third most highly perceived focus of discrimination by women, after gender- and age-based discrimination. Among men, it trailed behind race, age, appearance, ethnicity / nationality, and gender. The difference in perceived discrimination between men and women was statistically significant (4.9% vs. 10.3%, p<0.01), suggesting a very gendered discriminatory experience that qualitative research has otherwise documented.

So far I have focused on the biological, psychological and social determinants of obesity, a part of what is called the biopsychosocial model of health, and traced to a 1977 Science article by George L. Engel. He argued for the consideration of social factors in the theorizing, diagnosis and treatment of illness, writing that:

The boundaries between health and disease, between well and sick, are far from clear and never will be clear, for they are diffused by cultural, social, and psychological considerations. The traditional biomedical view, that biological indices are the ultimate criteria defining disease, leads to the present paradox that some people with positive laboratory findings are told that they are in need of treatment when in fact they are feeling quite well, while others feeling sick are assured that they are well, that is, they have no “disease” (Engel 1977, pp. 132-3).
Obese people are universally told they are sick because they meet the ultimate criterion of disease: a BMI over 30. As we have seen, this may not be the most accurate nor more appropriate model to inform future research and policy.

The best model to describe the causes and consequences of obesity, and to frame obesity would build off of Kim and Popkin’s (2006) model on the one hand, and off of Ernsberger (2009) on the other. There are some environmental factors that shape and constrain people’s choices, and consequently their weight and health. Similarly, weight stigma impacts health. Research and policy need to investigate all the pathways identified by Kim and Popkin without forgetting the social context within which obese patients live, and the pressures it exerts on health.

4.3 Strategies to Maximize Health

It is unclear whether obesity in itself is a disease, or whether it is the symptom of underlying disease or environmental triggers. Instead of focusing on weight loss, a more productive strategy would focus on promoting the behaviors that are likely to improve health, and constraining those that are likely to undermine it. If the United States is serious about addressing ill health in its population, educational campaigns that celebrate healthy eating and exercise will not suffice. As we have seen, (1) nutritional knowledge doesn’t always translate into healthier eating habits (Germov and Williams 1996; Lakdawalla et al. 2005); (2) food choice may be dependent on food availability, food prices, caloric density, and income (Drewnovski and Specter 2004); (3) regulatory means can have an impact on the food industry (e.g. trans fat across the United States, salt contents in New York City).

Table 4.1 translates the key findings from Part I into health strategies.
Table 4.1. Part I findings and associated health-enhancing strategies

<table>
<thead>
<tr>
<th>Finding</th>
<th>Strategy</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Food Supply</strong></td>
<td></td>
</tr>
<tr>
<td>Nutritional knowledge does not always translate into healthier eating habits. Changes are easily made for a short period of time, but over the long run, people tend to revert back to their old eating habits.</td>
<td>Healthy eating will have to be easier, with an increase in healthy options and associated decrease in unhealthy ones. Research should be done to investigate means to change eating and exercise habits in the long run. Some have advocated for the use of information technologies and group activities.</td>
</tr>
<tr>
<td>Food prices are a barrier to healthy eating for low-SES people.</td>
<td>The Federal government should revisit its subsidies scheme. More money should be invested into vegetables, legumes, and nuts.</td>
</tr>
<tr>
<td>Eating habits often originate in the home.</td>
<td>Parents will need to be taught healthy eating habits, and the barrier to healthy food provision will have to be lowered.</td>
</tr>
<tr>
<td>Americans tend to underestimate their caloric consumption. Processed food is often high in added sugar, salt, and fat, substances that increase hunger and caloric consumption, beyond their actual caloric content.</td>
<td>Food labeling has been in place for over a decade, and may have helped people better estimate their caloric intake, although portion size reported on labels often do not constitute the size of the portion actually eaten. This discrepancy should be fixed. Restaurant portions should be shrunken to count no more than one third of the daily recommended intake values), and caloric information disclosed. Guidelines should be imposed on the food industry.</td>
</tr>
<tr>
<td>Some neighborhoods are food deserts. They do not have enough sources of healthy food within an accessible radius. Unsurprisingly, in the United States, those deserts are found in poorer communities.</td>
<td>Local governments should provide retailers with incentives to locate stores in these neighborhoods.</td>
</tr>
<tr>
<td>The abundance of choice that is a defining characteristic of today’s American foodscape works against healthy choices, and participates in the creation of a toxic, obesogenic environment.</td>
<td>A twinkie tax – a tax on junk food – may not be sufficient to make healthy food choices easier. Restriction on the sale of junk food, either by reducing the number of available items, mandating reduction of floor space dedicated to snacks and junk food, and the sponsoring of healthy foods may be required. The sale of sugary beverages (including fruit juice) and of salty, fatty, and sugary snacks should be prohibited in schools. School cafeterias should be funded at the level where managers can plan healthy menus.</td>
</tr>
<tr>
<td>Exercise</td>
<td>Care for Large People</td>
</tr>
<tr>
<td>-------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Children spend long periods of time in front of the television or a computer. Sedentary behavior is unhealthy in itself, and facilitates weight gain.</td>
<td>Proper funding for physical education will be necessary, and classes be attended two to three times a week. The construction of parks, and staffing to insure their safety will be important, especially in urban settings. Afterschool programs should focus on movement-based activities. Varsity sports will have to be complemented with non-varsity team sports and other non-competition-based, movement-oriented activities.</td>
</tr>
<tr>
<td>Obese people often find it difficult to find appropriate workout clothes. Furthermore, some exercise machines are not designed to support really heavy individuals, which constitutes a barrier for physical exercise among the heaviest.</td>
<td>Businesses should be encouraged to take on this market: exercise machines, clothes, and adapted workouts. A market evaluation could be sponsored by the federal government.</td>
</tr>
<tr>
<td>Care for Large People</td>
<td></td>
</tr>
<tr>
<td>Obese people tend to stay obese. It is extremely difficult to make an obese person lose weight permanently, and there is strong evidence that weight loss is typically followed by regain in excess of lost weight. Obese people who maintain their weight, eat well, exercise, and have a support network are as healthy as they can be.</td>
<td>Instead of focusing on weight loss, public health strategies should focus on health-enhancing activities. The Health at Every Size is a great framework to start from. Fat people should not be advised to lose weight, but rather to maintain their weight and improve their self-care habits: exercise, eat well, develop stress-management tools, etc.</td>
</tr>
<tr>
<td>Weigh ins are the source of appointment cancellations, and thus lead to delayed care.</td>
<td>BMI could be used in medical research, but not collected for treatment purposes. While eyeballing weight is a difficult task, it is easy to evaluate de visu whether someone is obese or not. In most health care situations, it should be sufficient. In cases when it is not, such as for surgery and for health insurance claims for bariatric surgery, patients would need to be weighed, and the reason for the weigh-in clearly explained.</td>
</tr>
<tr>
<td>Health professionals hold prejudicial views of obese people, and provide discriminatory care. This, in turn, accentuates health disparities between the obese and the non-obese.</td>
<td>Health professionals should get instruction on the role of stigma in diagnosis, treatment, and health outcomes. They should be provided with sensitivity training. Health professionals’ view of obesity should be broadened beyond Equation 4.1. above to include the social determinants of health and</td>
</tr>
</tbody>
</table>
### Maternity, Families and Health

<table>
<thead>
<tr>
<th>Maternal obesity and gestational diabetes have long-term health consequences on infants, through to adulthood.</th>
<th>Health education programs in high schools, when covering reproductive health, should emphasize the importance of healthy eating habits and exercise, and note the importance of consulting health professionals when conceiving, without mentioning weight. Health professionals should advise women at the early stages of conception to monitor their weight and increase their caloric intake only by what the fetus is expected to need.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Children are the subgroup where weight gain has been the greatest over the past decades. Minority and low-income children are particularly affected. Youth obesity is a predictor of adult obesity, and is associated with discrimination and lower educational achievement, especially among girls.</td>
<td>Prevention will have to start early, and families instructed with clear guidelines about food needs for growing children. Children of low-income families should be targeted, healthy foods subsidized, and healthy eating guidelines should inform federal food provision programs.</td>
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</table>

### Medical Research

<table>
<thead>
<tr>
<th>Medical research has not fully investigated the mitigating effects of cardiorespiratory fitness in defining health outcomes for the obese.</th>
<th>Funding should continue to be available to study the impact of exercise-based life interventions that do not include weight-loss goals. Federally-funded weight research should be required to include cardiorespiratory fitness as a control variable when using live subjects. Money should be invested into the development of measures of cardiorespiratory fitness that can be used in population-based studies and actually correlate with fitness tests.</th>
</tr>
</thead>
<tbody>
<tr>
<td>The processes of food habit and preferences development does not figure in the literature.</td>
<td>Research should try to understand the critical periods for the development of certain tastes in food, and investigate them over the life course.</td>
</tr>
<tr>
<td>Weight-related stigma has been growing over the past decade. We known little about the mechanisms whereby stigma impacts psychosocial behaviors and health.</td>
<td>Research should look into the development of stigma, its sources. Inspiration for research could come from social psychological research on gender beliefs and stereotypes.</td>
</tr>
</tbody>
</table>

While the health costs of obesity net of other factors aren’t fully clear, the benefits of a healthy diet are clear, for lean and fat people alike. Most individuals have heard the messages warning against fat, sugar, and salt, yet they either lack the resources to avoid
such fat, sugary, and salty foods or, as a friend said once, choose to enjoy life (i.e. fried food) rather than eat their vegetables (an argument also made by Lakdawalla, Philipson, & Bhattacharya, 2005). We thus need to both help people eat healthy food, and help them not eat unhealthy food. This will likely require regulation and subsidies away from meat, dairy, and grains and towards fruits, vegetables, nuts and legumes. Income constrains food choices, and that poor neighborhood often at once lack food outlets where quality foods are sold, and abound with fast-food options: restaurant chains and corner stores where soda, beer, chips, ramen, chocolate bars and ice cream will constitute dinner.

The benefits of exercise are also clear, and they are also clear across the weight spectrum. Because children and adults are less likely to be active when their neighborhoods are unsafe, the needs of populations in poorer urban areas will also have to be addressed. The best strategy among adults, then, seems to be approaches that combine physical activity with a better diet and pushes for weight maintenance rather than weight loss (Bacon 2006; Eckel 2005; Ross and Bradshaw 2009). Among youth, it will be prevention through parental education, healthy education, and the encouragement of physical activity (see Chapter 7 for more).

In sum, deep structural reforms and a coherent strategy to address the American “too much food for thought” problem (Davey 2004). Piecemeal interventions and individual-level solutions will only be skin deep.
Part II: The Changing Meanings of Fat
Chapter 5: The Medicalization and Healthicization of Fat.

Why do Americans, the press, Surgeon General and White House alike care about weight? Why has the Center for Disease Control carefully monitored the weight of Americans since the 60s? In this chapter, I argue that one of the reasons why our culture pays so much attention to obesity is not merely a combination of esthetic concerns and moral outrage, but rather also the result of the rise of medicine and of individualism.

To many of the scholars who have turned to history to understand the current “war on obesity,” fat phobia stems from an exaggerated obsession with thinness and its glorification. The argument roughly goes: As thinness has become more attractive and the province of the wealthy, fat has come to be demonized and found repulsive. The glamour associated with leanness has masked its health costs, while the health hazards of obesity have been amplified.

The burden, in this interpretation, is justifiably said to be disproportionately women’s, whose bodies are unruly and the object of greater scrutiny and more exacting standards. Society at large and academicians have paid attention to anorexia nervosa – weight obsession at its extreme –, especially since the 1970s, when psychiatrists and statisticians “discovered” anorexia and noticed increasing rates among young women (Brumberg, 2000). Still, some argue that attention to the risks associated with thinness and dieting have been shoved under the carpet as researchers turned their gaze to the more lucrative and more morally troublesome issue of obesity (Campos et al., 2006; Germov and Williams, 2006).

Oliver (2006b) argues that while there are many circulating hypotheses that explain weight gain, “none of them really explains the rise of obesity. In other words, none
of the theories about why Americans started gaining weight also explain why this weight gain has been interpreted as a medical condition” (original emphasis, p. 38). We have seen in Chapter 3 the blind spots of medical research, and its understudied assumptions. In this chapter, I trace the history of the transformation of fat into a medical problem, and argue that the historical development of medical interest in fat set medical researchers on a path that confirmed time and again the role of fat in ill health. Initially, this interest may or may not have been motivated by fat phobia. The claim I am making here is not a story of origins, but rather one of the institutionalization of certain ways of thinking, methods, and practices. I argue that their sedimentation led to the state of medical research discussed in Part I, and that inertia is part of the answer. To support this claim, I trace a history of the medicalization of fat. I combine secondary sources with primary sources to build a general overview of the path taken by fat in the medical imagination. But first, I review the broad ideas that shape my analysis.

5.1 Medicalization and Healthicization

Conrad (1992) defines medicalization as “a process by which nonmedical problems become defined and treated as medical problems, usually in terms of illnesses or disorders” (p. 209). Zola (1983) defined it as a “process whereby more and more of everyday life has come under medical dominion, influence and supervision” (cited in Conrad, 1992, p. 210).

Beyond medicalization, sociologists have recently theorized a new form of medicalized intervention: “healthism” (Crawford 1980) or “healthicization” (Conrad 1992). Crawford (1980) writes:

Briefly, healthism is defined here as the preoccupation with personal health as a primary – often the primary – focus for the definition and achievement of well-being; a goal which is to be attained primarily
through the modification of life styles, with or without therapeutic help. The etiology of disease may be seen as complex, but healthism treats individual behavior, attitudes, and emotions as the relevant symptoms needing attention. Healthists will acknowledge, in other words, that health problems may originate outside the individual, e.g. in the American diet, but since these problems are also behavioral, solutions are seen to lie within the realm of individual choice. Hence, they require above all else the assumption of individual responsibility (Crawford 1980, p. 368).

According to Crawford (1980), medicalization morphed into healthicization partly because of the disillusioned optimistic collectivism of the late 1960s. Others have pointed to a growing social skepticism towards the promises of post-war positivistic medicine, which had hoped to reduce all disease to causal biological agents and solve every medical problem with medication and surgical interventions (Le Fanu 1999).

Healthism is not incompatible nor coextensive with medicalization. Conrad (1992) summarizes the distinction: “Medicalization proposes biomedical causes and interventions; healthicization proposes lifestyle and behavioral causes and interventions. One turns the moral into the medical, the other turns health into the moral” (p. 223). While Crawford’s (1980) case studies investigated health-consciousness as manifested outside the formal medical establishment, I would like to point out that the medical profession has also become healthist: focusing on individual solutions (i.e. patient education and empowerment) to problems of all natures, individual or societal. Indeed, as noted by Greenhalgh & Wessely (2004) “[p]atient empowerment is a popular theme in both government policy and the health services research literature” (p. 207). Investigating the medical discourse at the global level in WHO documents, Inoue (2003) has found something similar: the rise of health promotion to put the onus of health and well-being in the hands of the patient rather than the physician or the system.

Health promotion is cost effective at both the individual and national levels. Yet it tends to have moral implications by placing responsibility and blame in the hands of
individuals. Crawford sees this “intersection of morality and blame with illness and health” as “one of the most complex subjects facing medical sociologists and social historians” (Crawford 1980, p. 378). Some authors are more acerbic in their critiques.

Germov and Williams (1996) write:

> The individualistic ethos of the lifestyle approach resonates with the ideology of conservative economic and political theories such as economic liberalism and public choice theory which emphasize the concept of the sovereign (health) consumer. This idealized health consumer has allegedly free choice over whether they are healthy or not, simply by modifying their individual lifestyle. The cornerstone of this health ideology, aside from the obvious undertones of “blame the victim”, are the key precepts of conservatism: self-surveillance and self-regulation (Germov and Williams, 1996 p. 103)

Similarly, Check (2008) sees healthism as a “new conservatism” that permeates our daily lives, while Fitzgerald (1994) acknowledges the positive sides of health but nevertheless calls the new focus on health a “tyranny.” He traces our social obsession with health to the WHO’s 1946 redefinition of health as “a state of complete physical, mental, and social well being, and not merely the absence of disease or infirmity,” and to the extension of the realm of medical intervention “from physical problems to character flaws, poverty, crime, unhappiness, and even unattractiveness” (p. 196). Furthermore,

> Certain failures of self-care have become, in a sense, crimes against society, because society has to pay for their consequences. And society now looks to health care providers for the education and direction to eliminate behavior that leads to disease (Fitzgerald 1994, p. 198).

He suggests that this normative trend has become “clearest” with issues of “self-abuse”: obesity, alcoholism, smoking, heart disease, intravenous drug abuse, and human immunodeficiency virus (HIV) infection (p. 198). Becker (1993) has noted a similar phenomenon, and pointed to Thomas’ (1983) comment that our obsession with health has turned into a fear of many aspects of our daily lives (food, the environment, sleep and stress, for example), such that there is now an “epidemic of apprehension.”
Others before me have discussed the medicalization of fat. Conrad and Schneider (1992) have described the process whereby a morally problematic issue gets turned into an illness via medicalization. In their book on deviance and medicalization, they note that while the medicalization of obesity, opiate addiction and hyperactivity may be framed as progress, the progress may not be equally beneficial to all (Conrad and Schneider, 1992, p. 33-4). Sobal (1995) also notes the medicalization of fat. He notes how fat evolved from something with value to badness to illness and to a politically acceptable condition, and thus from a moral model to a medical model and then to a political model.

Chang and Christakis (2002) investigated the changing norms around body fat in the *Cecil Textbook of Medicine* from 1927 to 2000, and found that obesity shifted from something that people do (a moral failure) to something that people are done to or experience (an environmental press).

Both Sobal (1995) and Chang and Christakis (2002) suggest that the move from moral to medical have lead to the de-stigmatization of fat people. Saguy and Almeling (2005) challenge this view, and note how the medicalization of fat bodies stigmatizes these bodies as ill. Indeed, they note, “notions of morality play a central role in the controversy over obesity, as in many medical disputes, and illustrate how medical arguments about body weight can be used to stymie rights claims and justify morality-based fears” (Saguy and Almeling, 2005, p. 870). Later empirical analyses by Saguy and Riley (2008) highlight how the translation of medical science into popular press accounts further blames individuals for their weight. Importantly, Saguy and Riley’s research contextualizes medical research and decouples it from popular press and cultural understandings of the causes and consequences of obesity.

In what follows, I discuss the historical medicalization and healthicization of fat.
5.2 The Medicalization and Healthicization of Fat

In Never Satisfied, historian Hillel Schwartz (1986) argues that “[w]eight is a cultural condition. A scale does not make it more or less real. Fatness too is a cultural condition. Calipers are a pretext and pretense” (p. 4). He shows, through careful document analysis, how early our culture started caring about the body being too large or weighing too much, and the different conceptualizations of weight upon which (medical) models were built, and bodies analyzed.

If we believe the Online Etymology Dictionary, “adipose” was first used in the English language in 1743 to refer to things that are made of or related to fat. Because the use of adipose was traced to a borrowing from the modern Latin “adiposus,” and because modern Latin was almost solely used as a scientific language after the collapse of ancient Rome in the 4th century, it seems that in the early-to-mid 18th century began the medicalization of fat.

As early as 1779, writes Schwartz, weight loss was recommended as a strategy to reduce the risk of angina pectoris, and thus connected to health and mortality. Like many others, he argues that even before World War I, conceptualizations of the human body such as a machine led to methods of bodily management that would parallel industrial management ones, as was the case with Taylorism, for example (Taylor 2006 [1916]). The streamlined bodies made no room for fat. Since, bodily tolerances have only gotten lower.

In the 1920s, according to Brumberg (2000 [1988]), “the basic institutions of American beauty culture,” by which she means the “fashion and cosmetic industries; beauty contests; the modeling profession; and the movies,” were being established and promoted a culture where dieting slowly became the norm if not an expected state of womanhood (p. 229). Simultaneously, the medical profession took disordered eating
under their care, such that fasting women who were previously seen as saints were slowly becoming mentally and physically ill patients (Brumberg 2000 [1988]; see also Gemzöe 2005).

In 1924, in an article published in the *Journal of the American Medical Association*, McLester, a medical doctor in Alabama, discusses a treatment course for obese patients. “The obese,” he writes, “either eats too much or exercises too little.” The medical doctor’s role is to help the patient balance “metabolic activity and food intake” by suggesting the proper macronutrient composition of the patient’s diet, prescribing cold baths and encouraging exercise (McLester 1924, p. 2103). Concern is not with obesity as a killer or as an epidemic, but rather about middle-age weight gain, esthetics and longevity: “To be thin is fashionable. Likewise, if not carried too far, it is conducive to comfort and longevity” (McLester 1924, p. 2103).

In 1921, actuaries established guidelines for adult weight based on mortality risk (Schwartz 1986). In the early 1940s, Metropolitan Life started circulating tables that presented ideal weights along with what constituted overweight, warning that “[o]verweight … is so common that it constitutes a national health problem of the first order” (Schwartz 1986, citing Metropolitan Life advertisement from 1941, p. 159). By 1947, genetics had made inroads into mainstream medical textbooks, and obesity was partly blamed on hereditary factors (Chang and Christakis 2002, p. 156).

The first medical professional organizations interested in obesity were founded in that period: 1949 for the American Society of Bariatric Physicians, 1967 for the Association for the Study of Obesity (United Kingdom). As noted by Sobal (1995), the medicalization of obesity further picked up steam in the 1950s when popular press books started developing the discourse around obesity as a social problem. In 1952, NIH
director Dr. W. H. Sebrell, Jr. identified obesity as the number one nutritional problem in the United States (quote found in Science News Letter article by Schwartz 1986). At the same time, surgical procedures for the treatment of obesity were developed. Jaw wiring came first, to be followed by intestinal bypass surgery developed by Kremen and colleagues in 1954, and jejunoileal bypass surgery by Payne and DeWind in 1969 (Sobal 1995, p. 73).

In the late 1960s, obesity was characterized as a disease of affluent societies (Chang and Christakis 2002, p. 157).

In the 1970s, weight had become a national obsession, with the rise of Twiggy as a model of femininity, rising rates of anorexia nervosa (Brumberg 2000 [1988]; Seid 1988), and the multiplication of publications on obesity. A 1974 The Lancet editorial identified infant and adult obesity as “the most important nutritional disease in the affluent countries of the world” (Nestle and Jacobson 2000, p. 12). In 1978, the NIH issued its first consensus statement on surgery for obesity, titled “Surgical Treatment of Morbid Obesity.” In 1982, the Obesity Society (United States) was founded, and in 1985, the NIH chose obesity as a topic for a consensus conference that resulted in the publication of the “Health Implications of Obesity” consensus statement, which recommended the use of BMI by medical practitioners. In this document, overweight BMI was then defined as above the 85th percentile of population data, which translated then into a BMI of 27.8 for men, and 27.3 for women, higher values than the ones used today. Simultaneously, the 1985 edition of the Cecil Textbook of Medicine first discussed obesity as a disease (Chang and Christakis 2002, 158).

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10 As we will see in Chapter 6 below, the number of articles published on obesity have been rising sharply since the middle of the 20th century, while specialized medical journals on obesity have been proliferating as well, since the early 1970s.
In 1986, health specialists from around the world convened in Ottawa, Canada, for the First International Conference on Health Promotion. The Ottawa Charter for Health Promotion, a short, four-page document that is best described by its optimism, listed the “fundamental conditions and resources for health” which are no less than peace, shelter, education, food, income, a stable eco-system, sustainable resources, social justice, and equity,” and hoped to achieve health for all by 2000.

By 1990, obesity had found its way in the International Classification of Diseases (ICD-9-CM 1990), and was thus fully considered a condition worthy of medical attention worldwide (Sobal 1995). The second consensus statement on surgery for obesity came in 1991, and titled “Gastrointestinal Surgery for Severe Obesity” (NIH Consensus Development Program, 2010). In 1993, a panel of WHO experts convened to adopt uniform categories for the anthropomorphic evaluation of weight, and chose the BMI guidelines we know today: underweight is defined as a BMI below 18.5 kg/m²; healthy weight from 18.5 ≤ BMI < 25; overweight from 25 ≤ BMI < 30; obese from 30 ≤ BMI < 40; and morbidly obese BMI ≤ 40 (WHO 1995 Technical Report Series 854, p. 312). These BMI categories were not used until 1998 in the United States after the convening of an NIH National Heart, Lung and Blood Institute expert panel, which published the “Clinical Guidelines on the Identification, Evaluation and Treatment of Overweight and Obesity in Adults: The Evidence Report” (NIH / NHLBI 1998). This same year, the WHO was calling for mobilization against obesity with its Technical Report titled “Obesity: Preventing and Managing the Global Epidemic” (WHO 1998, Technical Report Series 894).

Interest in obesity as a public health problem seems to have hit a transition point in the early 2000, and at the same time obesity grew in American people’s consciousness.
Oliver (2006a, 2006b) argues that the transformation by William Dietz and Ali Mokdad of obesity data tables into color-coded maps in the late 1990s was a tipping point in the making the idea of obesity as an epidemic “stick.”

In 2001, the Surgeon General Tommy G. Thompson made his first *Call to Action to Prevent and Decrease Overweight and Obesity*, and used color-coded maps to show rising rates of obesity by state, comparing 1991 data with 2000 data. The *Call*, which starts with the claim that “Overweight and obesity have reached nationwide epidemic proportions,” (p. v), lists the following five fundamental intervention principles (U.S. Department of Health and Human Services, 2001, p. v):

- Promote the recognition of overweight and obesity as major public health problems.
- Assist Americans in balancing healthful eating with regular physical activity to achieve and maintain a healthy or healthier body weight.
- Identify effective and culturally appropriate interventions to prevent and treat overweight and obesity.
- Encourage environmental changes that help prevent overweight and obesity.
- Develop and enhance public-private partnerships to help implement this vision.

By 2002, as Schlesinger (2005) notes, Americans began seeing overweight and obesity as a serious national health problem. Starting in 2003, they came to see it as America’s “second most salient concern, trailing only cancer as a perceived health problem” (Schlesinger 2005, p. 786).

In November 2007, the Health and Human Services launched its *Childhood Overweight and Obesity Prevention Initiative*; in 2008 it published its *Physical Activity Guidelines for Americans*. Then, on January 28, 2009, Surgeon General Regina Benjamin claimed that she hoped to transform the current conversation on obesity and illness to focus on “being healthy and being fit” at any size, recommending that we all “eat nutritious foods, exercise regularly, and have fun doing it” (U.S. DHHS, 2009).
This video was shortly followed in April 2009 by the launch by First Lady Michelle Obama of the White House’s Let’s Move! program, and in January 2010 by the Surgeon General’s printed and online Vision for a Healthy and Fit Nation, document that reiterates her wish to refocus discussions of obesity from illness to health and fitness. “History,” she writes, “has shown that grassroots movements can make positive changes in their communities” (p. 11). Beyond grassroots mobilization, however, what Americans need to stop the obesity epidemic is to realize that health-enhancing behaviors will allow them to “embrace each day and live their lives to the fullest—without disease, disability, or lost productivity” (U.S. DHHS, 2010, p. 12).

The language used by the Surgeon General is decidedly healthist, and individualistic: it is about health improvement for its own sake, achieved mostly through health-informed decision making and community (grassroot!) mobilization, in order to boost demand and influence marketing trends (p. 1), as well as physician support (p. 10).

Further Feminist Considerations

Fat is a feminist issue (Orbach 1988 [1978]). Feminists have studied the body as a privileged location for the investigation of power relations between genders, and noted how “[v]isual culture has disturbed girls’ and women’s relationships to their own bodies” so that “what they see in the mirror fails to please them. They see themselves and they experience themselves as too fat. They are fat in their minds” (Orbach 2006, p. 68).

In discussing the connection between science, medicine and women’s bodies, feminists have investigated anorexia nervosa (Brumberg 2000 [1988]), breast pumps and breast feeding (Boyer and Boswell-Penc 2010), breast cancer (Jain 2010; Sandell 2008), body modification (Pitts 1999), and cosmetic surgery (Pitts-Taylor 2007), and argued for a very societal construction of the body. In doing so, they moved the issue beyond the
individual choice paradigm and pushed for an understanding of the body as embedded in a gendered culture. Much of this research shows how medical practice legitimizes certain bodily practices while delegitimizing others.

Brumberg (2000 [1988]), for example, has shown how the rise of the medical profession and the decline of religion in the 18th and 19th century transformed women’s fasting from religious symbol to pathology. Pitts-Taylor (2007) similarly argues that the rise of cosmetic surgery and cosmetic surgeons coupled with the discourse on individual choice and empowerment via the body to boost surgical procedures. More dramatically, Sandell (2008) argues based on breast cancer patients’ stories on breast reconstruction surgery the extent to which womanhood is tied to a specific embodiment among reconstructive surgeons.

Fat studies research has investigated the gendered consequences of fat hatred in American society and beyond (Boero 2009; Isono, Watkins and Lian 2009; Royce 2009). Yet very little research has investigated the gendered consequences of the medicalization of fat. There are a few notable exceptions.

The work of Boero (2009), Solovay (2000), and Campos (2004) have shown the pressures felt by mothers of fat children, sometimes as a result of a medical establishment that pathologizes fat and of a legal system that criminalizes fat. Boero (2009) puts fatness in a historical context where social problems lead to mother blame. She notes:

As they are usually charged with the preparation, regulation, and purchase of food for their children, mothers – working mothers in particular – are held responsible for their children’s ‘poor’ eating patterns and their assumed-related ‘obesity’ … The weight of one’s children has increasingly become a litmus test of good mothering (Boero, 2009, p. 113).

We still know very little about these connections, but do know that at least in the United States, the celebration of “chubbiness” in children is over (Schwartz, 1986).
Chang and Christakis’ (2002) study of medical textbooks showed the psychiatric pathologization of fat people, noting for example how fatness is perceived to be a means to escape certain forms of interactions, or the result of unsatisfied desires (p. 162). There is a real potential for a rising stigmatization of fat kids, mothers of fat kids, and fat mothers as a result of the rising discourse on the pathological consequences of bodily fat. Future research could turn to the different ways mothers internalize or resist the popular and medical discourse on body fat and the health of their families. It could also investigate the responses of non-fat individuals towards fat mothers, the mothers of fat children, and fat kids. Finally, it could turn to media representations of these populations.

Another hypothesis would be that controlling for socioeconomic status, more socially-conservative individuals – those who believe in the unavoidability of gender differences and the importance of motherhood – are also more likely to blame mothers for their children’s weight, and to blame fat mothers for endangering their kids’ health. Controlling for socioeconomic status here will be critical, given its negative correlation with obesity.

All of these research endeavors would benefit from longitudinal and cross-national investigation. In line with the framework put forward in this paper, one starting hypothesis would be that as scientific authority and an individual culture are established, attitudes toward fat kids, fat mothers and mothers of fat children become increasingly negative. As cultures go through the nutrition transition are they all becoming more health focused? Boero (2009) suggests that the individualistic discourse on adult obesity is not so with children. Are mothers increasingly or decreasingly likely to be blamed for their children’s fat?
Conclusion: Health, for Better and for Worse

In this chapter, I have tried to show how the current concern with obesity is based on a historical record that is larger than mere esthetic and moral considerations. The institutionalization of fat as a medical problem happened as medical doctors, medical researchers, and national and international organizations went through the motions of legitimate science, gradually turning obesity into a medical problem, and soon into a social crisis: an epidemic. This process is parallel to that discussed by several scholars of science before me (Fleck 1979 [1935]; Hacking 1990; Latour and Woolgar 1986 [1979]; Longino 2006).

While the medicalization of fat has been celebrated by some, including Yanovski (1998), who wrote in an *Archives of Family Medicine* editorial that the “change in [the medical profession’s] view of obesity from a shameful social problem to a medical disorder worthy of attention is to be commended” (p. 385), the major shortcomings of the medical profession in providing care to “large patients” were already well known in 1998, and are much more so today, as seen in Chapter 3.

To date, there is very little empirical work that discusses the healthicization of fat explicitly. The evolution of the medical discourse on obesity presented here, and summarized in Appendix III below, contains the traces of both medicalization and healthicization. On the one hand, doctors are seen as an important piece in the how-to-solve-obesity puzzle. Surgery and medication are still studied and promoted. On the other, doctors and clinicians are mobilized to help their patients make the healthy choices, and transition to a fun, healthy lifestyle.

On the whole, it is fair to say that the solutions brought forward by the United States’ government and health institutions typically focus on empowerment, inner
growth, therapy, etc. As noted by Nestle and Jacobson (2000), most of the policy
guidelines published by U.S. governmental agencies and health organizations since the
1950s have been focused on individuals and addressed diet, exercising or both. More
recently, the combined fat = bad and lean = good equations have been challenged, by
activists but also in the most recent Surgeon General call for action and in some
governmental initiatives. Yet the emphasis on individual growth and individual health is
stronger than ever, with an added zest of fun.

In Chapter 6, I look at the medical literature on obesity at the aggregate level to
investigate the internal dynamics of the field, and evaluate them in conjunction with the
events noted in this chapter.
Chapter 6: Expanding Waistlines, Expansive Discourse: Obesity in the Medical Literature

In this chapter, I propose a macro-level overview of the evolution of the medical discourse on obesity and the so-called obesity epidemic, starting in 1950. Using PubMed data on frequency of publication for obesity and different co-morbidities, I show the immense growth obesity research has experienced over the last 60 years. This growth happened at a time when waistlines were also expanding, but as we will see, the growth was time lagged, substantiating the opinion voiced by Stevens et al. (2006) that the scientific community did not pay attention to obesity for decades.

In what follows, I first look at the timing of the expansion of the medical literature on obesity, and document the magnitude of this expansion. I then zoom in and investigate obesity and its associations with other chronic conditions, as well as within the public health discourse. Finally, I turn to the obesity epidemic discourse in the hopes to better understand its origins, scope, and expansion.

6.1 The Growth and Pervasiveness of Obesity Research over Time

We have seen in Chapter 5 that obesity has come under medical scrutiny late in the 18th century, and that by the 1940s, the connection between obesity and mortality had been institutionalized into weight tables, and used for insurance purposes. In this section, I look at two indicators of the medicalization of obesity: professional journals and expansion of disciplinary research on the topic.

6.1.1 Obesity Journals

A first way to investigate the medicalization process is to look at the founding years of different medical journals specifically devoted to obesity. The table below
provides a breakdown of journal titles by year. Notably, the first obesity journal’s title did not include the term obesity. Founded in 1972, *Bariatric Medicine* was renamed *Obesity & Bariatric Medicine* before being stopped in 1984. When these dates are plotted over time, one sees the clustering of publication foundation dates first in the early 1990s, and the regularity at which they are founded from 1999 to 2009, at an average close to one per year. Note, also, the number of journals whose titles include surgery (2), health, fitness or wellness (2), management, regulation, or therapy (3), or are associated with other medical conditions such as diabetes, or metabolism and metabolic syndrome (4). The *Health at Every Size Journal* is considered by fat scholars to be the safe haven of fat acceptance researchers (Saguy and Riley 2005). Its name nevertheless falls within the healthist paradigm.

6.1.2 Obesity as a Topic of Scientific Investigation

Another way to investigate the growth of the obesity discourse over time is to look at the frequency of use of the word “obesity” in titles and abstracts in the medical literature. Figure 6.1 presents such general trends as a ratio of total publications recorded in PubMed (multiplied by one million). We see first that interest in obesity (full black line) was relatively low until the 1970s: from 1970-74, there were 1,204 obesity articles published and recorded in PubMed. Twenty years later, there were five times as many articles published over the 1990-94 period (5,988), and by the 2005-9 time period, there were close to 40,000. This is a spectacular growth from 479 at the beginning of the time period: a multiplication by a factor of 83.

The number of articles published in PubMed is recorded in Figure 6.1 with a dark grey line, and plotted on the right-side vertical axis. The knowledge base in medicine has clearly been growing, but obesity research has been growing faster than the overall
PubMed database, as we see from the ratio of obesity articles to the total number of articles recorded in PubMed. Obesity research has increased 10 fold from 1950-4 to 2005-9, from less than 0.1% of all publications in PubMed to slightly over 1%, signifying a ten times greater growth rate for obesity research compared to medical research overall. As we see on Figure 6.1, the ratio of obesity articles to PubMed also spiked over the past ten years.

Table 6.1: Year of Foundation and Names of Different Obesity-Related Journals

<table>
<thead>
<tr>
<th>Journal Title</th>
<th>Year</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obesity &amp; Bariatric Medicine (previously called &quot;Bariatric Medicine&quot;)</td>
<td>1972</td>
</tr>
<tr>
<td>International Journal of Obesity</td>
<td>1976</td>
</tr>
<tr>
<td>Journal of Obesity and Weight Regulation</td>
<td>1981</td>
</tr>
<tr>
<td>(previously called &quot;Obesity and Metabolism&quot;)</td>
<td></td>
</tr>
<tr>
<td>Health at Every Size Journal</td>
<td>1988</td>
</tr>
<tr>
<td>(previously called &quot;Healthy Weight Journal,&quot; and &quot;Obesity and Health&quot;)</td>
<td></td>
</tr>
<tr>
<td>Obesity Surgery</td>
<td>1991</td>
</tr>
<tr>
<td>Obesity</td>
<td>1992</td>
</tr>
<tr>
<td>Obesity Research</td>
<td>1992</td>
</tr>
<tr>
<td>Current Opinion in Endocrinology, Diabetes, and Obesity</td>
<td>1993</td>
</tr>
<tr>
<td>Diabetes, Obesity &amp; Metabolism</td>
<td>1999</td>
</tr>
<tr>
<td>Obesity, Fitness &amp; Wellness Week</td>
<td>2000</td>
</tr>
<tr>
<td>Obesity Reviews</td>
<td>2000</td>
</tr>
<tr>
<td>Obesity &amp; Diabetes Week</td>
<td>2003</td>
</tr>
<tr>
<td>Obesity Management</td>
<td>2005</td>
</tr>
<tr>
<td>Surgery for Obesity and Related Diseases</td>
<td>2005</td>
</tr>
<tr>
<td>Diabetes, Metabolic Syndrome and Obesity: Targets and Therapy</td>
<td>2008</td>
</tr>
<tr>
<td>Obesity Facts</td>
<td>2008</td>
</tr>
<tr>
<td>The Open Obesity Journal</td>
<td>2009</td>
</tr>
</tbody>
</table>

Table 6.2 shows frequencies of articles on “obesity,” “obesity AND cancer,” “obesity AND diabetes,” “obesity AND cardiovascular disease,” or “obesity AND childhood” at three time points: 1989, 1999, and 2009. It also shows the 1989-99 and 1999-2009 growth rate calculated from these time points, as well as annualized 1989-2009 growth rate. Table 6.3 shows the same information, but presents the publications as a ratio of all PubMed data. This data that can also be seen on Figure 6.2.
<table>
<thead>
<tr>
<th>Year of Growth Rate Period</th>
<th>Obesiry AND Childhood Total Published</th>
<th>Childhood</th>
<th>CVD</th>
<th>Diabetes</th>
<th>Cancer</th>
<th>Obesity</th>
</tr>
</thead>
<tbody>
<tr>
<td>1989-2009</td>
<td>1.1728</td>
<td>0.0757</td>
<td>0.1716</td>
<td>0.1021</td>
<td>0.0622</td>
<td>0.2339</td>
</tr>
</tbody>
</table>

Table 6.2: Published Database Publications on Obesity and Related Keywords (Title and Abstract).
Fig. 6.1: Obesity In, and As Proportion of, PubMed Database 1950-2009

- Obesity in Titles and Abstracts
- Ratio Obesity to PubMed (x 1M)
- Total PubMed (in M)
Fig. 6.2: Obscured and associated keywords as proportion of PubMed Database, 1990-2009.
Growth rates over the time interval $t_1$-$t_2$ are calculated by substracting the number of publications at time 1 from the number of publications at time 2, and by dividing this number by the number of publication at time 1. The ratio is multiplied by 100 to express the growth rate in percentages. Note the likely under-estimation of the number of publications on these topics in the PubMed database in 2009, due to artifactual lags in article uploading and recording in PubMed in recent years, such that later growth rate are thus likely to be underestimated.

What we see in Table 6.2 is a raw growth rate of obesity-related publication that far exceeds that of PubMed overall. Even as a ratio of PubMed total publications, as we see in Table 6.3, the growth rate of obesity-related publications is impressive. From 1989 to 1999, the ratio of publications with “obesity” in their title or abstract to PubMed grew by almost 100%, from 0.23% to 0.46%. Over 20 years, the ratio quadrupled (growth rate of 401.4%), to 1.17. Publications on obesity and cancer as a ratio of total PubMed publications grew by 633.5%; on obesity and diabetes 616.5%; on obesity and CVD 521.0%. The annualized growth rates for obesity-related publications as a ratio of total PubMed publications ranged from 20.1% to 36.2%, indicating they grew at a much faster rate than the total medical field. Research on childhood and obesity has seen the greatest expansion over this time period, a growth rate of 1,651% in the number of publications, and one of 724.7% controlling for the expansion of PubMed.

Comparing the growth rate of publications on obesity to total PubMed publications with population trends in weight gain, we see the time lag discussed by Stevens et al. (2006). Weights started increasing in the mid 1970s, while the medical profession was almost silent until the 1990s.

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11 The reader may back to Chapter 1 for this non-obvious connection.
When we plot the proportion of publications devoted to cancer, HIV and obesity in PubMed over time (not shown), we find that cancer’s share has been growing steadily since 1980, while HIV’s share has increased dramatically from 1986 to 1998, before declining sharply until 2002, and continuing since then on a slow steady, decline. Obesity, in contrast, has been rising slowly from 1980 to 1994. An inflection point can be seen in 1994, and another again around 2000. As we have seen in Chapter 5, WHO experts convened to define anthropometric standards that would define obesity cutoff points in 1993. The new millennium brought more interest in obesity, and a yet faster growth rate in publications on the topic.

Obesity, then, hasn’t featured in the medical conscience the way cancer has, in regular, growing, and progressive fashion. Nor has it grown like HIV, in epidemic-like fashion, with a sharp growth followed by a gradual decline as the problem stabilized in the West. In contrast, interest in obesity has grown in spurts, and those spurts are probably connected to external events such as the addition of obesity to the International Classification of Diseases in 1990, the WHO anthropometric standards in 1995, the 1998 NIH / NHLBI Guidelines and WHO Global Obesity Epidemic Report, and the 2001 Surgeon General Call to Action to Prevent and Decrease Overweight and Obesity (see Chapter 5).

6.2 Obesity as a Public Health Issue

Obesity has been framed by health officials as an important public health issue in the United States since at least 1952, when then NIH director W.H. Sebrell claimed that obesity had become the United States’ number one nutritional problem (Science News Letter, 27 December 1952). Based on the sociological literature, one would expect the public health literature to have expanded seriously over recent years as prevention started to replace acute care as preferred interventions.
Figure 6.4: Number of articles on the health costs of obesity, 1992-2009 PubMed Database.
Using the ratio of articles using “public health” as a MeSH term to the total number of PubMed articles that interest in public health has not increased regularly over time. Rather, it climbed steadily until 1970-4 before declining and picking back up starting in 1980-4 (Figure 6.3). If we focus on the 1980-2009 time period, however, we see a rise in the ratio of publications on public health to the total number of publications in PubMed. The ratio of public-health articles that are specifically about obesity to PubMed has also been rising over that time period, and has been growing over time at a faster rate than public health overall, confirming the view of obesity as one of the most pressing public health issues. If we plot the public-health-focused obesity articles against all public health articles, we find that the steepest section of the curve starts in 1995, at about the same time as the medical discourse of obesity as an epidemic started, and as the WHO was establishing its new anthropometric guidelines.

6.3 The Economic Costs of Obesity

In Chapter 1 I briefly discussed the economic costs of obesity, and discussed issues with estimates of this financial tab. To understand when scientists first attempted to evaluate the financial costs of obesity, I queried ISI Web of Knowledge and PubMed with “economi*” and “obesity” as topics for ISI, and “obesity” among Titles and Abstracts and “health costs” as a MeSH term for PubMed. ISI Web of Knowledge returned 9,915 titles. None of the 50 most-cited articles on obesity found on ISI mentions the financial burden of obesity. PubMed returned 316 articles, including 299 in the 1992-2009 time range. Figure 6.4 shows the irregular but overall clear growth in the number of articles published on the economic costs of obesity over time.12

12 See also Chapter 1, section 1.9.
The first scientific article I could find on the economic costs of obesity was published in 1952 in the Medical Journal of Australia, titled “Standards of fitness from the life assurance viewpoint.” It received no citations, if we trust ISI Web of Knowledge. In PubMed, the first recorded instance is found in 1992, in an article by Graham Colditz cited 315 times, titled: “Economic costs of obesity.” In it, we learn that “a conservative estimate of the economic costs of obesity [is] $39.3 billion dollars [annually], or 5.5% of the costs of illness in 1986” for the 34 million Americans who are obese.

6.4 The Obesity Epidemic in Historical and Comparative Perspective

As we have seen in Chapter 3, some scholars have been critical of the use of the word “epidemic” to qualify the rise of obesity in the United States and across the world (Boero 2007, 2009; Campos et al. 2006; Oliver 2006a, 2006b). This critique has not looked comparatively at the use of “epidemic” to qualify non-communicable diseases. Given how some epidemiologists – including Flegal (2006), herself a fat studies ally – see nothing uncanny in an epidemic of non-communicable diseases, it appeared worthwhile to investigate historically and comparatively the use of “epidemic” in conjunction with other non-communicable diseases (NCDs). I queried the PubMed database for “obesity,” “cardiovascular disease,” and “diabetes” along with the word “epidemic” over the 1950 to 2009 time period. I also queried PubMed for uses of “obesity,” “childhood,” and “epidemic” over the same time period. Data are presented below are normalized for the growth in the number of publications recorded in the PubMed database.

Because the associations of these keywords with the epidemic discourse happened in very low frequencies until the 1990s (average of 2.75, 9.75, and 22.25 mentions per decade between 1950 and 1989 for “obesity AND epidemic,” “diabetes AND epidemic,” and “cardiovascular disease AND epidemic” respectively), Figure 6.5 documents the
trends in usage of the epidemics language in association with different keywords from 1990 to 2009.

When was epidemic first associated with non-communicable diseases? Querying PubMed, we find that “diabetes” was first used alongside “epidemic” in 1954; with “cardiovascular disease” in 1960; and with “obesity” in 1976, although this first use was associated with epidemic rates of coronary accidents, rather than epidemic obesity.

Diabetes seems to have been one of the first NCDs to be called an epidemic in the medical literature. As we see on Figure 6.5, in the early 1990s, the mentions of epidemic diabetes trumped other epidemic associations with NCDs, trading the lead position with cardiovascular disease until 1999, when epidemic obesity trumped both. Starting in 2000, mentions of “epidemic diabetes” decidedly trumped mentions of “epidemic cardiovascular disease,” but obesity stayed in the lead position and increased its share inconsistently since.

The actual phrase “epidemic obesity” was first used in the medical press in a commentary made in 1993 by J. Stamler on an Archives of Internal Medicine article published earlier that year by Denke, Sempos, and Grundy (1993), and titled “Excess body weight. An underrecognized contributor to high blood cholesterol levels in white American men.” Stamler’s commentary has only been cited 72 times, as seen in the ISI Web of Knowledge database, and thus can’t be argued to have been the initiator of a large linguistic revolution. The next instances are not found until 1996. Similarly, childhood obesity was first used with epidemics in a 1994 article published by the Canadian Medical Association Journal, and not used again until 1998. These early instances, then, do not suffice to explain the explosion of this discourse over subsequent years.
Fig. 6.5: Epidemics Discourse in Titles and Abstracts as Ratio of Total PubMed Publications, 1990-2009

- Obesiry AND Epidemic AND Childhood
- Cardiovascular Diseases AND Epidemic
- Diabetes AND Epidemic
- Obesity AND Epidemic
Indeed, the quick rise of the association between obesity and epidemic in PubMed, as well as the numerical dominance of the obesity epidemic discourse, is visible starting in 1996. In 1999, “obesity” was used in conjunction with “epidemic” in just over one publication per ten thousand; by 2009, it was used in close to six publications per ten thousand, close to twice as often as diabetes, and almost three times as often as cardiovascular disease. The exact phrase “obesity epidemic” or “epidemic obesity” was used in about half of these publications (222 out of 477, or 47%), and the association between obesity, diabetes, and the phrase epidemic was almost equivalent to one third of all these associations (142 out of 477, or 30%). Clearly, obesity has quickly became associated with the epidemics discourse, and it seems safe to hypothesize that its connection to diabetes may have played an important part in the use of the epidemic language.

Obesity as an epidemic had, however, already been used in the popular press. A LexisNexis query into World News for “epidemic of obesity” or “obesity epidemic” traced the first instance to 1977, in a Newsweek article titled “Diet Crazes.” By May 1993, when Stamler published his comment, there had been as little as 13 instances of epidemic obesity in the news, 8 of them published in February of 1993 as a response to the publication of a study in *Pediatrics* by Robinson et al. (1993) titled “Does Television Viewing Increase Obesity and Reduce Physical Activity? Cross-Sectional and Longitudinal Analyses Among Adolescent Girls,” a study that did not include the word epidemic. In 1997, a year before the WHO Report on the global epidemic of obesity, 38 articles used the phrase; in 1998, 37. In 2000, the year before the Surgeon General’s *Call to Action to Prevent and Decrease Overweight and Obesity* which declared that the United States was experiencing an epidemic of obesity, there were 114; in 2001, 178; in 2004, 1,380; and
in 2009, 1,017, at a very high 2.8 articles per day in the printed press only. For every medical article on obesity, there were five news articles on the topic in the United States. Embedded as they are in a broader culture, medical doctors and researchers are likely to have been influenced by this extensive coverage.

Turning to childhood obesity in particular, I first return to Table 6.2, where we saw the immense growth in interest on childhood obesity in recent decades. Figure 6.5 suggests that the epidemic language has also been increasingly applied to childhood obesity. When we probe into the association between “childhood,” “obesity,” and “epidemic,” we see a confirmation of the growing interest in childhood obesity and its labeling as an epidemic. In 1990, there were only 33 articles on childhood obesity, and zero on the childhood obesity epidemic. By 2009, there were 683 articles on childhood obesity, 81 (or 11.9%) of which also used the word “epidemic.”

It is reasonable to expect that the childhood socialization assumption – that the child is father (sic!) of the man (sic!) – facilitates and exacerbates the medical and educational concerns in childhood obesity. What was previously seen as cute baby fat is now seen as a danger, an ominous sign of future health problems (Schwartz, 1986), but also the sign of future financial illness for the nation. As we have seen in section 6.3 above, interest in obesity as an economic crisis has been on the rise.

**Conclusion: Growth, Healthicization, and an Epidemic of Epidemics**

In this Chapter I have shown the impressive growth of obesity as an area of medical inquiry. The number of professional journals on obesity, as well as the number of publication on the topic across areas of inquiry has been impressive, and has experienced a growth that has exceeded the overall growth of the medical knowledge production. Starting in the 1970s, medical researchers and public health specialists have taken on the
investigation of every single aspect of obesity and its association with other health conditions. Mimicking previous trends in the use of “epidemic” in association with non-communicable diseases, in the late 1990s the idea of an obesity epidemic surfaced, and by 2002, obesity was a major presence in the medical discourse, and the epidemics discourse was expanding immensely.

Growth in the obesity discourse has not been linear; it has rather come in spurts that appear to have coincided with some of the major governmental and international enterprises to address the issue, and has been more pronounced over the last decade. The process whereby the medical literature influenced (inter)governmental action is unknown, and so is the process whereby (inter)governmental action influenced the medical literature. Yet the literature reviewed by Saguy and Almeling (2008) shows that this growth has not happened in a cultural vacuum.

The medicalization hypothesis is an important starting point to understand the rise of obesity in the medical discourse. Yet it may not be sufficient to explain the history of diabetes, cardiovascular disease and obesity as epidemics, nor to explain why obesity outpaced other epidemics in the late 1990s and has been taking a bigger share of medical publications since. Oliver (2006a) argues that the rise and ubiquity of the “obesity epidemic” language results from the combined interests of the medical profession, of the weight-loss industry, and public health bureaucracy. Obesity, in his account, is only but one case of the “transformation of nonpathological physical states into diseases” based on a change in the “supply and demand of medicine,” in other words: medicalization. His explanation thus focuses on the “health-industrial complex:” the individual and organizational actors who pushed for the recognition of obesity as a disease and thus enabled medical doctors to treat it and insurance companies to cover this treatment.
Further research into the history of the epidemics language in comparative perspective is likely to provide a sociologically-grounded explanation of the rise of several epidemics of non-communicable diseases in the medical discourse starting in the 1950s, and its spread around the world. This research may ask, first: Who are the actors pushing for the epidemics language? How do they rationalize their use of epidemics for non-communicable diseases? Second, it may turn to the factors which make epidemics “stick” and get traction. Why did obesity gain such prevalence among the epidemics discourse? Factors to consider may include (1a) the straightforwardness of the diagnosis (BMI in the case of obesity; more complex tests in the case of diabetes; and a range of tests or a cardiovascular failure in the case of CVDs) and (1b) the associated easiness of trends following at the population level; (2) the seeming simplicity of the causal chain that leads to the “disease” (a caloric imbalance in the case of obesity; more complex causes in the case of diabetes and CVDs); and (3) the existence or mobilization of a community of practitioners and researchers to “solve” a perceived problem. Both Chang and Christakis (2002) and Sobal (1995) have argued that over the course of the 20th century the medical understanding of obesity shifted from a individual failure to an environmental casualty, from something one “does” to something one is “done to.” Is there an enlightened community of scientific converts or proselytizing patients trying to save the soul and health of fat people from environmental pressures? The research of Oliver (2006a) and of Bernstein and St. John (2009) suggests so. Were there such communities rallying behind diabetes and cardiovascular diseases?

Third, research into this issue may ask: Why did it gain momentum and dominance in the late 1990s? Is the 1998 WHO Report on the global epidemic of obesity
widely cited among the early obesity epidemic papers? What connections did early authors have to the WHO Report, the NIH, etc.?
Part III: America’s Children
Chapter 7: Fat Kids and The Rise of Obesity as an Educational Problem

Think about every problem, every challenge we face. The solution to each starts with education. For the sake of the future – of our children and the nation – we must transform America’s schools. President Bush Sr. (cited in Tyack & Cuban, 1995, p. 110).

This chapter addresses two intertwined issues: the growing medical interest in childhood obesity, and the growing discursive mobilization of schools and education to address adults and children’s obesity. In Chapter 1, I have noted the influence of maternal obesity on children’s future body weight and likelihood of impaired glucose tolerance and Type 2 diabetes mellitus. In Chapter 2, I documented the growing weights of children using NHANES data, and noted how 20% of youth aged 6-11 and 15% of youth aged 12-19 are currently overweight. Chapter 3 discussed among other things data showing that girls as young as 5 fear weight gain, that fat children are more depressed and isolated than normal-weight children, and that fat students are less likely to find academic support than similarly qualified students. In Chapter 5 I relied on Hillel Schwartz historical research to note how children’s weight became a cultural obsession starting in the 1940s, before extending to infants’ and toddlers’ in the 1970s. And in Chapter 6 I looked at the association of obesity with the following keywords: childhood, cardiovascular disease, cancer, diabetes, epidemic, and childhood epidemic. I noted how the first article using the phrase “childhood obesity” was published in 1935, but that widespread concern over the issue didn’t start until the 1980s, and escalated quickly, with a 725% growth rate as a proportion of PubMed from 1989-2009.

In this chapter, I document the growth in publications on obesity and on the obesity epidemic in its associations with schools, education and children. I then take a
closer look at the most cited papers on childhood obesity to map the paradigm of this research, and whether it is associated with schools. Finally, I look at the role education plays in the national plans to fight obesity of two very different countries, the United States and Singapore, to show differences and similarities in national strategies.

In my analyses, below, I sometimes group education and schools together, but most of the time keep them separate. Schools are relatively concrete: they are establishments where kids go to receive an education. Education is abstract, given its breadth as a category. One can educate parents and grandparents, pregnant mothers, infants, toddlers, children, adolescents, adults, the middle-aged and the elderly. Education includes schools, the media, informal education, parental influences, social and peer pressures; and it is both a process (the education of the masses) and a good (she has a lot of education).

But education is also the location of modern faith in the future. As famously noted by Tyack and Cuban (1995), educational reforms are often utopian: their instigators hope that by changing schools, they will be able to “correct perceived social and educational problems” (p. 4). Repeatedly, they write,

Americans have followed a common pattern in devising educational prescriptions for specific social or economic ills. Once they had discovered a problem, they labeled it and taught a course on the subject: alcohol or drug instruction to fight addictions; sex education to combat syphilis or AIDS; home economics to lower the divorce [sic] rate; driver education to eliminate carnage on the highway; and vocational training or courses in computer literacy to keep the United States economically competitive (Tyack & Cuban, 1995, p. 4).

Obesity has followed such a course: obesity was named as a social problem and, as I will show in this Chapter, health education and health promotion in schools has increasingly been mobilized to solve it.

Oliver (2006b), has noted:
The most obvious target for government intervention is schools. Most American children are in public schools that are financed by taxes and governed by state legislatures and locally elected boards. What happens within their walls is a legitimate target of public concern and one can reasonably assume that our schools should be doing everything possible to promote their students’ health, including encouraging physical activity, teaching proper health and nutrition, and making sure kids are eating foods high in nutritional value. … [M]ost American schools are not meeting these goals (Oliver, 2006b, p. 161)

Have education and schools been mobilized in the medical literature to address obesity in general, childhood obesity, and the childhood obesity epidemic? Trends from 1950 to 2009 show an irregular but clear growth for all these instances. Because there were very few instances of mention of schools in the medical literature on obesity until the 1980s, the analyses in this chapter focus on what happened in the medical literature from 1980 to 2009. Looking at the PubMed database over time, I discuss the association between obesity with education and schools, before turning to childhood obesity and its association with education. I then zoom in on the childhood obesity publications, in order to understand their contents and evaluate the connections they make or assume between schools, education, and childhood obesity.

7.1 Fat Lessons, Fat Kids: Obesity, Epidemics, Education and Schools in PubMed

7.1.1 Obesity, Education and Schools

Figure 7.1 shows the immense growth of the discursive association between education, schools and obesity in the PubMed database. From 1980 to 2000, growth was slow and uniform. Then, in 2002, the number of publications exploded. Between 2002 and 2009, articles on obesity that mention education rose from 116 to 469 (a 304.3% growth), while the number of obesity articles mentioning schools grew from 80 to 391 (a 388.8% growth).
Fig. 7.2: Ratio of Publications on Obesity and Associated Keywords to Total PubMed by Decade, 1980-2009
Fig. 7.3: Ratio of Publications on Obesity Epidemic and Associated Keywords to Total PubMed by Decade, 1980-2009
Because the annual number of publication is small and tends to fluctuate from year to year, and because the PubMed database itself has been expanding, for the sake of comparison it is useful to sum frequencies of publications by decade as a proportion of the PubMed database. As we can see on Figure 7.2, the ratios of publications on obesity and education and on obesity and school have seen a massive increase every decade from the 1980s to the 2000s: from 0.0042% to 0.0440% for education (947.6% growth), and from 0.0035% to 0.0288% for schools (722.9% growth). The raw numbers are also striking: from 139 publications in the 1980s, and 511 in the 90s, obesity and education have figured in the titles and abstracts of 2,820 publications in the 2000s. Publications on schools similarly increased: from 114 mentions in the 1980s, to 305 in the 90s, and to 1,936 in the 2000s.

The share of obesity publications that mention education or schools to the total number of publications on obesity has also been growing over time. Education featured in 1.9% of obesity publications in the 1980s, and in 14.1% of them in the 2000s, and schools were mentioned in 1.5% of obesity publications in the 1980s, and 9.7% over 2000-2009.

7.1.2 Childhood Obesity, Education and Schools

Overall, it appears that in the case of obesity broadly understood, education trumps schools in the medical discourse. Is it also the case with childhood obesity? The first recorded instance of the use of “childhood obesity” in a title or an abstract in PubMed is Fletcher’s (1935) case study of a child with “multiple congenital abnormalities.” Frequencies of publication on obesity among children, infants, and youth were very low until the year 2000, and infant obesity wasn’t of real interest throughout most of the period, reaching 50 annual publications only in 2007. Child obesity reached
that level in 2000, and youth obesity in 2005. The use of either infant, child or youth obesity in titles or abstracts reached 50 annual instances only in 1998, even though the share of PubMed publications combining obesity and one of these three keywords in titles or abstracts has grown over time (see Figure 7.2).

Returning to Figure 7.1, we see an important growth in the number of publications on childhood obesity and both schools and education. In this case, however, schools are more often associated with childhood obesity than education, with 122 and 61 mentions in 2009, respectively, and a total of 696 publications on schools and childhood obesity, and 401 publications on education and childhood obesity from 1980 to 2009.

Figure 7.2 also shows how controlling for the expansion of PubMed, the ratio of publications on obesity and infant, child or youth, as well as in the use of the phrase childhood obesity, have also grown massively. In the 1980s, there were 229 mentions of childhood obesity; in the first decade of the 2000s, there were a staggering 3,772.

The ratio of articles on childhood obesity that also mention education or schools has also been growing: as a proportion of all PubMed articles, but also as a proportion of the articles on childhood obesity. Indeed, from 5.7% of articles on childhood obesity in the 1980s, articles that mention education in their titles and abstracts grew to 9.0% over 2000-9. Similarly, articles on childhood obesity and schools represented 9.2% of childhood obesity articles in the 1980s, and 16.2% over 2000-9.

7.1.3 Obesity Epidemic, Childhood Obesity Epidemic, Education and Schools

Figure 7.3 shows similar trends in the use of education and school in association with “obesity epidemic.” While the number of such articles is small, the growth remains impressive, in particular for the use of childhood obesity in association with the
epidemics language: from one instance in the 1980s, the childhood, obesity, and epidemic query got 438 hits in the first decade of the new millennium. The proportion of articles on the childhood obesity epidemic that refer to education and schools in the 2000s is also high: 12.1% in the case of education, and 22.4% in the case of schools.

The data reviewed here suggests that education and schools have been an important and growing part of the literature on obesity, childhood obesity, and the childhood obesity epidemic. Yet the number of articles on childhood obesity, totaling 4,773 over 60 years (79.55/yr on average), or on the childhood obesity epidemic, totaling 446 over 60 years (7.43/yr on average), is very small, and unlikely to have fallen into the ear of public health officials merely because of its magnitude. The story I am telling here, then, is incomplete, and will have to be complemented by further investigations.

7.2 What’s The Fuss All About? The Most Cited Articles on Childhood Obesity

To further probe into the current concern with childhood obesity, I queried the ISI Web of Knowledge database, which allows to rank scientific publications by the number of citations they received. Of the 14,209 articles with childhood obesity as a topic or title, 2,935 of them, or 20.7%, used childhood obesity in their title. 2,384 articles mentioned education, 1,697 mentioned schools, and 687 included both education and schools, or 16.8%, 11.9%, and 4.8% of all childhood obesity articles, respectively. Childhood obesity articles that included the “epidemic” term numbered at 959 articles, or 6.7% of the total.

Over the past five years, of the 7,028 articles on childhood obesity (49.5% of all articles), 988 also had education as a topic, 1,495 had schools as a topic, and 440 had both (14.1%, 21.3%, and 6.3% of total, respectively). Comparing these numbers with those for
the full time period, we find a confirmation of the PubMed results discussed above, but with a twist. On the one hand, there is an immense growth in the number of articles on childhood obesity since 2005, yet education as a whole has shrunken slightly over the past 5 years, while the discussion of schools has dramatically expanded, at about twice the overall proportion it had over the full time period covered, from 1940 until 2010: from 11.9% to 21.3%. Schools have come to the forefront of the discussion of childhood obesity, featuring in more than one in five articles published on childhood obesity since 2005, perhaps as the need for more concrete solutions to the “problem” arose.

To complement the macro-level portrait traced above, I queried ISI Web of Knowledge and collected the ten most-cited articles for each of the following keyword combinations: “childhood obesity” as (1) a title or (2) a topic, and “childhood obesity” and “epidemic” in either (3) titles or (4) topics. This sampling strategy lead to the identification of 34 articles, with a mean number of citations of 380.7 (s.d. 305.86). The thirty-four articles coded are listed in Appendix IV. Codes were developed inductively and iteratively: as I read through the articles, I developed the list of codes, refined it, and applied it to all articles, making changes as limits of the coding scheme and new possibilities surfaced. The final coding scheme, found in Appendix V, consists of five main categories: (1) specifics about the title and abstract; (2) causal attributions for obesity, and potential intervention loci; (3) the type of study; and (4) publication journal characteristics.

Selected articles were grouped into five categories for analytic and comparative purposes: ten most-cited (average citation = 764.5), ten oldest (median publication year = 1998), ten most recent (median publication year = 2007), fifteen articles with “epidemic”
in their title, and all thirty-four articles. Table 7.1 shows coding results. Given the very small sample sizes, the following discussion should be seen as an exploratory effort.

**Titles and Abstracts.** A large proportion of all articles used obesity in their title, although most recent articles seemed to do it more than the older ones. Epidemic was used by 15 of the 34, which is not surprising since they were over sampled. The titles of all but two of the most cited articles include references to childhood obesity; the two who do not

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**Table 7.1: Characteristic of Chosen Subsets of 34 Most Cited Articles, Childhood Obesity and Childhood Obesity Epidemic, ISI Web of Knowledge**

<table>
<thead>
<tr>
<th>Basics</th>
<th>10 Most Cited</th>
<th>10 Oldest</th>
<th>10 Most Recent</th>
<th>15 with Epidemic in Title</th>
<th>All 34 Articles</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of Citations (s.d.)</td>
<td>764.5</td>
<td>274.7</td>
<td>590.9</td>
<td>135.1</td>
<td>380.7</td>
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<tr>
<td></td>
<td>(168.55)</td>
<td>(264.25)</td>
<td>(328.39)</td>
<td>(146.74)</td>
<td>(365.86)</td>
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<tr>
<th>Title and Abstracts</th>
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<th>n</th>
<th>n</th>
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<th>n</th>
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<tbody>
<tr>
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<td>6</td>
<td>9</td>
<td>12</td>
<td>80</td>
<td>27</td>
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<td>Overweight in Title</td>
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<td>3</td>
<td>2</td>
<td>13</td>
<td>7</td>
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<tr>
<td>Overweight or Obesity in Title</td>
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<td>9</td>
<td>9</td>
<td>13</td>
<td>87</td>
<td>31</td>
</tr>
<tr>
<td>Epidemic in Title</td>
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<td>6</td>
<td>15</td>
<td>100</td>
<td>15</td>
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<td>n</td>
<td>n</td>
<td>n</td>
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<tr>
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<td>0</td>
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<td>7</td>
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<td>3</td>
<td>20</td>
<td>7</td>
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<tr>
<td>Policy</td>
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<td>4</td>
<td>5</td>
<td>6</td>
<td>40</td>
<td>13</td>
</tr>
<tr>
<td>Average per article</td>
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<td>1.2</td>
<td>1.3</td>
<td>1.4</td>
<td>1.3</td>
<td></td>
</tr>
</tbody>
</table>

| Type of Journal                | 1.1| 1.2| 1.3| 1.4| 1.3|     |
| Pediatrics Journal             | 3  | 3  | 1  | 0  | 6  | 18  |
| Obesity Journal                | 0  | 1  | 2  | 3  | 20 | 5  |
| Top 20 Journal (ISI Science Impact Factor) | 5  | 3  | 3  | 3  | 20 | 12 |
| Nutrition                      | 1  | 2  | 0  | 1  | 7  | 2  |
| Diabetes                       | 0  | 0  | 0  | 1  | 7  | 1  |
| Public Health Journal          | 1  | 1  | 0  | 1  | 7  | 2  |

Notes:

- Includes six of the 10 most-cited papers.
- Includes two of the 10 most-cited papers.
include obesity use “overweight” in its technical definition for children: a BMI above the 85th percentile of age and gender appropriate growth curves.

Type of Study. Only two articles (6%) investigated biological processes, and they were both among the most recent articles. One of them, Frayling et al. (2007), titled “A common variant in the FTO gene is associated with body mass index and predisposes to childhood and adult obesity,” was also among the ten most cited, with 669 citations.

The most frequent type of study was correlational studies, by which I refer to studies where authors discuss large-n aggregate population data analyses or small-n studies to investigate the relationship between specific variables. There were fourteen such articles (41%) among the 34 most cited, including two that documented growing obesity trends among children.

The second most frequent type of study was the review article, with nine out of 34 (26%). Only one review was among the ten most cited articles. Five such studies had epidemic in their title, and were written to justify the need for public intervention.

Editorial, policy or theory pieces were next, at six articles (or 18%). Most of these articles (4 out of 6, or 66%) argued for more governmental intervention and regulation, and half of them discussed the toxic environment (3 out of 6, or 50%). Only one of these, an editorial by Rocchini (2002), was published in a top-20 journal, the New England Journal of Medicine. Titled “Childhood obesity and a diabetes epidemic,” it received 87 citations, making it the 27th article in line.

Only two articles were intervention studies: one a randomized controlled trial published in JAMA (Robinson et al. 1999), the other an observational study (Freedman et al. 1999). Both were among the ten most cited (with 682 and 698 citations), both were published in 1999, both investigated the effect of changing children’s habits (television
watching vs. soft drinks, respectively) on children’s BMIs, and both found positive effects.

*Causal Factors and Intervention Loci.* Policy, parents and the toxic environment were the most often mentioned causes of obesity and/or intervention loci in this articles, out of seven different attributions. 38% of all articles mentioned policy, 24% mentioned parents, and 21% mentioned toxic environmental conditions. On average, the most recent articles mentioned more factors and loci than both older and most-cited articles. Epidemics articles lead the group, with an average of 1.4 attributions per article.

Parents were discussed in 8 of the 34 articles (24%). Three of these investigated the link between obesity in childhood or adulthood with parental obesity; most of the others included parents as part of policy recommendations. They were over-represented among the oldest articles (40%), articles which evaluated the parental influences on childhood obesity.

Surprisingly given the data we saw earlier, schools or education were mentioned in merely two of the articles, who approach schools as vectors of information, and as privileged location for addressing the bad habits of children through proper socialization.

In sum, this exercise has shown that schools are not central to the arguments made in the most-cited articles on childhood obesity. While recent articles have become more focused on noting potential solutions for childhood obesity, schools and education still do not figure prominently. The small sample size does not warrant large claims, but rather highlights a potential puzzle: Are schools and education taken for granted as privileged location and strategy for socializing youth into healthy eating and exercise? If yes, then how can we explain recent policy emphases on schooling? The following section describes such emphases.
7.4 The U.S. Government and the Role of Schools

Schools and education have featured at least discursively in many of the action plans developed by health officials in order to address growing population weights. In what follows, I discuss three such action plans where schools feature prominently: two documents by the Public Health Service’s Office of the Surgeon General, published in 2001 and 2010; and the 2009 Let’s Move! initiative by the White House. I close this section with a quick review of Bill H.R. 5504.

7.4.1 Surgeon General’s Call to Action, 2001

The 2001 Surgeon General Call to Action to Prevent and Decrease Overweight and Obesity espoused the “CARE: Communication, Action, Research and Evaluation” framework, which it applied to “five key settings: families and communities, schools, health care, media and communications, and worksites” (p. 16). Strategies for families and communities focused on information provision and education, as well as on the creation of safe spaces for physical activity, including walking, cycling, and stairs climbing. The need for more research on the “factors contributing to the disproportionate burden” placed on low income populations and racial and ethnic minorities is noted, but little else targets this connection. Taxes for the wealthy or on junk food are certainly nowhere in sight; nor are discussions of financing.

Schools are “identified as a key setting for public health strategies” (p. 19). The central communication strategy focused on the education of teachers, food service staff, coaches, nurses, parents and all other adults who interact with children in (1) “the contribution of proper nutrition and activity” for weight maintenance, (2) the value of school physical activity, (3) their importance as role models, (4) the importance of body
size acceptance, and (5) the “problems encountered by the overweight child” (pp. 19-20).

Action strategies targeted the student, who is to be provided with

age-appropriate and culturally sensitive instruction in health education that helps ... develop the knowledge, attitudes, skills and behaviors to adopt, maintain, and enjoy healthy eating habits and a physically active lifestyle (Surgeon General 2001, p. 20).

Section 3, “The Power of People and Ideas,” calls for private/public interventions to translate the ideas of the Call for Action into “meaningful action” (p. 27).

The power of industry on the determination of the nutritional quality of food is noted, but how change will happen in this area isn’t noted. The belief in information and education for social change is alive and well here. Educate the child in the benefits of healthy eating and exercise, and s/he will grow up to be a healthy, active – and lean – adult.

7.4.2 Surgeon General’s Vision for a Healthy and Fit Nation, 2010

In her Vision for a Healthy and Fit Nation 2010, Surgeon General Regina M. Benjamin writes that what Americans need to stop the obesity epidemic is to:

remember that [they] will be more likely to change their behavior if they have a meaningful reward—something more than just reaching a certain weight or BMI measurement. The real reward has to be something that people can feel and enjoy and celebrate. That reward is invigorating, energizing, joyous health. It is a level of health that allows people to embrace each day and live their lives to the fullest—without disease, disability, or lost productivity (Surgeon General 2010, p. 12).

Nine years after her predecessor, Surgeon General Benjamin hopes to change the pathologizing language around obesity into something more positive: health, and fun. She also focuses on prevention. Schools, in that regard, have an important role, as they “play a pivotal role in preventing obesity among children and teenagers.” Schools are expected to design programs and wellness plans that will “promote physical activity and healthy
eating, reduce the rate of overweight and obesity among children and teenagers, and improve academic achievement” (p. 8). While they are not alone, but rather work in conjunction with parents, the medical community, and the communities they are part of, schools hold responsibility and promise for the future, be it measured as health, weight, or academic achievement.

Surgeon General Benjamin also has specific recommendations for the promotion of both nutrition and physical activity. Better, more appealing, and healthier foods should be available, along with water across campuses; high-calorie snacks should be limited in availability. School systems should require daily physical education and recess of at least 20 minutes daily, provide opportunities for intramural physical activity, and implement walk- and bike-to-school programs.

In comparing the 2010 strategy with the 2001 strategy, one sees both similarities and differences. Both reports share a structure and a philosophy: scientific evidence meets the two vectors of health (healthy eating and exercise) and an upbeat tone pushing for the mobilization of the whole American population into making the nation healthier. Education, and information are central pillars in both documents – furthering an individualistic vision of health, and reinforcing the belief in the power of education to change behavior and social conditions.

The 2010 strategies will not strike anyone as novel, and in some respects they have regressed since 2001. For example, raising awareness to the struggles of overweight children has disappeared from the agenda, and so have research priorities. In 2010 as in 2001, the issue of finding the financial or human resources to make school reform happen is dodged: there is no mention of resources, taxes, money, or funds in the
document. Everything rests on the good will and good judgment of individuals, with a brand new addition: the fun that can be had being healthy.

7.4.3 The White House's Let's Move! Program, 2009

Unsurprisingly, the White House's Let's Move! Program rests on foundations similar to those found in the Surgeon General's (2010) Vision. The website for the program, http://www.letsmove.gov, features bright colors, pictures of the Obamas in action (playing with children, speaking with youth, at a table with papers in a lofty room), and invitations to listen to the First Lady answering your questions, to watch videos of youth exercising with the President, or to the contest to design the official Let’s Move! application for iPhone. Technology is clearly on the side of American health: the website is rife with invitations to follow Let’s Move! on Facebook™, read its blog, join a local Meetup™ group, see videos on YouTube™, watch pictures on Flickr™ and sign up for email updates.

Five colorful tabs crown the website, and invite the reader, presumably mostly parents, to the site’s five main sections: (1) Learn the Facts – Raising Healthier Kids; (2) Eat Healthy – Food & Nutrition; (3) Get Active – Physical Activity; (4) Take Action – Simple Steps to Success; and (5) Join Us – Let’s Move Together. At the bottom of the home page, buttons invite parents, kids, schools, and more (!) to take action with “5 simple steps to success,” in a fun, playful font.

The structure of the site will be familiar to the reader. The edifice of scientific knowledge (section 1, why obesity is bad for children) is combined with practical guidelines on the two main vectors of health improvement, healthy eating (section 2) and physical activity (section 3), and a word choice that invites for collective mobilization (or speaks of peer pressure, depending on one’s ideological makeup): “Take Action!” “Simple
Steps!” “Join us!” and “Let’s Move Together!” Why? Because doing so will “solve the challenge of childhood obesity within a generation” (White House, 2010). References to empirical materials are conspicuously absent.

The contents of the site are very similar to the Surgeon General’s documents, although designed to be friendlier and more inviting. Parents and caregivers are encouraged to “set a great example for the whole family by creating a healthy environment at home,” to make fruits and vegetables available to children, and to encourage “walking, and playing, even shopping together … to make a real difference” (White House, 2010). They are also told that children “need 60 minutes of active and vigorous play every day to grow up to a healthy weight,” again without scientific references.

A story about the First Lady’s op-ed on the importance of a Child Nutrition Bill was among the “Most Popular” hits of the home page from its publication through the end of August (last visited August 31, 2010). The editorial in question, published in the Washington Post on August 2nd, combines personal stories with “facts” about the financial costs of obesity ($150 billion every year), a promotion of Let’s Move! and, more importantly, a plea for Congress to pass H.R. 5504 “as soon as possible” (Obama, 2010). Four hundred and eighty-eight people commented on the Washington Post article before the comments were closed.

The emphasis on movement in the White House initiative may seem to be a counterpoint to the trend noticed in Chapter 3 about cardiorespiratory fitness in medical research. It is important to note here the great progress that cardiorespiratory fitness has made on the public health agenda since the 70s (interview with S.N. Blair, 09/21/10). There is plenty of evidence that connects cardiorespiratory fitness to health, and it is
widely acknowledged in public health campaigns. It is the mediating effect of cardiorespiratory fitness in the case of obesity is what has been understudied, not its actual role in health.

7.4.4 H.R. 5504: The Child Nutrition Act

The Bill for which Michelle Obama wrote the op-ed, H.R.5504, Improving Nutrition for America’s Children Act, makes revisions to five federally-sponsored food programs (the school lunch and school breakfast programs, the summer food service program, the child and adult care food program, and the special supplemental nutrition program for women, infants and children). It attempts to streamline access to programs, increase funding, and align the food distributed in these programs with federal dietary guidelines. It has four Titles: I – A path to end childhood hunger; II – Improving nutrition quality; III – Improving the management and integrity of child nutrition programs; and IV – Miscellaneous, a rubric for the distribution of tasks, and other administrative details. This Bill received support from a whole range of constituencies and had a Full Committee on Education and Labor Hearing on July 1, 2010, which was followed by a Markup on July 14 and 15 and passed by a 32-13 vote. It is currently in line to be brought in front of the House for a vote (Committee on Education and Labor, 2010).

Estimated by the Congressional Budget Office to increase direct governmental spending by $2.0 billion over the 2011-2015 period, and by $6.5 billion dollars over the 2011-2020 period, H.R. 5504 has, surprisingly, received support corporatist support (see Appendix VII), and only private anger on websites and among comments on the First Lady’s Washington Post op-ed. It breaks with previous attempts at managing the health of the nation through individualistic, educational strategies. Instead, it follows in the path set
by Nestle and Jacobson (2000), and directly works at eliminating among children some of the “environmental barriers to healthy food choices and active lifestyles” (p. 12).

Beyond their educational and preventive functions, with H.R. 5504 schools are being given a strong hold onto children’s diets, particularly among the populations most affected by the federal programs under revision. Adults are intervening into the choices of children, and may also bring money along.

7.5 Singapore

Singapore is a city-state that has industrialized rapidly since its independence from Britain in 1965. Its population of close to 5 million consists in a large proportion foreign-born individuals, from three major ethnic groups: Chinese (majority), Malay, and Indian. Its government is centralized and authoritarian. As of 2008, 90% of Singapore’s food is imported (Soon et al. 2008).

Economic growth coincided with weight gain in the Singaporean population (Isono et al. 2009; Toh et al. 2002). According to Toh, Chew and Tan (2002), cancers and cardiovascular diseases were the main killers in Singapore in 2000, partly because of an “epidemiological transition” in housing, sanitary conditions, vaccination campaigns, disease surveillance, and “affluent lifestyles,” leading to the eradication of communicable diseases (p. 333-4).

In 1991 a committee made recommendations for improving the health of Singaporeans through health promotion and disease prevention. As a result, in 1992 the National Healthy Lifestyle Programme was adopted. From 1980 to 1991, obesity rates among students from primary, secondary and pre-university schools had almost tripled, from 5.4% to 15.2% (Toh, Chew and Tan, 2002). The Programme “adopted a multisectoral approach involving government ministries and organizations, health

154
professionals, employers, unions, and community organizations” (Cutter et al. 2001, p. 909). It included the Great Singapore Workout, launched in 1993 by Prime Minister Goh Chok Tong in order to invite Singaporeans to exercise more regularly.

Cutter, Tan and Chew’s (2001) evaluation of the National Healthy Lifestyle Programme compared NHS data from 1992 with data from 1998, and concluded that the “intervention had mixed results after six years” (p. 908). The first National Health Survey (NHS, 1992) had found that 8.4% of the population had diabetes mellitus, 5.1% were obese, and only 13.6% exercised at least three times a week. By 1998, rates of diabetes had gone down and obesity rates gone up, but not significantly so (compare with the U.S. rates, 23.3% in 1988-94, and 31.1% in 1999-2002). Exercise rates, however, had increased significantly over that time period. A second update, with 2004 data, showed diabetes declining -0.7% (NS), obesity rising 1% (NS), and regular exercise going up 9.0% (p<0.001) (Bhalla et al. 2006).

Children were specifically targeted as a part of the National Healthy Lifestyle Campaign of 1992 by the Trim and Fit (TAF) Program, whose slogan “Let’s be TAF,” is quite playful. It was intended to help all kids become more healthy, but targeted overweight and obese kids in order to “make losing weight through increased physical activity as enjoyable as possible” (Lee 2003, p. 49). It forced overweight youth to exercise weekly in school, often in view of their peers, and to count calories at meals. It also provided TAF awards to reward primary and secondary schools “for their efforts in creating greater awareness in students, teachers and parents of the importance and principles of healthy living” (Singapore Ministry of Education, 2010).

The language on the Trim and Fit website (Health Promotion Board, undated) is similar to that used by Let’sMove.gov when giving healthy eating advice: fruits and
vegetables, avoid fat, salt and sugar, and to drink water rather than soft drinks. When it comes to exercise, however, the tone is sometimes more authoritarian, and inconsistently gender neutral: along with the “exercise as a family” advice, we find “Get your child to exercise for 30 minutes, at least five days a week, until he or she perspires and breathes deeply without getting breathless,” and “Get him to help you wash the car or mop the house.”

Finally, the American goal of health and fun finds a Singaporean precedent in the following: “With a healthy, balanced diet and enough exercise, you can be sure of a trim and fit child who is healthier, as well as looks and feels better. As a parent, I am sure that is exactly what you wish for your child.” Goals are health, of course, but also physical appearance, something that doesn’t find its way to the American website.

While TAF has generally accepted as successful in reaching its goals, it was criticized for being stigmatizing, with kids in the TAF program often dubbed FAT kids (AFP, *Times Live South Africa*, February 2005; Isono, Watkins & Lian, 2009). Indeed, among anorexic patients studied by H.Y. Lee et al. (2005), 11.1% noted they were members of the TAF program, and about half of them listed this fact as a precipitator of their illness. Several of the eating-disordered youth interviewed by Isono, Watkins and Lian (2009) reported that TAF had contributed to their psychological distress. One of them, Lisa, said: “There was a stigma associated with going. I used to hate telling people I was in it because they used to call it fat club in school. Think abut it, TAF is the backwards of FAT. Not a nice acronym at all” (p. 133).

TAF was replaced in 2007 by the Holistic Health Framework (HHF). According to its website, the Holistic Health Framework aims to help schools “bring together in a purposeful manner the key areas, programmes and processes that develop the physical,
mental and social health of their students” (Singapore Ministry of Education, 2010). Its three guiding principles, total well-being, inclusion, and quality delivery, are aligned with the “holistic” goal, healthist individualistic ideas on the power of information, and also clearly a response to the criticism of apartheid and exclusion waged at the TAF program, as three sections will make clear: one titled “Why introduce the Holistic Health Framework,” where one reads that health promotion had to be broadened “beyond obesity and fitness management”; another titled “Is the Trim and Fit (TAF) Programme scrapped?”; and a third titled “Resources for Weight Management” that links to 17 different Ministry of Education web pages and PDFs.

The HHF, just like TAF, focuses on schools as intervention sites for health improvement. It encourages schools to develop comprehensive health programs and awards US$5,000 CHERISH prizes (Championing Efforts Resulting in Improved School Health) to every levels of schooling (Soon et al. 2008, p. 8).

**Conclusion**

In this chapter, I have shown how education and schools have been increasingly mobilized in the medical literature on obesity overall, as well as in the literature on childhood obesity. I have argued that while this growth has been important, the relatively small role played by education and schools in the medical literature suggests that current initiatives to manage American weights were not merely driven by the medical establishment.

Obesity and childhood obesity have increasingly been framed as social problems, and even as crises. Gloomy predictions have been made about the precariousness of the United States’ economy in the face of an ageing and increasingly obese population. Tyack and Cuban (1995) note how, often, reform is triggered by “some major societal change –
typically called a ‘crisis’,” that is either domestic or international (p. 43). Is it the case with obesity? The evidence reviewed in this chapter isn’t sufficient to answer this question.

Yet the apparent decoupling of mentions of schools and education from the main object of study among the most-cited articles on childhood obesity seems to illustrate the process described by Tyack and Cuban whereby social problems are made into educational problems. The utopia of schooling as a panacea for all social ills and social discomforts may have exerted its influence in the case of obesity, as it has before for divorce rates, falling international test scores, and teen pregnancy.

Like sex education, anti-obesity education of the form promoted by the White House attempts to solve what has come to be defined as a social problem by teaching children proper eating behaviors and exercise regimen. Socializing citizens into healthy ways of life starts in schools, and is expected to carry on into children’s adult lives.

As we have seen, in 1992 Singapore funded school-based interventions targeting overweight kids. They were overturned for their stigmatizing effects in 2007, and replaced by the Holistic Health Framework. The new program, which did away with individual targeting, has the same goals, the same strategies, yet an extended focus: all kids are now at risk of obesity and ill health, and thus they must all partake into the socialization into healthy behavior.

The bottom-up strategies of the United States, the well-meaning parents, school teachers, and communities targeted by the Surgeon General reports of 2001 and 2010 stand in sharp contrast with the top-down strategies of Singapore. Yet H.R. 5504, the renewed Child Nutrition Act, uses schools for purposes other than anticipatory socialization, the preferred American strategy. Indeed, while changing the American diet may be beyond the educational reach of schools, as decades of health education with
worsening health outcomes belie, the “feeding function” of school might well be a first, unpopular step, in the direction of greater governmental intervention in the control of weight.
Chapter 8: Conclusion and Future Research

In this dissertation, I have tried to historicize our current cultural obsession with weight. I have discussed the different steps that turned fat into a medical problem, a social problem, an epidemic and a serious child health issue. I have also argued that beyond the medical aspects of fat as a health issue, inertia along the path of medicalization has made the study of fat grow faster than population’s weights.

In Chapter 1, we have seen the documented health effects of obesity, along with some of the mechanisms whereby extra adipose tissue may lead to these effects. In Chapter 2, we have seen the rising weights of the population since the 60s. These findings were put in perspective using the evidence highlighted by “fat scholars,” vocal critics of the dominant medical paradigm.

In Chapter 4, I have argued for a mixed model of obesity, one in which the health consequences of being fat are dependent both on physiological mechanisms, and on social pressures. I have repeated the limitations of our knowledge on the medical impacts of weight net of other factors such as weight cycling and cardiorespiratory fitness, and suggested we exercise caution in our public health messages and interventions.

In Chapter 5, we have seen that although concern with the health risks of extra weight started in the late 18th century, it isn’t until the late 20th century that it actually became a disease and that the medical and public health establishment mobilized to address it. We have also seen the historical medicalization of fat, and a more recent healthicization of fat.

Chapter 6 supported the medicalization of fat argument. Rates of publication of articles on obesity showed a dramatic increase in the medical discourse in recent years, across different obesity-related health conditions. The early 1990s saw a dramatic
explosion of the number of publications on obesity, at the same time as the medical establishment was building the foundations of the medicalization of obesity, with its inclusion in the *International Classification of Diseases*, a new NIH consensus on the use of surgery for the treatment of obesity, the convening of an expert panel at the WHO, the publication of anthropometric guidelines for its evaluation, and the publication in 1998 of a WHO Report on the globalization of the obesity epidemic.

Public health interest in obesity has fluctuated since the 50s, but has been growing since the early 80s, finding its fastest growth rate after 1995.

The obesity epidemic discourse was put in perspective through a comparative approach to other epidemic non-communicable diseases. This discourse was shown to have first emerged with diabetes in the 1950s, then with cardiovascular disease in the 1960s. The obesity epidemic was absent from medical publications until a 1993 editorial, but grew significantly in the late 1990s to trump all other epidemic discourses.

In Chapter 7, I investigated the growing use of education and schools as means to solve the obesity “problem,” as well as the growing and changing mobilization of schools to solve childhood obesity. Among the most cited articles on childhood obesity, schools and education were not often mobilized. They were, however, pillars in the strategies of the Surgeon General to solve the national obesity “epidemic,” both in 2001 and in 2010, and are central in the White House’s *Let’s Move!* Program.

Finally, I have argued that Bill H.R. 5504 may be the source of the first significant leverage schools have had to address child health since the 1960s, by moving beyond their educational mission to their food-to-mouth mission. By providing financial resources to schools in order to improve child nutrition in situ, the federal government is
giving teeth to the previous discourse on education and information as salvation for the nation.

Medicine and medical research, given their authoritative position in American society, have shaped our lay understandings of body size: they have made us lipoliterate who see in fat more than merely an issue of esthetics. We read fat as an indicator of poor health.

Institutionalist views of science as legitimizing system would predict the growth of a medicalized view of fat, and the taken-for-grantedness of its ill-effects on health. The evidence featured in this dissertation points strongly in this direction: immense discursive growth rates across subfields, taken-for-grantedness of certain “facts” about the importance of exercise and of weight loss even in the absence of rock-solid evidence.

This dissertation also highlighted the important constraints faced by individuals when it comes to food consumption, exercise, weight and health. It also stresses the trickle-down effects of the pathologization of fat on stigma experienced by fat people.

The rise of individualistic and non-interventionist politics since the 18th century, in parallel with the rise of medicine as a social institution, have partly led us where we are today: with a set of beliefs about the individual as master of his or her own house / body, and the belief in education as a solution for most social ills. Indeed, contemporaries are strong believers in the salvation power of anticipatory socialization. In the case of health, this is manifest in the rise of public health and of preventive strategies, and in the recent mobilizations of schools to address both adult and childhood obesity.

As concern over growing weights spread around the world, such that there is current talk about a “global epidemic of obesity,” the United States is not alone in its mobilization against weight gain. We have seen, with the case of Singapore, how almost
two decades ago governments initiated strategies to that effect. Singapore, with its authoritarian government, acted strongly and early; the United States, non-interventionist state par excellence, is lagging behind. Ten years after the Singaporeans, the Surgeon General rang the bell. Twenty years later, the House of Representatives may approve of a bill to address junk food behaviors in school, and finance school-based programs to intervene in kids’ lives and “eradicate” childhood obesity within a generation, if we believe First Lady Michelle Obama.

This dissertation has highlighted the need for more research across a range of fields. First, in the medical research field, the connection between cardiorespiratory fitness and health outcomes deserves further study. Critical area of study would include: the development of measures for use in population-level research; the understanding of habit formation; and the impact of weight-neutral strategies on health outcomes.

Second, at the intersection of medicine and sociology, the role of stigma in the production of negative health outcomes will matter, particularly when it comes to the definition of the mechanisms that connect one with the other, and in the education of medical professionals.

Third, sociological inquiries in the rise of the epidemics language in the United States and around the world will shed light on the processes of knowledge production and diffusion. The role of expertise, of world society, of the measurability and portability of outcomes will be worth exploring.

Fourth, the gendered consequences of the medicalization of fat should be further investigated. How have women and men responded to the medicalization of bodily fat? How do they understand and relate to the discourse on health and fat? Has this discourse also affected families?
These four areas for future research will all benefit from historical, comparative study. Which macrosociological factors influence country-level responses to rising bodily weight? Are there cultures where being fat does not cause illness, and if so, what would this mean for our understandings of fat-related pathologies, stress and stigmatization?

When and where does “healthy living” first show up in textbooks, and participate in the definition of appropriate childhood and appropriate anticipatory socialization of citizens?

Given current obesity rates and the difficulty of weight loss reviewed in Chapter 3, it is unlikely that such rapid change will happen. Comprehensive strategies at all levels of government will be necessary to counteract the effects of the toxic environment and the nutritional transition characteristic of urbanization and post-industrial cities. Changing the meaning of fat will have to be part of these comprehensive strategies, if we want to minimize harm and maximize health.
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</tbody>
</table>
Appendix II – Fifty Most-Cited Articles on Obesity


Lagouge, M., Argmann, C., Gerhart-Hines, Z., Meziane, H., Lerin, C., Daussin, F.,


Appendix III – Important Dates in the Medicalization and Healthicization of Fat

1779(2) Weight loss advised to reduce risk of angina pectoris
1920s(3) Basic institutions of American beauty culture are established (fashion, cosmetics, beauty pageants, modeling profession, movies)
1921(2) Actuarial tables of weight and mortality risk
1940s(2) Growing interest in youth obesity
1942(2) Metropolitan Life ideal weight tables note that obesity is major national problem
1946(6) World Health Organization Charter defines health as a state of complete physical, mental, and social well being, and not merely the absence of disease or infirmity
1952(1) American Heart Association: Food for Your Heart
1954(4) Kremen and colleagues develop intestinal bypass surgery
1967(*) Creation of the Association for the Study of Obesity (UK)
1969(4) Payne and DeWind invent jejunoileal bypass surgery
1970s(2) Growing interest in infants’ and toddlers’ weights
1970s(3,5) Weight has become national obsession: Twiggy and rising rates of anorexia nervosa
1970(1) White House Conference on Food, Nutrition, and Health
1971(1) American Diabetes Association: Principles of Nutrition and Dietary Recommendations
1974(1) National Institutes of Health: Obesity in Perspective
1974(1) The Lancet editorial claims that adult and child obesity are the most important nutritional diseases in affluent countries
1977(1) National Institutes of Health: Obesity in America
1978(*) National Institutes of Health Consensus Statement: Surgical Treatment of Morbid Obesity
1980(1) US Department of Agriculture and US Department of Health and Human Services: Dietary Guidelines for Americans
1982(*) Creation of the Obesity Society (US)
1985(*) National Institutes of Health: Health Implications of Obesity. Recommends the use of BMI by medical practitioners, based on 85th percentile cutoff points.
1986(*) First International Conference on Health Promotion: Ottawa Charter for Health Promotion
1989(1) National Research Council: Diet and Health: Implications for Reducing Chronic Disease Risk
1990(6) Obesity is found in the International Classification of Diseases
2001(*) U.S. Department of Health and Human Services: *Surgeon General’s Call to Action to Prevent and Decrease Overweight and Obesity*

2002(7) Polls show that Americans see obesity as the third most important public health problem in the United States, after cancer and HIV.

2007(*) US Department of Health and Human Services: *Childhood Overweight and Obesity Prevention Initiative*

2008(*) US Department of Health and Human Services: *Physical Activity Guidelines for Americans*

2009(*) Center for Disease Control and Prevention: *Recommended Community Strategies and Measurements to Prevent Obesity in the United States*

2009(*) (January) U.S. Department of Health and Human Services: Surgeon General makes a public declaration in favor of reframing the national discourse on obesity toward health at every size, rather than disease.

2009(*) (April) Michelle Obama launches the *Let’s Move!* program.

2010(*) (January) U.S. Department of Health and Human Services: *The Surgeon General’s Vision for a Healthy and Fit Nation*

Sources:
Appendix IV – Articles on Childhood Obesity Coded for Section 7.2.


Lucas, A. and A. Singhal (2010). Reducing the extent or occurrence of long-term adverse health effects e.g. long-term vascular effect, development of atherosclerosis and propensity to obesity, comprises feeding a nutrient-containing-formula to the newborn infants. Univ College London.


Appendix V – Coding Scheme, Most Cited Articles on Childhood Obesity and the Childhood Obesity Epidemic, ISI Web of Knowledge

**Basics**

- Number of Citation Received (As listed in ISI Web of Knowledge on August 24, 2010)
- Year of Publication

**Title and Abstracts**

- Obesity in Title (Y/N)
- Overweight in Title (Y/N)
- Overweight or Obesity in Title (Y/N)
- Epidemic in Title (Y/N)

**Type of Study**

- Biological Processes (Y/N): Authors discuss the discovery of a new biological process.
- Correlational Study (Y/N): Authors discuss large-n aggregate population data analyses or small-n studies and investigate the relationship between specific variables is investigated.
- Editorial, Policy or Theory Piece (Y/N): Authors express their opinion on a topic and back it up with data.
- Guidelines (Y/N): Authors develop guidelines for the study or treatment of a condition.
- Intervention Study (Y/N): Authors investigates the effects of a treatment on an outcome.
- Review (Y/N): Authors review a body of literature systematically in order to advance the state of knowledge or develop policy interventions.

**Causal Factors and Intervention Loci**

- Gene (Y/N)
- Diet (Y/N)
- Physical Activity (Y/N)
- Parents / Family (Y/N)
- Schools or Education (Y/N)
- Toxic Environment (Y/N)
- Policy (Y/N)

**Type of Journal**

- Pediatrics Journal (Y/N)
- Obesity Journal (Y/N)
- Top 20 Journal (ISI Science Impact Factor) (Y/N)
- Nutrition (Y/N)
- Diabetes (Y/N)
- Public Health Journal (Y/N)

Education

Provide federal funding to state public health departments for mass media health promotion campaigns that emphasize healthful eating and physical activity patterns.

Require instruction in nutrition and weight management as part of the school curriculum for future health-education teachers.

Make a plant-based diet the focus of dietary guidance.

Ban required watching of commercials for foods high in calories, fat, or sugar on school television programs (for example, Channel One).

Declare and organize an annual National "No-TV" Week.

Require and fund daily physical education and sports programs in primary and secondary schools, extending the school day if necessary.

Develop culturally relevant obesity prevention campaigns for high-risk and low-income Americans.

Promote healthy eating in government cafeterias, Veterans Administration medical centers, military installations, prisons, and other venues.

Institute campaigns to promote healthy eating and activity patterns among federal and state employees in all departments.

Food labeling and advertising

Require chain restaurants to provide information about calorie content on menus or menu boards and nutrition labeling on wrappers.

Require that containers for soft drinks and snacks sold in movie theaters, convenience stores, and other venues bear information about calorie, fat, or sugar content.

Require nutrition labeling on fresh meat and poultry products.

Restrict advertising of high-calorie, low-nutrient foods on television shows commonly watched by children or require broadcasters to provide equal time for messages promoting healthy eating and physical activity.

Require print advertisements to disclose the caloric content of the foods being marketed.

Food assistance programs

Protect school food programs by eliminating the sale of soft drinks, candy bars, and foods high in calories, fat, or sugar in school buildings.

Require that any foods that compete with school meals be consistent with federal recommendations for fat, saturated fat, cholesterol, sugar, and sodium content.

Develop an incentive system to encourage Food Stamp recipients to purchase fruits, vegetables, whole grains, and other healthful foods, such as by earmarking increases in Food Stamp benefits for the purchase of those foods.

Health care and training

Require medical, nursing, and other health professions curricula to teach the principles and benefits of healthful diet and exercise patterns.
Require health care providers to learn about behavioral risks for obesity and how to counsel patients about health-promoting behavior change.

Develop and fund a research agenda focused on behavioral as well as metabolic determinants of weight gain and maintenance, and on the most cost-effective methods for promoting healthful diet and activity patterns.

Revise Medicaid and Medicare regulations to provide incentives to health care providers for nutrition and obesity counseling and other interventions that meet specified standards of cost and effectiveness.

**Transportation and urban development**

Provide funding and other incentives for bicycle paths, recreation centers, swimming pools, parks, and sidewalks.

Develop and provide guides for cities, zoning authorities, and urban planners on ways to modify zoning requirements, designate downtown areas as pedestrian malls and automobile-free zones, and modify residential neighborhoods, workplaces, and shopping centers to promote physical activity.

**Taxes**

Levy city, state, or federal taxes on soft drinks and other foods high in calories, fat, or sugar to fund campaigns to promote good nutrition and physical activity.

Subsidize the costs of low-calorie nutritious foods, perhaps by raising the costs of selected high-calorie, low-nutrient foods.

Remove sales taxes on, or provide other incentives for, purchase of exercise equipment.

Provide tax incentives to encourage employers to provide weight management programs.

**Policy development**

Use the National Nutrition Summit to develop a national campaign to prevent obesity.


Expand the scope of the President's Council on Physical Fitness and Sports to include nutrition and to emphasize obesity prevention.

Develop a coordinated federal implementation plan for the Healthy People 2010 nutrition and physical activity objectives.
Appendix VII – Supporters of Bill H.R. 5504 – Improving Nutrition for America’s Children Act

<table>
<thead>
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<td>International Dairy Foods Association</td>
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<td>American Academy of Pediatrics</td>
<td>International Union, United Automobile, Aerospace &amp; Agricultural Implement Workers of America (UAW)</td>
</tr>
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<td>American Beverage Association</td>
<td>National Advocacy Center of the Sisters of the Good Shepherd</td>
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<td>American Dental Association</td>
<td>National Association of School Nurses</td>
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<td>American Diabetes Association</td>
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<td>National Council of La Raza</td>
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<tr>
<td>American Federation of State, County and Municipal Employees</td>
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<td>American Heart Association</td>
<td>National Parent Teacher Association</td>
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<td>American Humane Association</td>
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<td>Bread for the World</td>
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<td>Campaign to End Obesity</td>
<td>Service Employees International Union</td>
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<td>Catholic Charities USA</td>
<td>Sun-Maid Growers of California</td>
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<td>Center on Budget and Policy Priorities</td>
<td>United Fresh Produce Association</td>
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</table>

Source: [http://www.govtrack.us](http://www.govtrack.us)
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Epidemiology, 35, 79-80.


Solomon, C. G., & Manson, J. E. (1997a). Obesity and mortality: a review of the
the Surgeon General.


