GLOBALIZATION AND OBESITY: A LONGITUDINAL, CROSS-NATIONAL

EXAMINATION OF A GLOBAL EPIDEMIC

by

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ABSTRACT

Obesity is gaining attention as a worldwide problem, particularly among poor and emerging economies. Evaluating leading obesogenic theoretical pathways from a global structural perspective exposes the effect of globalization on body weight. I test competing obesogenic pathways cross-nationally to assess economic development and food security mechanisms among poor nations. I also test the influence of structural convergence theories on body mass to measure their respective magnitudes. Cross-national longitudinal regression analyses are implemented to develop obesity theory sympathetic to macrostructural research in economic development. Results suggest international obesity to be, in part, the effect of counterintuitive effects of foreign investment exacerbated by existing economic vulnerabilities.

TABLE OF CONTENTS

Chapters

APPENDIX

LIST OF FIGURES

INTRODUCTION: THE DETERMINANTS OF

GLOBAL OBESITY

Obesity prevalence has increased at an alarming rate in both developed and developing nations in recent years (Caballero 2007; Dietel 2002; Ezzati et al. 2005; Kumanyika et al. 2010; WHO 2000). Worldwide prevalence of global obesity is estimated to outnumber those suffering malnutrition, reaching over 1.5 billion as of 2008 (WHO 2012). The most important trend though, is that while obesity has traditionally concentrated among the wealthy countries, it is now rapidly growing among lower income countries. What was once a disease of affluence is now manifesting itself as a disease of poverty (Flegal 1999; Popkin 1999; Popkin and Doak 1998; WHO 2000).

With so many presumed pathways of obesity in the global context, this project parses out their relative importance. In the following chapters, I examine the lenses through which obesity can be seen at the global level particularly as it is concomitant with economic development. In Chapter 1, I compare two perspectives that explain the rise of obesity among developing countries either as a function of economic marginalization or greater overall caloric availability using the influence of childhood stunting, nutritional access and several forms of economic development on body mass to clarify how obesity "spreads." In Chapter 2, I examine the role of contrasting global convergence perspectives in globalization and test the obesity convergence assumption. I

test whether the growing obesity epidemic is likely the result of the most notable form of global expansion, foreign investment, whilst comparing the influence of anti-obesityapproaches among civil society organizations. Based on my findings, I conclude by arguing that influences on obesity are not equal across all world regions and that the international obesity literature suffers from incomplete assumptions. I also argue global obesity trends are overshadowed by counterintuitive pressures resulting from economic development that lower body mass but are indicative of other, perhaps more serious negative health outcomes.

Relatively little research has been applied to how patterns of obesity relate to growing obesity prevalence among a diversity of countries. One of the guiding aims of this project is to more clearly articulate competing explanations for the growing obesity epidemic. The field of international obesity has grown to encompass several disciplines such as epidemiology, medicine, and public health. While such diversity has enabled broad consensus regarding its problematic trends few researchers have brought a theoretical paradigm to the problem. Among those who have, little research compares competing explanations in ways to clarify the gradual widening and deepening of global obesity rates. I elucidate how processes associated with international development and global market integration have brought the obesity epidemic to middle and low-income countries. This project aims to refine several theoretical frameworks which are useful, like the nutrition transition (Popkin and Doak 1998), but have proved incomplete in framing international obesity scholarship. I use the growing obesity problem to examine the extent to which global structures – specifically, economic markets and civil society – have a corporeal impact. These comparisons expose the paradox of poorer health granted

associated with global food distribution and how alleviating hunger may be taken for in the obesity literature. The goal of this project is to identify the key aspects of deterritorialized influences instrumental in driving the world wide obesity epidemic.

In this introduction, I first discuss why obesity matters. Obesity outcomes are prone to increase adverse health and financial burdens, but greater theoretical clarity is needed in order to unify diverse obesity-causing contexts and how, in doing so, understanding of public health outcomes are likely to be improved through applying findings from macrostructural research.

Next, I outline several areas that have stood to obscure a clear understanding of obesogenic pathways. I explain common methods of obesity measurement, and why Body Mass Index (BMI) has been accepted as the most useful. I then articulate how a major problem in conceptualizing obesity has arisen from the myriad ways it is contracted and its common health outcomes that are manifest among the population. These points, I argue, have served to dilute the use of coherent theoretical modeling of obesity and its structural antecedents. Subsequently, I argue, this area of scholarship could substantially benefit from a comparison between influential paradigms to which this research speaks. I then discuss a cross-national explanation of obesity which ranges across all levels of society such as calorie density, urbanization, and economic development. I focus on those influences which are most relevant in explaining the puzzling trend of growing obesity among both the poor and the wealthy. Lastly, I introduce the research and methods used in Chapters 1 and 2 and their respective contributions to sociological and obesity literatures.

Why Global Obesity Matters

The significance of this project could be viewed in several ways. First, there are substantial consequences of obesity, particularly in health outcomes, but also in the financial burdens it imposes. Obese individuals are more likely to experience poor health from comorbidty with several chronic conditions such as type II diabetes, cardiovascular disease, hypertension, stroke, asthma, sleep apnea, and several types of cancers (Lobstein and Jackson-Leach 2006; Rosin 2008). The leading causes of death worldwide – cardiovascular disease and diabetes – represent the greatest mortality risk from obesity (Bonow et al. 2002; Kumanyika et al. 2010; Zimmet, Alberti and Shaw 2001). In Latin America, one estimate indicated that 1 out of every 3 days in the hospital was diabetes related (Yach, Stuckler and Brownell 2006). Obesity represents a disease burden that is expected to increase. Initial projections suggested that obesity shortened the lifespan by an average of .17 years for every one percent of excess weight (Pauling 1958). Subsequent estimates confirm that obesity contributes to lower life expectancy (Olshansky et al. 2005; Preston and Stokes 2011; Swinburn et al. 2011). A definitive estimate of more precise loss in life expectancy is challenging because of a wide range of individual factors but comorbidities generally start at about 21 BMI and grow exponentially as BMI increases (Stevens, McClain and Truesdale 2006). In the case of the United States, the obesity epidemic has been estimated to cost the population the equivalent of 20 years (Yach, Stuckler and Brownell 2006).

Obesity also imposes individual and national financial burdens. There are both direct and indirect financial costs of obesity. Average annual medical expenditures are larger for obese individuals relative to those who are not obese (Finkelstein, Fiebelkorn

and Wang 2003). This figure can be expected to rise world wide because of persistent co-morbidities with obesity. In China for example, 30 percent of households in poverty cited healthcare costs as primary reason for poverty (Yach, Stuckler and Brownell 2006). Obese women are reported to be 2.5 times more likely to be unemployed, and receive 7 percent less in wages than nonobese women (Finkelstein, Ruhm and Kosa 2005). Obesity also accounts for between 5 and 7 percent of annual health care expenditures in the United States (Finkelstein, Fiebelkorn and Wang 2003). Obesity and overweight are increasingly associated with racial minorities the world over, likely due to lower socio-economic status (Kumanyika et al. 2010; McTigue, Garrett and Popkin 2002; Ogden et al. 2010). As such, much of the burden of paying for obesity falls to the public sector (Finkelstein, Ruhm and Kosa 2005).

Second, in obesity literature, more theoretical work is needed to improve the dialogue between applied global health research on the one hand and basic globalization researchers on the other hand. A project such as this one that does not squarely fall into any one discipline or area is likely to be criticized on the basis of using methodology or variables foreign to its audiences on either side. However, in global obesity literature, there are clear disconnects between public health and globalization approaches. For epidemiologists and public health advocates, the mechanisms of how globalization has influenced obesity are not clearly incorporated, and from the globalization perspective, there is trouble translating macrostructural cross-national research to the lives of individuals.

This project aims to improve this dialogue so that public health understanding can be enhanced by overarching global mechanisms where appropriate. Societies today are

densely connected through their economic, cultural, political, and social contacts. As Bozorgmher explains (2010), the term "global health" can be used to connote the de-territorialization of social determinants of health such that the pathways influencing health extend to anywhere on Earth. Put this way, global health is an outcome of overarching social determinants which act through the same mechanisms at work in "globalization." According to this conceptualization, health issues can be connected anywhere in the world through the pervasive reach of international trade agreements, worldwide governance structures, and unified logics functioning at the global – or deterritorialized – level. Conceptualizing global health in deterritorialized space allows for local contexts to matter, and at the same time permits the actions in one place to be felt in another. Obesity, then, is characteristic of the global linkages which underlie structural determinants of health. Examining a supraterritorial nature of the determinants of health occurring across the world at similar time periods enhances views from public health and globalization theory (Bozorgmher 2010).

Third, the significance of this study is enhanced from the perspective, in hindsight, that many of the proposed hypotheses do not bear their projected fruit. In a few instances, there were surprising outcomes among the results which do not negate hypothesis' value per se, but underscore the need for greater certainty in the application of obesity theory. This project supplies evidence for a more clearly applied methodological approach in global obesity. Lastly, this study underscores the importance of a comprehensive view on obesity epidemics. As these studies show, there are very real problems which overshadow obesity that are related somewhat counter intuitively. Given that the pathways leading to obesity are diverse, a wide net examining its precipitating

influences is justified. Particularly, the discrepancy in understanding obesity between obesity researchers and health practitioners and the general public underscores the need for more unified voices articulating the environmental nature of weight gain (Wickins-Drazilova and Williams 2010).

Defining Obesity

Obesity, a condition of excess body weight wherein health is adversely affected, is primarily measured by the BMI: the ratio of kilograms to meters squared. In the United States, obesity related data come from various waves of the Behavioral Risk Factor Surveillance Survey (BRFSS) and the National Health and Nutrition Examination Survey (NHANES) which make use of both interview and physical examination methodology. They began in 1960. The most comprehensive obesity data from Europe derives from the World Health Organization's (WHO) Multinational Monitoring of trends and determinants in Cardiovascular disease (MONICA) project established to assess trends in changing cardiovascular health in the early 1980s until the late 1990s across 21 countries. Subsequently, the WHO database on BMI has compiled comprehensive age and sex standardized BMI estimates across a wide sample frame from 1980 to 2010.

BMI, though, is actually not well suited to obesity diagnosis for several reasons. In general, it does not distinguish well between individual or even group variation in adiposity or fat storage tissue. Obesity in men and women is measured in the same way, however, fat is dealt with differently based on inherent biological differences – natural height, among others. Similarly, BMI does not compensate for different body types in different races[1] (Ettinger et al. 1997). Even the location of fat on the body has different

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[1] Some controversy has arisen in recent years regarding the applicability of BMI cut-off

outcomes which BMI does little to account for. Some people develop visceral fat – fat surrounding organs – that is closely associated with type II diabetes, whereas other patients are more prone to develop subcutaneous fat – fat below the epidermis – that is less prone to develop comorbidity (Livingston 2012; Montague and O'Rahilly 2000). Furthermore, BMI does not account for naturally occurring distortions in the ratio of weight to height common to the "average" person for which BMI was originally designed (Campos 2004). Critics have argued that BMI actually only accounts for 60-70 percent of the variation in individual adult body fat content (Ross 2005).

From early on in its use, BMI was recognized to be a general measure based primarily on weight, and strongly associated with direct measurements of body mass (Khosla and Lowe 1967). Other measures of adiposity have been illustrated as more precise measures of body fat, but they too are not without major limitations (Kopelman 2000). The first major limitation among anthropocentric measures of body fat is that they require trained personnel to gather. Because BMI is based on well known information of weight and height, its derivation is self-reportable, making it very widely available. Second, each of the commonly accepted means of measuring body fat tend to focus on a specific type of fat at the exclusion of other relevant types. For example, waist circumference is typically taken between the pelvis and the lower ribs in order to assess abdominal fat which, while strongly capturing central fat, is weak in measuring the visceral fat which acts as a key predictor in many outcomes of adverse health. Skinfold

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scores to all populations, notably Asian populations. According to a WHO Expert Consultation (2004), evidence suggested that cardiovascular and diabetes risk was lower among Asian populations compared to the typical "overweight" classification of 25 kilograms/m^2 or higher, but that also, no clear cut-off score was necessarily more appropriate. Thus, as they argued, WHO classifications for overweight and obese should be used for international classifications.

thickness measured by calipers is used at multiple sites for a composite score that is intended to assess subcutaneous fat, but poorly includes fat in the torso or muscles. More technologically advanced means of measuring adiposity include bio-impedance – the process of measuring electric current through body tissue. Fat and lean mass conduct electricity differently, thus measuring the speed of electric current can readily estimate the proportion of fat mass. However, based on the prohibitive cost of technology, these methods are not widely available.

Perhaps most importantly, the same problems leveled at BMI fundamentally apply to any physiological measure of body fat: variations in individual body types threaten the applicability of any one measure to the wider population. Still, unlike other measures, BMI has been shown to be reliably estimated from grouped data which is a valuable asset as an epidemiological tool (Khosla and Lowe 1967). Hence, when assuming a normal distribution of body mass, obese categories are based on standard deviations from the mean, representing a valid means of generalizability at the population level. The 95th percentile of body mass and above constitutes "obese." The concern evoked by obesity trends in the US and the world results from the fastest BMI growth among the highest 5 percent (Bleich et al. 2007). Body mass categories are listed in Table 1. In spite of its limitations, BMI remains the most epidemiologically sound, widely available and reliably measured indicator of body mass and has been used nearly exclusively since 1989 as a means of assessing obesity (Monteiro et al. 2004).

Challenges to Obesity "Theory"

One of the problems facing obesity scholarship has been the ideological traction that criticism has gained in the popular, political and academic applications of obesity research including how obesity is measured, framing it as an illness, and the implications of labels associated with its growing trends. The criticism against obesity research proposes that BMI was developed for purposes other than obesity diagnosis. BMI gained popularity as a means to assess life insurability in the 1940s based on the correlation between good health and mortality without regard to various ways "good health" can be defined (Blair and LaMonte 2006; Campos 2006; Gaesser and Blair 2002). The cutoff of 25 BMI units as "overweight," by this view, is because BMI under 25 is not clearly associated with levels of fat. Also, BMI tends to be associated with noncommunicable disease starting before "overweight" categories e.g., BMI 19-21, which is not a clear enough association. Accordingly, this perspective does not view overweight as a disease because it is merely associated with other illnesses, rather than being a health problem itself (Ross 2005). "Pro-fat" advocates claim that the anti-obesity literature overstates the health claims of thinness, and that this pre-occupation – unsupported by data in their view – overly emphasizes that long-term weight loss can and will improve population health (Campos et al. 2006a; Saguy and Riley 2005).They argue that NHANES data suggest protective mortality effects for the mildly overweight, and that overweight poses no additional health risks until class 2 or 3 obesity. By supporting a "fat but fit" approach emphasizing exercise regardless of the body type, health advocates offer a more applicable public health goal than mere thinness (McAuley and Blair 2011).

These points have called into question the legitimacy of labeling obesity an

"epidemic" by the US Surgeon General and other health organizations. The importance of the term "epidemic" is not merely technical. Contention over the term comes in three main parts which I address in turn. First, obesity "science" is really the result of problem framing in moralistic terms (Kirkland 2011). Medicalizing and pathologizing obesity justifies prejudice against the obese by blaming them for having inflicted their condition on themselves (Saguy and Riley 2005). In this way, as Campos (2006) puts it, fat people are reprehensible because they have violated our moral sensibilities about common notions of individual responsibilities for health and weight. Obesity takes on the form of cultural moralism governing rules of consumption dictated by the elite who are able to self-regulate eating and exercise (Kirkland 2011). By this view, "class is performed in bodies," because, as the folk wisdom goes, thinness acts as a good proxy for health and wealth (Guthman and DuPuis 2006). The dialogue on the nature of obesity itself is stymied by moralizing and victim blaming characteristic of a "moral panic" rather than focusing on realistic health strategies (Campos 2006; Kirkland 2011; Saguy and Riley 2005). The contention is that the term "epidemic" applies to diseases with a particular set of health outcomes, and by using this logic, obesity itself does not cause adverse health, it therefore, should not be called an "epidemic."

The second contention with the term "epidemic" comes from comparing obesity – a noncommunicable disease – to a communicable one. The total weight gain in the US and world is modest – characterized by an additional 3-7 kilograms more than the last generation (Cutler, Edwards and Shapiro 2003; Flegal 2006). However, in 1998, the National Institutes of Health lowered overweight categorization on the basis of BMI from 27 to 25 due to faster weight gain among those already obese through the 1990s, thus

pushing several million Americans to overweight status at a time (Guthman 2011). By Campos's (2004) estimation, this equivalently manufactured the obesity "epidemic" based on classification rather than any actual public health risk (Campos et al. 2006a). Because obesity carries as many health risks as underweight and is not the direct cause of illness itself, as some have argued, obesity then, is not a disease to begin with (Ross 2005).

Third, an "epidemic" also frames the political nature of responses to illness, which some have criticized. The term itself can act as a rallying point to harness social and political attention to a problem. After the declaration of the US Surgeon General in 2000, by 2002 published academic articles increased by one thousand fold (Kersh and Morone 2005). Framing obesity as a public health "emergency" justifies the intrusion of governmental influence which, according to some, may be unwelcome. By framing obesity as an epidemic, it effectively contributes to forming public opinion as a personal issue that maintains the personal prerogative to make choices about food or a public issue requiring intervention against self-destructive or unregulated environments like aggressive advertising, or high fat diets (Kersh 2009; Mitchell and McTigue 2007).

According to most researchers, these arguments have served as distractions from the real problem of rising obesity in its extreme forms. BMI distribution has recently skewed upwards doubling BMI over 30 and quadrupling BMI over 40, representing roughly 10 million persons in the United States alone who have moved from overweight to obese in recent decades (Mitchell and McTigue 2007). Since the late 1990s, middle and developing countries obesity prevalence has increased from 30 to 100 percent (Caballero 2007). Additionally, obesity has been increasing in virtually every country

gathering data (Stevens, McClain and Truesdale 2006). This type of prevalence is compatible with the standard definition of epidemic as "the occurrence in a community or region of cases of an illness, specific health-related behavior, or other health-related events clearly in excess of normal expectancy (Stedman's Medical Dictionary (2000) as cited in Mitchell and McTigue (2007))."

Epidemiological and physiological evidence supports assertions that obesity itself leads to adverse health (see Kopelman (2000) for review). The problem with criticisms of obesity and its health risks tend to wrongly assume the ultimate cause of death to be obesity rather than an associated comorbidity which when included, more accurately convey adverse health effects in illness onset, economic costs and lower quality of life (Rigby 2006). More importantly, the logic of the criticism and its respective public health goals are somewhat disingenuous. As Campos et al. (2006) argue, the goal in reducing obesity should be a healthy lifestyle promoting exercise rather than thinness. The central claim – that exercise improves overall health and longevity, and that a healthy lifestyle is a more sustainable goal than thinness – forgets the embedded relationship that a more healthy lifestyle will naturally lower body mass since the change is regular, consistent exercise and better diet. The point of improving health through lifestyle change is moot given that the outcome of consistent exercise for the average person is weight loss (Stevens, McClain and Truesdale 2006). In one commentary, Kim and Popkin (2006) summarize detractors as "nullifying existing scientific evidence based on discriminatory use and fallacious interpretation of literature (pg. 61)."

The public and political response, largely fueled by the "epidemic" framing debate has distilled to essentially one of two views that trace roots to political persuasion (Kersh

2009; Niederdeppe, Shapiro and Porticella 2011). In one view, obesity is an individual problem represented by the right to make personal choices about lifestyle and the right of food suppliers to fill a profitable niche market. In the second view, obesity is a condition of toxic food environments which exploits vulnerabilities surrounding lifestyle constraints largely reinforced through the logic of capitalism (Guthman 2011). The future of obesity politics, predictably, is stymied by political stalemates much like those that have plagued the regulation on tobacco products (Brownell and Warner 2009; Kersh and Morone 2005). The point of the above discussion is to illustrate how obesity "theory" is disputed on several grounds. Further, such opposition is primarily held by nonpractioners and the lay public. Further theoretical refinement for developing the progression of obesity is clearly needed to move past unproductive views of obesity in the academic and political arena. One aim of this project is to create space for researchers with different foci on obesity to come together on common theoretical and empirical ground. I show in Chapter 1 how the patterns of global obesity can be substantively improved through comparing contemporary explanations of obesity. As Brownell (2005) indicates, this is a fruitful endeavor to pursue. She argues that public opinion reflects some dissonance in obesity attributions – people tend to attribute obesity to personal responsibility as a personal level illness, but also view the importance of some social measures to prevent its increase (Barry et al. 2009; Niederdeppe, Shapiro and Porticella 2011; Suggs and McIntyre 2011; Wickins-Drazilova and Williams 2010). This project is intended to highlight, at least partially, that there is greater nuance in an individual responsibility attribution.

Still, theory and policy supporting the advent of obesity as socially determined are

slowly being implemented, including notable examples such as the "Cheeseburger Bill" and more state-level initiatives to ban transfats across the US states with varying levels of success (Boehmer et al. 2008; Dietz, Benken and Hunter 2009; Seiders and Petty 2004). The framework of public legislation to curb unhealthy food product availability and marketing has also been recommended by the WHO to its member states (Chopra 2002; Lobstein 2010). Judiciary action rather than legislative action though has become more effective in enforcing anti-obesity initiatives (Kersh 2009). As I will discuss in Chapter 2, confronting obesity through the lens of social justice is being integrated into anti-obesity policy and research strategies. In accepting prosocial policy scripts through the actions of civil society, advocacy groups demonstrably show improvement in body mass and serves to offer substantial means of improving obesity outcomes.

Causes of the Global Obesity Epidemic

One the main sources of the problems in obesity measurement, framing, and definition is the wide array of overlapping causes of obesity which include reductive and expansive explanations. This problem is accentuated when attempting to compare obesogenic environments across diverse countries. A few ways to do that have been proposed. In this section I review the presumed causes of the obesity epidemic relevant to the current global experience. There are of course obesogenic influences involved at every level of social organization. The review here is limited to the mechanisms of dominant theoretical paradigms used to explain the global epidemic.

One of the first places to look for responsibility for obesity is genetic and metabolic processes. These explanations have been used on two levels. First, in

individuals, obesity and overweight run in families and has been linked to "candidate genes" which predispose individuals to the synthesis of particular peptide chains associated with particular illnesses (Kopelman 2000). One of those genes in obesity literature is referred to as the "Thrifty" genotype or insulin-resistance genes which essentially stores glycogen – cellular energy – in lush times as fat for energy in lean times (Kolterman et al. 1980). This process, which was supposedly adaptive to primitive humans living in calorically restricted environments, has become a liability due to caloric excess today (Lieberman 2003). Second, pregnant women in nutritionally poor environments tend to give birth to babies who are metabolically programmed to retain fats (Ely, Zavakis and Wilson 2011). Referred to as the thrifty phenotype hypothesis or "shantytown syndrome" (Critser 2003), this metabolic imprinting leads to obesity in two ways: infants and children become obese because of the "thrifty gene" at work, and/or malnourished fetuses have been associated with an underdeveloped pancreas associated with glucose intolerance (Uusitalo, Pietinen and Puska 2002; Wang, Monteiro and Popkin 2002). In these circumstances people are accustomed to nutrient impoverishment, but with caloric abundance, their metabolic functioning is poorly equipped to adequately handle excess calories. A significant problem among genetic explanations is that the rise of obesity has outpaced genetics as playing the sole role (Lieberman 2003). More epi-genetic explanations, however, are plausible as the social environment has rapidly changed since the obesity epidemic onset (Swinburn et al. 2011).

The role of economic development is also closely associated with obesity. Urbanization associated with economic development is among one of the strongest predictors of obesity onset internationally due to a number of factors (Loureiro and

Nayga 2005; Popkin 1999; Popkin 2001a; Popkin 2001b; Popkin and Gordon-Larsen 2004). First, cities are also sites where economies shift away from primary sector production towards more service and professional sector employment thus concentrating employment, income and reducing physical activity. A career in a sedentary job contributes to as much as 3.3 BMI units more than a highly active job (Lakdwalla and Philipson 2002). Among poor neighborhoods, lack of access to fresh foods is characterized as a "food desert" wherein food distributors market foods which are most profitable (Chopra 2002; Cummins and Macintyre 2006). Development has also reduced the amount of caloric expenditure in the tasks of daily living. Among one sample, it was estimated that as much as 60 percent of the rise in obesity could be explained by increased productivity in the home afforded by technology (Lakdwalla and Philipson 2002). Increases in income have been related to purchasing prepared food away from home among women (Finkelstein, Ruhm and Kosa 2005).

Second, economic development has facilitated the increased production of calories available on the market. Because cities serve as contact points for international trade (Sassen 2001), calories from the world market which tend to be higher in fat and sugar are more widely available. Foreign Direct Investment (FDI) – investment by one firm from a country other than that of the recipient company – has increased the proportion of highly processed foods for sale (Hawkes 2005). Proportionately, food imports have increased in lower developed countries, accompanied by increases in consumption (Rayner et al. 2006). Global brands such as Coca Cola and McDonald's restaurants have increased sales through competitive media campaigns that attempt to change traditional eating habits in local contexts by tailoring a "glocal" marketing

strategy such as images of sophistication and modernity (Hawkes 2002). Frito-lay and Pepsi, for example, have become the most successful snack products among Thai youth primarily because of the success of local affiliate brands (Hawkes 2006). Malaysia is among the highest consumers of sugars and sweeteners – between the US and Australia – due to consumption of sweetened drinks (Khor 2012). Television and other sedentary hobbies also play a role, particularly when viewed through the lens of aggressive advertising and snacking (Gable and Lutz 2000; Lobstein 2010). Some estimates have linked increases in food advertising exposure among obese and overweight children compared to normal weight children (Halford and Boyland 2011). The Uruguay Round of the General Agreement on Tariffs and Trade in 1994 and subsequently the World Trade Organization have facilitated changes in trade policy that have increased the food supply by lowering food trade barriers for processed food, meats, and increasing wider food availability (Chopra 2002; Thow and Hawkes 2009). One consequence of the rise of global food trade and marketing has been a global dietary convergence particularly from the increase in vegetable oils, animal products, and sweeteners from fast food production and normal household use (Kearney 2010; Popkin and Gordon-Larsen 2004). The expansion of the food supply, brought on by innovation in agricultural production, FDI, and marketing, has been associated with .7 BMI units, roughly corresponding to 40 percent of weight gain in one study (Lakdwalla and Philipson 2002).

Methodological Approach

In two self-supporting chapters, I introduce the background and methods used to examine existing empirical approaches to global obesity. The challenge of measuring the

mechanisms of obesity is that virtually all of them have dramatically changed over time. Cross-sectional data are not suited to examine long-term obesity trends because of their failure to capture how time impacts moving targets like nations and people (Drewnowski 2007). Longitudinal analysis makes use of controls that expose how the underlying changes have been influential over time. I employ repeated measures data on both wealthy and low-income countries from 1980 – 2008 using fixed and random effects modeling techniques.

The main benefit in using fixed and random effects modeling for cross-national obesity is how these models treat unobserved variation between cases that can be partialled out (Allison 2009). Fixed effects estimation uses inherent model controls to reduce bias in two ways. First, the fixed effects assumption allows unobserved variables to vary at random with model predictors, and in so doing, data are assumed to correlate across time, known as heterogeneity bias. Second, change between time periods control for this bias. Constant fixed parameters are differenced out of the model which reduces heterogeneity bias. Functionally, this assumption "discards" between-case variation in order to reduce model biases (Alderson and Nielsen 1999; Allison 2009). Random effects modeling assumes unobserved variables to be uncorrelated with model predictors. This allows for unobserved variables to be uncorrelated over time which introduces bias when unobserved variables are actually associated with model predictors. However, in order to control for this bias, random effects modeling uses both within- and between-case variation to improve the efficiency of model estimation. These methods are well suited for macrolevel analyses because of their ability to reduce omitted variable bias, as in the case of the fixed effects model, or take advantage of within- and between-case variation

to improve estimation when data are restricted as in the random effects model.

In Chapter 1, I use random effects techniques to test two theoretical paradigms that emphasize opposing views of economic development and caloric intake as an explanation of global obesity trends. Random effects are best suited to this question because of the very small sample frame available in using key predictors to test each theory. For example, diet structure and nutritional composition in food are commonly associated with economic development, which has increased health around the world. Referred to as the nutrition transition, when the level of economic development increases, the higher purchasing power afforded by economic success enables wider degree of calories and food access, thus contributing to higher body mass. The obesity epidemic is presumed to be best explained by this framework. However, obesity is not simply a function of economic prosperity as once thought. Both poor and wealthy countries alike have shown elevated obesity risks (Drewnowski and Specter 2004; Sassi et al. 2009). This creates two problems for the nutrition transition perspective. First, urbanization has been identified as a strong predictor of national obesity, but urbanization growth in many nations, particularly those facing rapid obesity onset, is driven by urban slums where food insecurity and malnutrition is common (UNHABITAT 2010). Second, growth stunting exists among children who are calorie and nutritionally deficient, particularly among the urban poor. The nutrition transition does not sufficiently account for malnourishment in diet transition because it assumes access to calories is increasing for all population segments. Instead, malnourishment may represent higher obesity risk, and, if true, this would paint the portrait of obesity to be more closely aligned with caloric deprivation rather than caloric abundance. Studies focusing on international obesity have

not yet examined competing obesogenic pathways as articulated in this project. In Chapter 1 I ask, how do the conceptual pathways of obesity incorporate emerging trends of obesity among the poor? Both perspectives emphasize caloric access due to economic development but they differ in explaining the rapid rise of obesity among poor countries. Using three different outcome measures – obesity prevalence, average BMI and total food consumption – I test the role of urban poverty and malnutrition to evaluate which presumed obesogenic route best explains emerging body mass trends among poor countries. I show that obesity is not simply a matter of overconsumption, or even necessarily driven by calorie dense foods. Instead, higher obesity prevalence, in many poor countries, is the result of the compounded problem of childhood malnutrition rather than mere over consumption.

In Chapter 2, I apply fixed effects modeling to test the mechanisms of two broad theories of global convergence in the role of rising body mass. The logic of global convergence argues that pressures from a variety of sources have contributed to consistent and intersecting cross-national adaptations regardless of local contexts. The global obesity epidemic is argued to be a manifestation of economic and cultural homogenization. Obesity, however, provides a unique way to view these pressures as there may be various ways such homogenization may occur. In the recent past, global economies have grown increasingly integrated under the assumption that free moving capital and goods are beneficial for states and people (Arrighi 2007). The expansion of food trade has operated on a similar assumption, and as such, has proliferated the expansion of foreign investment in food processing and agricultural output (Hawkes 2005). With rapid and expansive upward trends in FDI, research has identified the global

obesity epidemic resulting from the sudden caloric availability from calorically dense, nutritionally unhealthy foods endemic in Western diets (Iqbal et al. 2008). From this view, the rising trends of investment in the constant search of profitable markets suggest that the obesity epidemic will continue unabated. On the other hand, the rise of civil society advocacy groups acting apart from states and markets have become the medium of choice to confront many social ills (Reimann 2006). These groups also exert pressure on states to take policy action such as improving public health education, calorie information requirements or providing "watchdog" functions on behalf of the underserved. A key element of this literature examines how framing health as a human right has become a powerful tool in motivating state action against scientifically demonstrable social problems such as obesity (London and Schneider 2012). Obesity provides a way to view the relative contributions of two different sets of converging forces because of the direction of their influence. Economic integration presumably contributes to higher body mass through its key mechanisms, but civil society serves to lower obesity through its respective mechanisms. Both of these globalization perspectives are potentially relevant to identifying converging obesity epidemics. Still, using BMI as an outcome measure allows a view of their presumptive competing contributions. In Chapter 2, I examine how economic inputs compete with or are complementary to civil society influences on BMI. Furthermore, comparing two globalization theories contributes to breaking new theoretical ground by applying them to a novel outcome. I compare the influence of global integration through FDI, value-added agricultural production and agricultural trade openness to those of Obesity oriented International Nongovernmental Organization (OINGOs) membership, and Obesity oriented

Intergovernmental Organization (OIGOs) membership. Findings from this study are used to argue that obesity should not necessarily be viewed as the product of wider economic integration among emerging economies but that, contrary to prior global obesity literature, the effects of investment suppress body mass growth. Furthermore, civil society is most effective when administered through suprastate agencies such as the United Nations, which are likely to place direct pressure for change on sovereign state governments. In appendices to this chapter, I address methodological challenges confronting studies which attempt to separate many of the interrelated concepts of economic convergence.

Conclusion

This dissertation project concentrates on exposing the mechanisms of the "supply" of obesity to poor countries. As Swinburn et al. (2011) describe

> Obesity is the result of people responding normally to the obesogenic environments that they find themselves in, so too do these obesogenic environments arise because businesses and governments are responding normally to the broader economic and political environments that they find themselves in. (p. 810)

In the following analyses, I show how the process of globalization has produced both macro- and microlevel consequences. Essentially, I elucidate how structural convergence in the global system has influenced individual bodies. In the first portion of my research, I expose how the nutrition transitions among developing nations expose existing vulnerabilities among the urban poor. Second, I cast a wider net on the influences of obesity and show how convergence in the global system functions in poor regions of the world. At present, much of the research on global obesity has been descriptive, lacking a

theoretically driven, inductive approach regarding its pathways. In this project, I use

theoretically informed answers to enrich our understanding of an intractable and

expanding individual problem for millions around the world.

Table 1
Body Mass Index Categories
and Weight Status

BMI	Weight Status
Below 18.5	Underweight
18.5-24.9	Normal
25-29.9	Overweight (pre-obese)
30-34.9	Obese (moderately/class 1)
35-39.9	Obese (severely/class 2)
Above 40	Obese (extremely/class 3)

CHAPTER 1

PRECURSORS TO OBESITY: EVALUATING THE ROLE OF POVERTY,

FOOD SUPPLY, AND ECONOMIC DEVELOPMENT IN

THE GLOBAL OBESITY EPIDEMIC

Introduction

According to the World Health Organization (2000), obesity is known as a condition of abnormal or excessive fat accumulation in adipose tissue, to the extent that health may be impaired. In 2008, 1.4 billion adults were overweight and 65 percent of the world's population lives in countries where being overweight kills more people than being underweight (WHO 2012). The global obesity epidemic exists among a diverse group of countries but most notably among middle- and lower-income countries. While the absolute number of obese and overweight was greater among upper-middle and high-income countries, the relative growth of overweight prevalence was greater among lower-middle and low-income countries (WHO 2011).

Obesity has, over the last 20 years, become a problem worldwide. In every region of the world, excess weight prevalence is growing. International trend data suggest prevalence increases in many developed and developing nations (Wang, Monteiro and Popkin 2002). In Brazil, obesity prevalence has increased by .5 percent per year from

1975 to 1997, comparable to that of the United States growth of .6 percent over a similar time period. Obesity in China has increased at .2 percent per year between 1991 and 1997. In 2002, data collected from the National Nutrition and Health Survey indicated Chinese obesity is disproportionately an urban problem: urban obesity prevalence was reported to be 25 percent compared to 12.8 percent rural prevalence (Parizkova et al. 2007). According to a WHO (2000) report, in urban Samoa, obesity prevalence was reported to be 75 and 60 percent among women and men, respectively, and in 1990, 44 percent of women living in the South African cape peninsula were considered obese. In Mauritius, obesity among women grew from 10.4 to 15.2 percent from 1987 to 1992. Even among South East Asia where obesity prevalence data are poor, urban overweight has been recorded in 1991 to be 23.2 percent of women and 15.2 percent of men. Unfortunately, comparable data on obesity are relatively scarce. While data exists for numerous countries over time, most surveys do not capture nationally representative samples among comparable ages or populations. Table 2 shows obesity prevalence among select countries (Lobstein and Leach 2006).

Figure 1 shows mean obesity prevalence among six geographic regions from 1980 to 2009. Obesity prevalence data are not widespread, and as such, this figure could be somewhat misleading. For example, countries of the Pacific Islands show the highest obesity prevalence, on the order of 78.5 percent of people in Nauru, but because comparable population data are not common, obesity appears to have abated there in the late 1990s, only to resurface a decade later. This is due of course to relatively few corresponding data points among Oceanic countries. The same effect is present among other regions, but to a lesser extent. The trend, however, is an increase in obesity among

all reported regions. A better view of the increasing trend of obesity can be seen in Figure 2: Average Body Mass by region from 1980 to 2007. More contiguous data indicate a rise in body mass among all geographic regions. The international standard cutoff for obesity based on body mass index is 30.0, overweight at 25. BMI as a measure of obesity has been criticized for its difficulty accounting for individual variation in human physique, gender and, ethnic differences (Gard and Wright 2005). Though some skepticism should be used in interpreting these data, BMI is the most widely available and widely used indicator of obesity. Few regions could be characterized as purely "obese" based on this cutoff, but the trend is clear – BMI has increased among all reported regions. Flegal (2006) shows that in the United States, change in BMI from 26.8 to 27.9 from 1998 to 2002 represents an additional 7 kilograms for the average person.

I argue that while the obesity threat is growing to incorporate more economically diverse nations, greater clarity is needed to explain its pathways. As indicated, national level obesity trends have increased among a wide variety of socially, economically and geographically diverse nations. Evidence from wealthy countries with long-standing obesity trends reflect the highest obesity risk among the urban poor as do lower-income countries (Drewnowski and Specter 2004; Sassi et al. 2009), suggesting that obesity risk is not simply a function of a nation's prosperity. Instead, the urban poor may be at the greatest risk for obesity, which, if true, would help explain growing trends in international obesity.

National studies of obesity have uncovered substantial commonality in the precipitating causes of obesity in urbanization, rising incomes, and sedentary lifestyles; however, no studies have yet engaged this global problem using a global unit of analysis.

As indicated by Figure 2, the pace of BMI increase is not uniform. Applying a cross-national lens first allows the use of more data, where data are already limited, and also allows intermediate levels of analysis to uncover the relative pacing of obesity among different regions of the world. National level studies are limited in showing how the global obesity problem is actually global.

In this paper, I examine how economic development, urbanization, and urban poverty are empirically related to two theories of obesity risk– the nutrition transition and food security perspectives. Using cross-national longitudinal data from 1990 to 2007, I test influence of country-level poverty and malnutrition trends on national obesity prevalence during that 17 year time period. I assess differences in urban slums and per capita income across 3 macrogeographic regions, where data permit, and I also test the influence of poverty on food consumption to demonstrate the relative importance of food consumption compared to economic status. The bulk of international obesity theory is relatively new, and this paper will contribute to both the theoretical and empirical literature related to global obesity trends.

Background

Economic development arguably acts as the most important factor in obesity trends across the world. There are two aspects of economic development, in particular, that may be associated with higher population-level risk for obesity. First, urbanization, which generally occurs alongside economic development, has been associated with higher average BMI (Ely, Zavakis and Wilson 2011; Kumanyika 2008). Countries with higher agricultural economic concentration have lower measured BMIs, perhaps due to

lower energy expenditure in labor (Loureiro and Nayga 2005). Urbanization has also been associated with rapid transition to energy dense diets, lower physical activity, and lower workforce energy expenditure (Loureiro and Nayga 2005; Popkin 2001b; Popkin and Gordon-Larsen 2004; Popkin and Doak 1998). It is estimated that sedentary jobs can confer 3.3 units of BMI than someone in a more active job (Lakdwalla and Philipson 2002). Second, as countries develop, the work force often becomes more skilled, thus typically adopting a more sedentary, less active lifestyle (Swinburn et al. 2011). However, Bleich et al. (2007) argue that workplace energy expenditure declined prior to the global increase in obesity trends, indicating that obesity risk is not directly a result of changes in urbanization or workforce behavior. Given differential trends in obesity within and across countries by social class (Drewnowski 2003), it is possible that obesity risk may also be a function of the differential opportunities afforded to certain subgroups of the population. Some evidence suggests that, for example, a high-skilled labor force is associated with an inverse income-obesity gradient in higher-income countries (i.e., per capita GNP of $2500; Popkin and Gordon-Larsen 2004), whereas poverty lowers obesity risk in the lower-income countries. In other words, obesity risk appears to be a function of economic status, across and within countries, as well as the level of urbanization or overall development of the country. Thus, the nuanced relationship between economic development of a country and the obesity risk among its population warrants further exploration.

The nutrition transition theory of obesity – the predominant paradigm of the global obesity epidemic – refers to widespread changes in diet and nutrition that occur as a country enters various stages of economic development (Popkin 2001a). As countries

enter stages of industrial development, food becomes more accessible which generally leads to greater food security and ultimately a greater obesity risk (Popkin 2006). For example, national diets may move from one consisting of a few starchy vegetables to a higher consumption of fruits and vegetables, to finally, a consumption pattern high in animal products, sugars and processed foods (Popkin 2006). According to this framework, the current obesity epidemic occurring throughout the developed and developing world may be explained by major shifts in diet and nutrition that accompany increases in disposable income, and lower level physical activity that are associated with economic development. Accordingly, obesity is propelled through urban areas as sites of higher income and lower physical activity conferred from sedentary high-skilled labor and increase access to energy dense foods processed domestically and imported from abroad (Popkin 2006). In essence, food insecure nations move towards food security where obesity risks are more likely. A rising caloric tide is presumed to lift the body weights of all persons through wider food access. Many have argued that rising obesity rates in developing countries such as China, Brazil, Egypt, Mexico, South Africa, and Thailand are the results of wider global and regional economic integration, greater caloric access from domestic and foreign producers and a shift toward high-skilled labor (Popkin and Gordon-Larsen 2004). For example, several concepts consistent with this reasoning have included agricultural productivity, global agribusiness connectivity, foreign direct investment, urbanization, government support to agriculture, agricultural trade liberalization, female labor force participation and even nationalist ideology as a check of resistance to food colonization by Western fast food restaurants (Hawkes 2006; Lobstein 2010; Loureiro and Nayga 2005; Miller and Coble 2008; Rayner et al. 2006; Thow and

Hawkes 2009; Varman and Belk 2009). These types of indicators are aimed at assessing the relative influence of obesity inputs from abroad in raising overall caloric availability, especially as they relate to unhealthy calories.

Obesity risk has been shown to be highest in urban areas of developing nations, since urban areas are sites of higher income and lower physical activity conferred from sedentary, high-skilled labor in those areas (Drewnowski and Popkin 1997; Popkin 1999; Popkin and Doak 1998). Also, access to energy dense, processed foods from abroad are higher among those living in urban areas given the concentration of international cultural and economic imports in cities (Sassen 2001). Although the nutrition transition that typically accompanies economic development allows a country to transition from a food-insecure to a food-secure environment, the speed at which these changes occur increase the risk of obesity among malnourished and growth stunted children. As suggested, these issues may be particularly prominent among the urban poor.

Until relatively recently, research on world hunger has concentrated on food availability – the availability or scarcity of appropriate amounts of food. However, research on African famines during the 1980s shifted attention from unavailability to inaccessibility, or the political and economic rights to obtain food (FAO 2006; Jenkins and Scanlan 2001; Jenkins, Scanlan and Peterson 2007; Shipton 1990). Following this emphasis, food security research has concentrated primarily on chronic hunger as a function of social, political and economic inequity rather than availability. While food supply has increased globally (Caballero 2007; Dietel 2002), access continues to present challenges among the poor. Malnutrition – being poorly or wrongly fed – was estimated to affect 925 million people in 2010, comprising 16 percent of Least Developed Country

(LDC) populations (Ruane and Sonnino 2011). According to the United Nations Millennium Development Goals report (2012), world hunger has improved, but chronic malnutrition is still widespread, especially among urban slums (Fry, Cousins and Olivola 2002; UNHABITAT 2010).

Poor nutrition can influence BMI in different ways particularly when introducing food excess to chronic hunger environments. First, a common symptom of chronic under-nutrition is childhood growth stunting. Hackett et al. (2009) found that stunting and underweight increased proportionately as food insecurity persisted. They found that among Colombian children, food insecurity increased risk of stunting by 200 percent relative to food secure children. Recent evidence suggests that growth stunted children are much more likely to become obese in later stages of adolescence and adulthood than their linear growth counterparts (Walker, Chang and Powell 2007; Wang, Monteiro and Popkin 2002). Research literature has described consistent associations between childhood malnutrition, growth stunting and obesity among a variety of countries across the socioeconomic spectrum (Doak et al. 2002; Hackett, Melgar-Quiñonez and Álvarez 2009; Popkin 2003; Tanumihardjo et al. 2007). Stunted children have also been shown to increase obesity risk after being introduced to nutrient rich diets (Wang and Lobstein 2006). Therefore persons exposed to high-poverty, high food-insecure environments as children may be particularly susceptible to obesity when later exposed to a more food-secure environment.

Second, while counter-intuitive, over-nutrition can be comorbid with malnutrition in that calories required for energy requirements are met through basic macronutrients – proteins, fats and carbohydrates – but the diet lacks micronutrients such as iron or folic

acid required for healthy development. This has been labeled "hidden hunger" in that a person may be overweight but still be malnourished because they ingest large quantities of bulk energy without sufficient amounts of essential nutrients, thus resulting in an apparent obesity paradox (Tanumihardjo et al. 2007). In Brazil, China, and Mexico nutritional excesses among mothers and older siblings have been reported to co-exist with undernourished children and younger siblings exemplifying the existence of hidden hunger among persons living in an otherwise food-secure environment (Doak et al. 2002; Wang, Monteiro and Popkin 2002). Hence the transition from food scarcity to abundance can lead to higher body mass in two ways: first, insufficient vertical growth contributes to larger weight for height when food scarcity improves, and second, by ingesting excessive amounts of newly available, nutritionally poor and energy dense foods consisting of fewer essential nutrients.

Consistent with this line of reasoning, studies have typically assessed the trends of growth stunted and malnourished children in developing countries while fewer studies have attempted studies of causal pathways of malnutrition and obesity (Walker, Chang and Powell 2007; Wang 2001; Wang, Monteiro and Popkin 2002). Among studies that have linked malnourishment and stunting with obesity, covariates have included measures of out of home eating, economic vulnerability, and political equality, but most commonly, food availability from the food balance sheets of the Food and Agriculture Organization of the United Nations (FAO) (Bezerra and Sichieri 2009; Bleich, Blendon and Adams 2007; Brown and Gershoff 1989; Drewnowski and Popkin 1997; Jenkins and Scanlan 2001; Jenkins, Scanlan and Peterson 2007; Khor 2012; Silventoinen et al. 2004; Thow and Hawkes 2009). These types of variables are posited to be linked to obesity and

higher than average BMI through malnutrition related outcomes. Other approaches have examined the relative influence on obesity from price indexes of specific types of foods like soft drinks, and fast foods, typically high in sweeteners and fat content (Drewnowski 2003; Hawkes 2008; Thow and Hawkes 2009).

Research has confirmed inverse associations between food prices and weight gain (Chou, Grossman and Saffer 2004; French 2003; French et al. 2001), thus global food prices are another major contributor to food access and nutrition in developing countries. June 2008 food prices reached their highest levels in 30 years and reportedly pushed 115 million people into chronic hunger (FAO 2009). The main drivers for high and volatile food prices have been traced to new biofuel demands which displace food for consumption and record oil prices that increase costs for fertilization and transportation (Allen and Wilson 2008; FAO 2009). Since the zenith in 2008, food prices have declined but are still relatively higher than historical standards (FAO 2011). Cheap foods are mostly calorie dense foods which are also deficient in essential macronutrients that may contribute to obesity and hidden hunger (Cummins and Macintyre 2006; Doak et al. 2004; Popkin 2006; Tanumihardjo et al. 2007). Finkelstein et al. (2005) show that prices for dense foods in the US increased at a slower rate than fruits and vegetables, whereas prices for carbonated beverages grew at 20 percent per year compared to fresh fruits at 118 percent, from 1980 to 2000.

Given the trends of food pricing, hidden hunger and obesity are increasingly growing more associated with poverty. The poor most often rely on inexpensive, calorie dense, and nutritionally deficient foods for their calories (Guthman 2011). Evidence in recent scholarship indicates that the poor in emerging and developing countries are

currently experiencing the most rapidly growing obesity epidemics (WHO 2000). There are two theoretical pathways explaining obesity risk among the poor. First, the nutrition transition view, suggested by Popkin and colleagues, conceptualizes obesity to be the result of cheaper, higher calories available to everyone by virtue of increasing ability to purchase them. This suggests that the entire population would be at higher risk of obesity, but more so among lower-income segments of the population because of their reliance on inexpensive foods. On the other hand, the food security hypothesis attempts to place chronic poverty as the precursor to obesity. That is, the poorest segment of the population may have been exposed to undernourishment, malnourishment, and stunting during earlier phases of the life course (Walker, Chang and Powell 2007). Then, when the new calorie dense foods become more widely available, they are at particular risk for obesity (Uusitalo, Pietinen and Puska 2002).

At issue here are the different roles regarding caloric availability and economic growth. In both approaches to obesity theory, countries undergoing nutrition transitions because of economic development improve their caloric availability. As recent scholarship has indicated, the poor in emerging and developed nations are currently experiencing the most rapid obesity epidemics due to the increasing availability of food supply (Drewnowski and Specter 2004; Kearney 2010). The primary question being addressed in this paper is how do the conceptual pathways of obesity incorporate emerging trends of obesity among poverty. The theories presented above focus on two different pathways as they explain the routes of economic development and obesity risk.

Figure 3 diagrams different hypothesized routes to obesity as outlined by these two theoretical perspectives. Both emphasize the important role of wider caloric access and

dense calories as a result of economic development but they also differ in how they explain obesity's association with the poor. On the left, food security emphasizes the pathway from poverty to obesity passing through greater food access and malnutrition. Those who experience chronic hunger more often are more likely to consume high energy calories because of their relative cost compared to more nutrient rich foods. Malnutrition predispose the poor to obesity because of early life growth stunting. On the left, the nutrition transition emphasizes different pathways. Similar to food security, economic development creates the conditions for relative economic growth and prosperity enabling greater access to wider array of foods. Because of the overall higher economic prosperity and food availability, overconsumption is common, particularly calorie dense foods which lead to obesity. This analysis attempts reconcile how poverty commonly associated with obesity in wealthy countries, is also explained by predominant approaches in international obesity.

In sum, the rapid pace of economic development that is occurring throughout the developing world is creating a transition from under-nourishment to over-nourishment among a historically food insecure population in a relatively short amount of time. This has led to the onset of a global obesity epidemic. There is considerable empirical consensus of the association between poverty and obesity status at the individual level, particularly among wealthy or developed nations (Cummins and Macintyre 2006; McTigue, Garrett and Popkin 2002; Ogden et al. 2010). However, less research has been conducted on wider macrolevel comparisons, showing how the poverty and economic development of a country over time may be associated with the population's risk for obesity. Doing so would be useful for generalization among countries yet to undergo

obesity epidemics of their own.

I test two theoretical paradigms which both use economic development and caloric intake as an explanation of global obesity trends: nutrition transition and food security. I use urban slum prevalence as a measure of relative poverty because of the unique position it has in exposing emerging trends in global obesity. First, a key indicator of emerging obesity is urbanization. In cities, people have greater access to globally produced foods characterized by low fibre, high energy, processed ingredients. Second, most of the urbanization occurring worldwide is due to the growth in urban slums. In relative terms, urban slum populations in the developing world have decreased from 39 to 32 percent from 2000 to 2010, but in absolute terms, urban slum dwellings have grown based on population growth alone, including an additional 6 million people per year over the same time period (UNHABITAT 2010). It is expected that urban slum populations will reach 889 million by 2020. This growth is primarily concentrated in developing countries posited to be showing the most acute obesity risk in the future. Third, the association between poverty and obesity is growing in wealthy countries. For those on a limited income, often there is a tradeoff between the cost of food and the quality of food where low calorie foods are less expensive. The chronic poor living in urban slums may face these dilemmas regularly and this measure captures this relationship of the nutrition versus cost tradeoff in a calorie dense environment. Fourth, using an absolute measure of poverty would not respect the cross-national differences in food access but would essentially assume the world's poor face similar nutritional availability. By using a relative measure of urban poverty, I attempt to control for the uniqueness of urban slums in each country in order to compare urban poverty cross-nationally. Because so much of

absolute poverty occurs in rural areas without exposure to newly available, calorie dense, low cost, internationally produced, traded, and manufactured foods, much the effect of absolute poverty on obesity, if any, would be lost because of this inaccessibility.

Contrariwise, a food security perspective would argue that urban slums set the criterion for increasing obesity as those in a food-insecure environment would be more likely to purchase relatively more cheap foods. Accordingly we might also expect to see the influence of urban slums increase obesity prevalence. Essentially, the type of foods poor people eat, if its an accurate predictor of obesity prevalence, should increase the effect of urban poverty. Also, the logic of food security suggests that obesity among the poor is facilitated by stunted children as a result of food insecurity. I hypothesize the influence of childhood stunting will significantly and positively predict obesity prevalence. By definition, stunted growth increases BMI due to greater weight gain relative to slower height growth over the course of development. Practically speaking, a BMI measure as the ratio of weight to squared height will naturally increase BMIs in countries with higher proportions of stunting. In this view, childhood stunting should increase obesity prevalence strictly on the basis of how obesity is measured. Nonetheless, the effect of childhood stunting on obesity should also be influenced by the types of foods people eat. Should the type of calories increase the effect of childhood stunting on obesity prevalence, then it is the types of calories one consumes that predisposes one to obesity, as food security predicts. We thus have two hypotheses based on parallel obesogenic trajectories. Consistent with the food security hypothesis, urban slums should contribute positively to obesity, while a negative or null influence is consistent with the logic of nutrition transition theory.

Methods

To test these hypotheses, I first use obesity prevalence as a dependent variable, as estimated by the World Health Organization (2011). Obesity prevalence is the most direct measure available, however, sample sizes are restricted on this variable. Secondly, as a supplement, I use BMI as a dependent variable to compare with the influences on obesity prevalence also available from the WHO (2011)[2]. Unfortunately, obesity prevalence and BMI as choices of dependent variables is biased towards the nutrition transition perspective. As nutrition transition inherently projects, the eventual outcome of economic development is greater body weight for everyone. By using two complimentary but different dependent variables, I expect results to be similar but not identical.

However, a major influence on obesity is whether or not food access is actually increasing among all population strata. I test the relative influence of calorie dense foods on poverty as predictor of obesity prevalence and BMI. By using both obesity prevalence and BMI as outcome measures, I also include an inherent test of hidden hunger. Obesity prevalence and average BMI are different methods of measuring obesity. Obesity prevalence indicates the percentage of persons ranking in and above the 95th percentile of

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[2]   Comparing the results of analyses run on separate dependent variables is not an ideal means of testing these hypotheses without the following considerations. Obesity prevalence is not widely available, and in contrasting these analyses with BMI, another commonly used variable in obesity literature, represents a means of overcoming substantial data limitations, albeit one not without its challenges. Countries with higher BMI will naturally have a higher obesity prevalence, though not necessarily vice versa. For example, among African and Asian countries, BMI and obesity prevalence are well matched with correlations higher than 0.93 and among Latin American countries, they are correlated at .733. Though they are not identical, they are well suited as proxies. Using average body mass should provide a means of testing broad elements of each theory if, for example, results indicate a positive or negative influence of urban slums across both variables. Hence, in using these two dependent variables, we are looking for comparable findings rather than substantively contrasting ones.

body mass different from the average body mass of people in a given country. Thus a positive influence on obesity prevalence paired with a null or negative influence on BMI indicates that obesity risk exposure is greater for certain segments of the population than the national average and that the average nutrition is not improving for everyone. Furthermore, using childhood stunting as a measure of nutritional availability should indicate the effects of malnourishment on weight gain if any.

A crucial test of international obesity theory includes examining the generalizability of the link between poverty and obesity. Nutrition transition asserts that countries experiencing transformations of food supply and access should indicate no statistical influences from urban slums, as all segments of the population should have access to more calories. Including a regional test is beneficial for several reasons. Few direct comparisons between regions exist in global obesity literature and doing so would add to understanding how the obesity epidemic is progressing in a truly global sense. By examining the effects of poverty on obesity, I am illuminating the process of nutritional transition itself. If nutrition transition is, unlike economic development, occurring uniformly then there should be little differences between regions. Comparing regions may provide insight into the relative progress of regions nutritionally.

Thus for the second series of analyses, I examine the relative influence of urban slums and childhood stunting cross-regionally through the use of slope-dummy variables. One method to do this would be testing models on cases within individual regions, but doing so is infeasible given the data limitations of this analysis. Another method includes using categorical dummy variables coded by region to test differences between intercepts. However, neither of these methods allows one to view the factors involved in processes

of obesity operating cross-regionally (Jorgenson, Rice and Clark 2012). Slope-dummies allow for examining differences between slopes among categories by testing main effects and interactions between continuous and dichotomous variables (Jorgenson and Clark 2011).

A slope-dummy is an interaction term wherein a dichotomous variable, x_1 (Africa), is multiplied by a continuous variable, x_2 (Urban slum prevalence), which creates a new variable, x_1x_2 (urban slum prevalence*Africa). This new variable has the values of x_2 where $x_1 = $ "1" (urban slums) and "0" for all remaining cases (Allison 2009; Hamilton 1992; Jorgenson, Rice and Clark 2012). Homogeneity of slopes can be tested by entering into the regression equation the main effect (urban slum prevalence) and the slope-dummy variables created by the process above. Constructing variables in this manner primarily allows us to view how slums influence obesity in specific regions without sacrificing test cases. This technique in random effects model estimation was used to assess the comparative influence of urban slum prevalence, childhood stunting and GDP[3] across three geographic macroregions: Africa, Asia, and Latin America[4]. This method

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[3] Additional analyses were also examined using slope-dummy variables of urban slums by wealthy country status and childhood stunting by wealthy country status. These variables were created to surmount virtually no existing data on urban slums for European and OECD countries. While the median GDP per capita is a broad measure of national wealth, its breadth is justified to capture sufficient cases of urban slum for comparison. However, these additional variables did not substantively add to analyses already included. Results are reported as an appendix.

[4] Extending the logic of slope-dummy interactions allows one to include slope-dummies for all region categories to identify the couplings of a country's regional location in a more fully specified model including relevant controls. Analyses using slope-dummy methods were limited to Africa, Asia, and Latin America because of the lack of urban slums prevalence and childhood stunting data among North American, Oceanic and European countries. Slope-dummy analyses were limited to interacting urban slum prevalence and childhood stunting in only those three regions with all regions serving as reference categories in turn. Furthermore, unreasonably high variance inflation factor statistics, large standard errors, and correlated regression coefficients suggested multicollinearity unduly influenced fully saturated models, as

compares the generalizability of each perspective. In particular, eating habits have been shown to differ across regions (Iqbal et al. 2008), thus examining cross-regional differences allows us to view if these eating habits are sufficient to impact the relative inputs on BMI among the poor. Accordingly, if there are no differences among poor regions of the world, this speaks to the particularity of the influence of food security – that food insecurity affects the poor the same way in all areas of the world. However, on the other hand, if there are differences among regional inputs on obesity prevalence by urban slums and childhood stunting, then more accurate theorizing is needed to refine how relative poverty influences obesity in context specific ways. Since economic development plays a key role in providing excess calories, more economically developed regions should show more relative obesity risk than less economically developed regions. I hypothesize that there are regional differences on obesity prevalence by childhood stunting and urban slum prevalence. Obesity among African countries is more likely to be influenced by childhood stunting, but that obesity prevalence among Asian countries is more likely to be influenced by urban slum prevalence.

Third, I further examine the food security hypothesis by testing the effect of urban slums on food consumption itself. The logic of this second battery of tests is that if nutrition transition is the best explanation for the obesity epidemic, relative poverty should be indifferent to overall food consumption. On the other hand, if urban slum prevalence contributes to more calories, then obesity is based on the types of calories available to the poor. As an immediate input to obesity, food consumption should not

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would be expected. As such, models using poverty by region slope-dummies omitted two control variables – food consumption and population controls – in order to produce meaningful results. India and Vietnam were omitted due to overly influential Cook's distance results.

differ among poor populations. I follow the same methodological procedures outlined above using food consumption as a dependent variable.

A major limitation of this analysis is the range of available data. Longitudinal obesity prevalence is scarce even in the wealthiest of countries. Furthermore, obesity itself a condition located at an extreme of the body weight distribution and does not reflect the layered influences on weight gain as a whole. Average BMI, which is widely available cross-nationally serves as a useful proxy to gauge the overall influences of these statistical tests without suffering from data restrictions imposed by narrow samples[5]. Using BMI should provide a more generalized view of the influences of urban poverty, nutrition access and economic development on weight gain.

Fixed effects modeling is considered to be a more conservative test compared to other longitudinal estimation techniques like random effects. Fixed effects estimation differences out time invariant predictors using only within-case variation for model estimation (Allison 2009). This emphasis would impose practical and theoretical challenges on an already small data set reducing variation in this small sample size. Second, discarding between-case variation would hamper comparability of national circumstances across nations as these analyses are aimed at doing. The main challenge here is to avoid data limitation problems exacerbated by fixed effects modeling. Random effects modeling assumes unobserved variables to be uncorrelated with model predictors. This assumption allows for unobserved variables to be correlated over time. In order to

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[5]   Overweight prevalence was also considered as a test variable offering a more specific test on body weight itself. However, this variable limited the number of cases performed. Results of analyses on overweight prevalence were substantively similar to those of BMI. As such, BMI was considered a superior variable given its conceptual and practical differences from obesity prevalence in spite of its limitations as a test variable.

control for this bias, random effects modeling uses both within- and between-case variation to improve the efficiency of model estimation which also improves data availability limitations imposed by these data. For these reasons, I employ random effects modeling as it is well suited for macrolevel analyses. It is also theoretically preferred as it offers a comparison between countries and regions through its reliance on between-case variation.[6] Hausman specification tests of estimator efficiency were run on the preference of fixed effects but did not reveal that fixed effects estimation was more appropriate. Hence, I rely on random effects modeling over the theoretical and practical limitations fixed effects would impose on these data.

At first pass, lagged variables might seem appropriate given the delayed timing of the processes involved in growth stunting and economic development on emerging obesity. As Allison (2009) describes, introducing a lagged variable in fixed effect estimation introduces necessary correlations between predictors and error terms because of the lagged predictor's reciprocal relationship with the dependent variable. This biases estimation from violating model assumptions about the independence of variables, and would actually introduce heterogeneity bias which the panel model was intended to reduce. Also, because random effects estimation assumes no correlation for the fixed effect and error term, the necessary relationship introduced by the lagged predictor would be essentially assuming no relationship exists thus introducing more bias than already exists.

Furthermore, corrections for the autocorrelation problems introduced by lagged variables have, in the past, used instrumental variables in methods known as dynamic

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[6] Using the Breusch and Pagan (1980) lagrange multiplier test for random effects revealed that heteroskedasticity could influence standard errors but that clustered robust standard errors offer an appropriate correction.

panel models. Ideally, the instrumental variable would be a third variable independent of the outcome, but closely associated with the predictor that is used in first-differencing out the endogeneity or autocorrelation of related terms. Essentially, an instrument uses its unrelated variation with the outcome as means of parsing out the relationship of the variable of interest with the outcome.

Dynamic modeling then would use the causal variable of interest, lagged childhood stunting in this case, and its presumed uncorrelated relationship with the unobservable determinants of the dependent variable – obesity prevalence – to estimate unbiased beta coefficients. Lancaster (2000) indicates that the use of an instrumental variable, which in this case would be a lagged independent variable, should introduce new restrictions on the correlation of the instrument and other model parameters – namely that they should be independent. Using lagged indicators as instruments would not actually introduce any new data – as in an unrelated indicator – or new restrictions other than those already used in random effects maximum likelihood estimation. Thus the inclusion of lagged indicators would be inappropriate with random effects estimation, first through its additional associations with other predictors inherent in the model, but also in its failure to restrict bias inherent because of that association.[7]

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[7] Heckman corrections of sample selection bias was considered as a means of overcoming the missing data problem plaguing this research. However, heckman-type selection models require additional explanatory factors that influence sample inclusion but not the outcome of interest – in other words, an instrument (Sartori, 2003). Sartori explains that without instruments, heckman selection models base results off the distribution of residuals rather than the variation of explanatory variables. In using heckman selection models one either has to introduce an instrument which leads to additional restrictions of the independence of measures or estimate coefficients on the basis of residual distributions, in this case, derived from an already low sample N. Thus to appropriately use the heckman sample selection correction would introduce similar problems outlined above that ruled out the use of dynamic panel modeling. It is, however, worth considering that the pattern of missing data is not likely dependent on the outcome, and thus the main influence on the results would derive from simply

Sample

Countries used in baseline models for each dependent variable before listwise

deletion are listed by geographical region including years of data in Tables 1, 2 and 3.

Included countries are primarily low-and middle-income countries. I use cross-national

panel regression analysis using random effects estimation techniques to estimate the

effects of urban slum and stunting prevalence on obesity 1990-2007. This sample

included countries where partial or full data availability permitted analysis on dependent

and independent variables.[8]

Data

This analysis concentrates on lower-income urban populations at the national

level. Unfortunately, data availability prohibits examination at the urban level. I use two

measures of relative urban poverty to test the logic of nutrition transition against a food

security hypothesis. First, percent of urban population living in slums is taken from the

WHO Global Health Observatory database (2011) and is used to assess the influence of

urban slums on obesity. According to UN-HABITAT (UNHABITAT 2010), urban slum is

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having more cases to include in the analysis. Therefore in this sample, if there are
significant results with low N, it may reflect an actual relationship among variables
and those same associations would still exist with more data included in the analyses.

[8] Because of data availability requirements, imputation techniques were used on obesity
prevalence, slums population and stunting prevalence. The method of imputation
included averaging within-country data points for time 2 based on time 1 and time 3.
This procedure included imputing data on 41, 50, and 109 countries for obesity
prevalence, urban slums, and stunting prevalence, respectively. Subsequent
observations on these respective variables increased from 356 to 648 , 279 to 903, and
from 540 to 1,914. These imputation methods add substantial volume to key variables
and may be viewed with skepticism. However, this imputation method is a
conservative one based on change between existing time points. Without imputing,
these analyses would not be possible. Alternative imputation methods included
between-country imputation using year mean values, however, this method was not
justifiable given the average disparity between countries.

defined as one or a group of individuals under the same roof in an urban area lacking one or more of the following criteria: durable housing, sufficient living area for inhabitants, access to improved water and improved sanitation facilities. Conceptually, urban slums serves to assess the relative influence of urbanization on BMI given their concomitant rise among LDCs. Data include 5 time points; 1990, 1995, 2000, 2005 and 2007. Second, childhood stunting is used from the WHO (2011). This is coded as the ratio of the number of children aged 0 – 5 that fall below minus two standard deviations from median height-for-age of the WHO Child Growth Standards to the total number of children aged 0 – 5 measured (WHO 2011). Childhood stunting is caused by chronic undernourishment and serves as a common measure of under-nutrition. Childhood stunting is used to assess the influence of food insecurity directly. Because of the tendency for stunted children to be at higher risk for obesity later in life, this variable allows a direct view of the influences of food security on obesity prevalence. In using urban slum prevalence and childhood stunting, I test two theoretical puzzles of the global obesity epidemic. First, how are rapid increases in urban slums influencing BMI, as they are intimately associated with urbanization over the last several decades. Second, childhood stunting tests the effect of poverty in more absolute terms as chronic hunger concentrates among the poor.

Control variables include Gross Domestic Product (GDP, in constant 2000 US$) as a common control for absolute economic status. Logged total population was used as a country size control. In order to compare the relative influence of urban slums across countries, food consumption (kcal/capita/day) was also used to control for food access and consumption in order to better ascertain the influence of urban slums.[9] A measure of

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[9] In order to be able to compare nations according to obesity prevalence, a control for average body size was required, however, BMI as a covariate introduced several problems in this analysis. First, obesity measurement is itself based on BMI. As such

human capital was also introduced as a control by using percent of literate population[10] accessed from the World Bank Development Indicators database. Time was also introduced as a control including years from 1990-2007.[11]

Economic development plays a key role in both nutrition transition and food security hypotheses but each perspective differs in the way they view the effects of newly available calories. As discussed above, one pathway focuses on the obesity as a result of overconsumption from newly available calories, while the other pathway emphasizes obesity specifically among the chronically undernourished as a result of newly available calories. Because both perspectives emphasize the role of nutritionally poor, energy dense calories, I use fat and sugar consumption as a means to evaluate the role of poverty on obesity. Including calorie dense food types allows us to infer characteristics of the types of foods that have an effect on obesity by the urban poor. Fat and sweeteners are among the most energy dense types of foods and have been argued to be centrally involved in the obesity epidemic. These variables are available from 1980-2007 in average grams per capita per day from FAO Food Balance Sheets. I also use several development indicators to assess the overall influence of economic development and infrastructure on obesity

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BMI is highly collinear with the dependent variable with a serial correlation with obesity at .92. Second, BMI is also highly collinear with economic development correlated with GDP .84. As such, variance inflation factor statistics increased beyond 10 – a common cutoff – when including BMI. Third, BMI is a control variable which is too restrictive to be used in this analysis. Countries with low BMI would essentially be controlled for starvation, whereas high BMI countries would be controlled for obesity. Food consumption was reasoned to be a better control because of lower VIF scores and lower serial correlations with other covariates. Food consumption was used in models where fat and sugar consumption was not examined.

[10] Education presented another limiting variable, which by imputing following the method above, increased observations from 2792 to 3719 across 130 countries.

[11] Time was examined for linear effects through assigning each year a dichotomous dummy variable. Results indicated that time was more effective as a control when used as a continuous variable.

prevalence. Because economic and social development play such a large role in creating conditions for an obesity prevalence, I test various aspects of economic development that play crucial roles in increasing caloric supply and access. First, Foreign Direct Investment (FDI) stocks as a percentage of GDP included as a measure of global investment (UNCTAD 2012). Foreign investment has been argued to increase inequality and reduce economic growth (Alderson and Nielsen 1999) and has been identified as a key factor in food security and hunger in less developed countries (Jenkins and Scanlan 2001; Wimberly and Bello 1992). It also has been linked to reducing food costs throughout the global south (Hawkes 2002).

Both foreign and domestically processed foods have been shown to play large roles in global obesity (Kumanyika 2008). I use value-added agriculture output as a percent of GDP to assess the strength of the domestic food production and processing sector. An ideal measure would have been more closely related to food processing, but as such, because of its generality, it is also able to asses the wide variety of food production available as a country develops. On the other hand, processed foods are referred to as "value-added" in that a raw agricultural product like wheat is transformed into pastry, which is in large measure a key aspect of obesity (Tullao 2002). I use food imports as a percent of total merchandise imports to test for the amount of food products imported from the world market. This variable also could be considered too general for the purposes of assessing obesity, given the high proportion of food imports consisting of grains. Still, most of the world's calories come from grains, and their consumption is shifting from traditionally high fiber grains like millet and maize to high starch, refined grains like rice and wheat, especially in urban areas (Kearney 2010; Popkin 2001a;

Uusitalo, Pietinen and Puska 2002). Food trade is viewed as a key instigator of global

obesity (Chopra 2002; Hawkes 2006; Tullao 2002). Total female labor force participation

is used to control for time constraints related to food preparation in the nutrition transition

among LDCs (Bleich, Blendon and Adams 2007; Rashad and Grossman 2004). It is also

considered a measure of social development. Public health expenditure as percent of total

GDP is included to test for impact of preventative and curative health services including

family planning, nutrition activities and emergency aid.[12] Wealthy country status is

included as a dummy variable based on GDP median split as a means of general

comparison. A more detailed measure of GDP is already included as a control variable.

Thus in order to reduce collinearity, this measure was used as a broad means to make

general comparisons between countries of different income status. In addition, because of

the relatively low number of cases and relatively homogenous economic subgroup

included in the first analysis, a more generalized version of economic status was

preferred. This variable was used in subsequent analyses for consistency across analyses.

Tables 3, 4, and 5 list countries included in the analysis.

In the third analysis using food consumption as the dependent variable, I add an

indicator of political regime as a test of political autonomy. Food consumption has been

shown to be sensitive to political regimes type, political discrimination, militarism and

militarization characteristic in autocratic regimes (Jeanty and Hitzhausen 2006; Jenkins

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[12] GINI coefficients were considered as a measure of domestic income inequality. However, including GINI coefficients introduced a number of problems. First, when GINI was included in virtually all analyses, total cases in the analysis dropped below 30 which is commonly considered to be statistically unstable. Second, including GINI increased variance inflation factor by 5 times their values compared to models omitting GINI. Third, in these models, controlling for GINI did not change the influence of urban slums on obesity. Fourth, GINI figures were imputed on the basis of data limitation following the procedure outlined above but still did not yield sufficient cases for analyses. Based on these considerations, GINI was not included this analysis.

and Scanlan 2001; Wimberly and Bello 1992). I use an indicator of political regime as a control on food availability. Polity2 is widely available from the Inter-University Consortium for Social and Political Research and represents the most popular measure of a country's political regime (Plümper and Neumayer 2010). Polity2 refers to an institution-based measure of political regime that reflects the competitiveness and regulation of political participation, constraints on the chief executive, and the openness and competitiveness of executive recruitment on a 21 point continuum score from -10 (most autocratic) to 10 (most democratic) (Maystadt, Tan and Breisinger 2012). Lower-income countries form the reference category. All variables except time have been logged to reduce skew. Serial correlations are located in Table 6.

Results

Several analyses were conducted using several key independent variables and two primary dependent variables. In the first analysis, using obesity prevalence, I test the influence of urban slums and childhood stunting and the relative inputs by calorie dense foods. I test these same relationships on average BMI as a means to check for consistency. Next, I use development indicators to assess changes in the effects of urban slums and childhood stunting on obesity prevalence and BMI. I then use slope-dummy variables to assess the influence GDP and urban slum prevalence and childhood stunting across regions. Last, I analyze urban slums prevalence on food consumption to ascertain the mediating role of economic development in food access.

Table 7 displays the results of the effect of urban slums and childhood stunting on obesity prevalence. Model 1 tests the percent in urban slums and childhood stunting.[13]

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[13] Both urban slums and childhood stunting were run on obesity prevalence and found

Model 2 tests the influence of sugar consumption, model 3 tests fat consumption while model 4 includes both, and model 5 includes consumption variables and controls.

It is noteworthy that alone, urban slums has a negative effect on obesity prevalence, but childhood stunting is positively associated. While the magnitude of each key independent variable changes across each test, the overall substantive impact remains similar. The test of sugars indicates a negative influence on obesity prevalence in model 2 while the effect of fat consumption in model 3 is nonsignificant. However, when including control variables, a nonsignificant effect from fats switches to relatively strong positive association. Correspondingly, childhood stunting is nonsignificant and the negative effect of urban slums increases substantially.[14]

As a complimentary test of the influence of particular kinds of calories, Table 8 includes the same tests as Table 7 using average BMI as the dependent variable. Results indicate that urban slum prevalence has a negative association with average BMI, though that relationship appears to be influenced by other variables.

On the whole, it is not statistically significant. Childhood stunting is negatively associated with BMI, growing weaker as consumption variables are introduced. Models 2 and 3 show that sugars and fats increase BMI, respectively, but together both variables are not significant. When adding controls, only year is statically significant, indicating increasing BMI over time.

Table 9 shows the results of seven models on obesity prevalence that assess change introduced by each set of variables relevant to nutrition transition. Table 8 offers supplementary results of seven models using the same covariates on average BMI as the

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that alone, neither variable were statistically significant.

[14] It is also worth pointing out that cases in the fully saturated model are below 30 and should be viewed with relative caution.

dependent variable to compare against the data limitations from obesity prevalence in Table 5. Model 1 serves as baseline model consisting of percent living in urban slums and proportion of stunted children with control variables including GDP per capita, percent literate, total population size, and average food consumption. Models 2 through 7 add one test variable each to controls starting with foreign direct investment as a percent of GDP in model 2, food imports in model 3, health expenditure as a percent of GDP in model 4, value-added agriculture in model 5, female labor force participation in model 6 and finally, the wealthy country dummy variable in model 7. Based on calculations of Cooks distance, influential cases were omitted from these models.[15] Variance inflation factor statistics for all models did not indicate problematic multicollinearity. Across models, testing obesity prevalence as the dependent variable, in Table 8, the influence of urban slums is negative while childhood stunting is consistently positive. Control variables serve to intensify these relationships. GDP was positively associated with obesity while population size was negatively associated across models 1 through 7, as was time. No variables among test models were statistically significant, though each impacted the relative effects of urban slums and childhood stunting to some extent. Notably, food imports and public health expenditure served to reduce the negative effect of childhood slums on obesity.

Table 10 shows the influence of covariates on average BMI. It is notable that the number of cases included in this analysis increased dramatically compared to prior analyses but that increase did not accompany statistically significant results. Like the

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[15] In all models in Table 4, Cooks distance statistics suggested that Vietnam, India and Madagascar were overly influential cases and thus, they were omitted from the analysis. Sample size varies across models based on listwise deletion from missing data.

obesity prevalence models above, model 1 serves as the baseline model testing urban slums and childhood stunting on BMI with controls, and models 2 − 7 add test variables one at a time similar to Table 6 models. Urban slums are negatively associated with average BMI and statistically significant in the baseline model, net of controls, and when testing value-added agriculture, female labor force participation and wealthy status dummy variables. The effect of childhood stunting is negative and statistically significant but changes to positive and nonsignificant when including controls and test variables. Among control variables, GDP per capita tended to increase average BMI. Time also contributed to higher BMI across all models. Among test variables, food imports and female labor force participation increased average BMI, while all other test variables were nonsignificant.

In short, I find strong evidence of an inverse relationship between urban slums and obesity prevalence. Some test variables reduced urban slums to nonsignificance, while others did not. Also, childhood stunting playing a substantial consistent role in obesity prevalence though the corroborated evidence from BMI is less straight forward. Net of controls, childhood stunting was nonsignificant.

I also compared the effect of urban slums and stunting on obesity prevalence by region in Table 11. This set of models was designed to examine the differences of urban slums and childhood stunting across geographical region[16]. Model 1 tests urban slums and childhood stunting on obesity prevalence using slope-dummy interactions of percent living in urban slums by region, where Asia serves as the reference region. Model 2 adds control variables GDP, percent literate, and time. The main effect of urban slums is

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[16] Models using other reference categories showed consistent results with those reported. The product term of childhood stunting and Latin America were not statistically different from the reference category and were omitted from Figure 4.

consistent with prior models above – that urban slums decrease obesity prevalence though magnitudes and statistical significance varies by control variables.

Disaggregating regression coefficients of model 1 in Table 11 shows the relationship of key variables on obesity prevalence (Figure 4) across macroregions. Overall, not all macroregions demonstrate statistical significance of urban slums. Asian urban slums are negatively associated with obesity prevalence as evidenced from model 1 though it is only significant when adding additional control variables in model 2. In addition, while the urban slum effects vary between macroregion, the slum-region interaction terms are not significant. Asian childhood stunting increases obesity prevalence but African childhood stunting decreases obesity to a greater extent than Asia. Product terms between urban slums and region were not statistically significant indicating no differences between regions, and were therefore not graphically depicted. The main effect of Latin America is noteworthy – compared to Asia, Latin American countries alone show considerable higher obesity outcomes though this effect is offset by null interaction terms.

I also tested the effect of wealth on obesity prevalence among macroregions using slope-dummies between GDP and region in Table 12.[17] Figure 5 show disaggregated regression coefficients of wealth from model 1 across regions, with Asian countries as the reference group. Overall wealth increased obesity among Asian countries relative to other regions. Wealth among African and Latin American countries was negatively associated with obesity prevalence relative to Asia.[18] When adding control variables, the effect of

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[17] Table 10: Vietnam was identified an outlier based on extreme Cooks distance value. Including food consumption and population size as control variables among introduced multicollinearity made coefficients not readily meaningful. In addition, Europe and Oceania were omitted for lack of variability.

[18] Models with different reference groups also indicated similar results.

GDP among Latin American countries did not differ from those of Asian countries.

The above tests have been designed to examine the effectiveness of nutrition transition and food security logics in explaining the role of poverty in global obesity. By testing poverty and nutritional deficiency commonly associated with obesity, I showed that poverty is not more likely to increase body weight overall, and that there are regional differences in weight outcomes based on poverty. Slums among Latin American countries in particular, show greater associations with obesity and Asian slums show better obesity outcomes. Conversely, I showed how childhood stunting increases obesity overall, but that differences exist across macroregions. Notably Latin American stunting affects obesity greater than other macroregions, while African stunting does not contribute to obesity. Contrary to the food security hypothesis, these tests have served to rule out the idea that poverty is uniformly responsible for increasing obesity over time. However, on the whole, these analyses suggest only an indirect route to obesity and are not direct tests of food consumption among the poor.

In Table 13, I test the proposition of the food security perspective more directly by regressing poverty, economic integration and control variables on food consumption. If there is an effect of poverty on obesity over time, there should also be an effect on the amount of food consumed. Table 12 examines the effect of poverty on food consumption.[19] Model 1 serves as a baseline model testing only the effect of urban slums. Model 2 introduces controls – GDP, percent literate, population size, and time. Models 3 through 6 test development variables while model 7 gauges the influence of the wealthiest half of countries relative to the poorest. Model 8 tested the effect of political

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[19] Five influential cases based on Cooks distance were excluded including Chile, Zimbabwe, Suriname, Jamaica, Haiti and Dominican Republic.

regime on food consumption. Notably, urban slums are negatively associated with food consumption but its effect on food consumption are nullified by control variables. None of the the test variables are significant predictors of food consumption except political regime though it does not substantively change the influence of slums on food consumption.

Discussion

The central question in these analyses asked what the effect of poverty, if any, has on body weight. Two main perspectives emphasize the role of cheap calories and wider food access. Prior evidence indicates that among wealthy nations, the poor tend to be most afflicted by obesity, whereas the picture is less clear in developing countries. This study aimed at assessing the routes of obesity through food supply, and economic and social development indicators cross-nationally to strengthen existing empirical and theoretical pathways of the global obesity epidemic.

The fundamental cause of weight gain is undeniably more calories in than out. The question of global obesity is rooted in how calories are made available and to whom. As the nutrition transition suggests, economic development increases marketability, buying power and impetus to buy energy dense calories. The influx of cheaper calories makes them more available to everyone and ultimately obesity follows.

The food security hypothesis suggests that under-nutrition leaves physiological dispositions towards obesity through growth stunting which occurs primarily among the chronic poor. It suggests that the current obesity epidemic is being driven primarily because of food access inequality. However, evidence presented here would suggest a

more nuanced view of the poverty-obesity link is warranted than nutrition transition theory or food security suggest. According to food security, we should see the effect of sweets and fats convert the baseline urban slum effect to positive on obesity prevalence because, according to this perspective, the food insecure would be more likely to purchase more of these kinds of cheap foods. Calorie dense foods should contribute to obesity through the mechanism of childhood stunting. According to the nutrition transition hypothesis, fats and sweets should nullify the effect of urban poverty because all people should now have access to cheap calories. Fats and sweets essentially cancel out the negative effect of poverty because of their strong positive contributions to obesity. Fats and sweets should influence in some degree the effect of childhood stunting because both are nutritionally unhealthy. Given the rise of these kinds of calories, sugars and fats should increase the effect of childhood stunting.

First, the main findings from analyses including food content from Table 4 indicate the urban poor are less likely to be obese, regardless of what kind of fats they eat. Results show that when controlling for economic and social development, fat consumption removes the negative influence of malnutrition on obesity risk but in these cases, it contributes to higher obesity risk itself. Furthermore, when controlling for fat consumption the effect of poverty is lower than an already fairly consistent negative association Evidence would suggest that cheap calories are providing those in poverty with excess calories leading to obesity, but the strong negative effect of living in an urban slum overwhelms the positive influence of fat consumption. Sugar consumption itself reduces obesity prevalence but only slightly counteracts the effect of urban slums and childhood stunting on obesity prevalence. Overall, sweets do not predispose the poor to

be at higher risk of obesity, but it does chip away at the "protective factors" of poverty.

Results show that fat and sweetener consumption are not having the effect that nutrition transition is predicting. People having greater access to more calories does not necessarily increase obesity prevalence among the poor. At least this sample does not show adverse obesity effects among the poor from fats and sweets. Also, we are not seeing the effect sweets and fats predicted according to the food security hypothesis either. We see that the positive effect of childhood stunting on obesity is actually being improved by fats and sweets access. Basically, by providing fats among malnourished children we reduce obesity risk later in the life course. This effect is likely functioning through the mechanism of providing malnourished children with adequate calories for normal, healthy development.

Overall, the poor are not at higher risk of obesity regardless of region. However, the effect of GDP appears to have a substantial positive effect on obesity for Asian countries, but among African and Latin American countries, the association is negative. These findings may be explained in part by different regional eating cultures. For example, factor analysis from the INTERHEART study across 52 countries shows a dietary convergence into 3 main dietary patterns; one of which is the oriental pattern characterized by foods high in soy and tofu (Iqbal et al. 2008). The other two main types of foods are similar in that they emphasize typical Western diets high in fats and sodium. The relatively higher obesity risk among Asian countries could be the effect of a traditional healthier "oriental" diet transitioning to a Western diet from newly acquired wealth. The effect of wealth could be further evidence of a more nutritionally rich diet available to the Asian public. It might also be noted that in this sample, the average GDP

for Asian countries is more than one standard deviation higher than African countries. It stands to reason based on these findings that income serves to contribute to over-nutrition among Asian countries, and convey healthy diet options to those in Latin American and African countries. Another interpretation would suggest that among African countries in particular, the effect of GDP demonstrates how far these countries have to go to improve their overall nutrition.   Among these countries, income is negatively associated with obesity prevalence, but this may indicate that many African countries have yet to commence a full-scale nutrition transition of their own.

Second, these findings indicate that poverty does not substantively influence national BMI. Included as a check for data limitations, using BMI as its own outcome indicates interesting trends. Urban slums mostly do not effect BMI, nor do cheap foods net of control variables. The discrepancy is likely due to the level of analysis that each dependent variable contains. Overall, the effect of pockets of urban poverty does not statistically influence the whole of the national body mass. This should be encouraging given that the conditions of chronic hunger among urban poor do not disrupt most people of that country getting adequate nutrition.

The use of two dependent variables in obesity prevalence and BMI to ascertain the role of key variables on obesity trends is not ideal. However, additional analyses comparing test variables on both obesity prevalence and BMI using equal sample countries indicated similar findings as those reported on the full sample. These variables were not intended to be identical, nor necessarily complimentary. Indeed, they represent very different expressions of a country's weight profile. The analyses contrasting these variables show complimentary findings: urban poverty does not pose a threat to rising

obesity risk. Had the analyses using obesity prevalence and BMI been substantially different where the effects of slums been positive on obesity prevalence and negative on BMI or vice versa, this would have indicated greater theoretical challenges to both international obesity theories other than its consistent negative effect on obesity prevalence and no statistical effect on BMI.

Third, another main finding in this research is that childhood growth stunting does increase obesity prevalence, but it did not increase average BMI. The argument by the food security hypothesis reads that because growth stunting comes as a result of nutritional deficiency, it increases obesity but not average BMI because of the low nutritional content of the food poor children eat over time. As demonstrated by prior research, growth stunting is an obesity risk over the long-term but has a short-term suppressing effect on BMI given that stunted children are malnourished and underweight. Research from the United States has indicated that food deserts – poor urban areas without access to fresh or nutritionally complete foods– contributes to a higher proportion of obese given the nutritional content of ingested foods. While calories are being consumed in these areas, the type of calories people consume predispose them to obesity because of the energy density to nutritional content ratio (Cohen, Küpçü and Khana 2008; Cummins and Macintyre 2006; Willet 1998a; Willet 1998b).

Thus the increase in obesity prevalence relative to BMI reported above is determined not by the amount of food consumed but the *malnutrition* of the poor, or "hidden hunger" (Tanumihardjo et al. 2007; Uauy and Kain 2002). I find differential effects of nutritional content of food being consumed by the poor. As argued, negative effects of poverty on obesity prevalence compared to the null effects on BMI suggest that

adequate nutrition is not occurring universally. Recent research into obesity "paradoxes" confirms this view. Among six middle-income countries, 66 percent of households with an underweight person also contained an overweight person (Doak et al. 2004). Greater linear growth in later adolescence and early adulthood increases the propensity for obesity among stunted children (Doak et al. 2004; Uauy and Kain 2002; Walker, Chang and Powell 2007). My findings accentuate the different pathways between stunting, obesity, and BMI – that hidden hunger is likely to exist among households with growth stunted children because of where they live and what they eat.[20] I find that fat consumption essentially evens out obesity risk for malnourished children, but it does not counteract the nutritional inadequacies of poverty alone. Poor parents may be undernourished themselves, but their growth stunted children may be obese.

Lastly, it is also worth noting that among many of the economic development indicators,

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[20] Additional tentative support for this conclusion is based on a particular anomaly found in the results. Tables 8 and 9 show the influence of regressors on obesity prevalence and BMI, respectively. Food consumption is often argued to be a direct correlate of both obesity and high BMI. However, the effect of food consumption on both outcomes is largely insignificant except in the case of using public health expenditure as a test variable. In Table 9 model 4, the effect of food consumption goes from nonsignificant to positive and significant at the .01 level. First, public health expenditure includes nutrition related expenses as part of the indicator, so holding it constant allows the effect of nutrition related expenses to be held constant which biases this variable towards wealthier countries included in the analysis. Second, holding food consumption constant biases towards low calorie ingestion since there are more countries with lower food consumption included in these analyses than wealthy countries with higher caloric ingestion. Third, the effect of public health expenditure reflects the overlap between food consumption which biases towards poorer countries, and the bias of wealthier countries which have higher public health expenditure. Fourth, food consumption is a measure of caloric intake and is consistently nonsignificant across all models except where nutritional supplements are controlled for. This finding then reflects the effect of calories included in nutritional supplements to the malnourished by public health expenditure over and above the effect of food consumption alone. Nutritional supplements included in public health expenditure balance the effects of food consumption and malnutrition biases thus allowing the net effect of total calories to positively contribute to higher BMI, and not obesity prevalence.

many did not pose obesity risks themselves, change in a meaningful way obesity risks of the poor, or even improve food consumption among the urban poor. Table 9 shows some important implications for economic development on BMI. First, foreign direct investment appeared to change negative obesity risk among the urban poor to nonsignificant, as did food imports, and public health expenditure. These tests are not directly examining nutrition among the poor, but indirectly these findings show that given these inputs, at least poverty does not matter for obesity outcomes, as compared to malnutrition that the negative association implies.

In sum, the evidence suggests greater theoretical evidence is warranted in exposing the global obesity epidemic. Urban poverty is not uniformly associated with obesity. Also, the effect of urban slums on BMI is mostly changed through childhood stunting, but not by the type of food available. As these data show, urban slums produce a consistent negative association with obesity. Fat consumption does contribute to obesity, to the extent that it removes the risk of childhood stunting on obesity, but not sufficient to nullify the effects of poverty.

The influence of urban slums on obesity across macroregions are roughly equal. The effect of childhood stunting also does not play out according to the food security hypotheses particularly where childhood stunting is more common. Among African countries, childhood stunting is negative, where one might theoretically expect it to be positive. It would appear then that the food security hypothesis requires additional qualifications to be applicable in all contexts. It may be that more defined income gradients not included in this analysis would more clearly delineate countries with higher poverty-obesity risk. Perhaps more importantly, food security predicted a stronger effect

of urban slums on food consumption. Instead, we see that urban poverty is not related to food consumption, nor are most of the control economic integration test variables included here.

In actuality, food consumption is mostly determined by GDP, as nutrition transition would indicate. Also, economic development as measured by GDP, is only positively associated with obesity in Asian contexts. If these data showed more evidence of nutrition transition, one might have expected the effect of chronic poverty to be less negatively associated with with obesity prevalence and to have a more uniform effect on obesity, particularly among regions with growing obesity prevalence, like South America. Hence, nutrition transition appears to be more substantively supported, in as much as the sample included here contains many countries that have fully completed their nutrition transition.

Limitations

There are several limitations to this study. First, the nature of these test variables adds complications to the testing process. Economic development, obesity, and food consumption are examples of sources of collinearity among variables. These relationships tend to violate primary assumptions on which these methods rely.

Second, data limitations comprise the largest challenge in this study. Proportions of obese populations are primarily available for Organization for Economic Cooperation and Development (OECD) member countries. However, OECD countries do not exhibit the same economic and political vulnerability as do many nations of the global south. In general, the world has grown heavier over time, represented by a shift in mean BMI since

the 1980s. This, however, does not mean that all portions of the population have increased their BMI, nor that the national BMI represents each particular obese demographic across the world. Obesity prevalence would skew BMI measures up, but BMI does not necessarily indicate the effects of smaller proportions of obese. Also, more detailed food consumption data would be useful – such as proportion of prepackaged foods or meals eaten out would be ideal in this sort of analysis. However, to deal with data limitations, I have presented analyses of poverty on both obesity prevalence and BMI with readily available correlates of calorie dense foods, which have produced comparable results.

Another major data limitation is the absence of suitable physical activity data. Urbanization has been acknowledged and used in prior research as a proxy for physical activity, as has value-added agricultural output (Bleich et al. 2007; Sobal and McIntosh 2009). Admittedly, these may be poor proxies. It is well noted that many in urban areas rely on public transportation, bicycles and walking for transportation and this omission may be a source of omitted variable bias. However, random effects modeling techniques was used as a means of mediating this bias. For example, the random effects model has been shown to be a special case of the fixed effects (Allison 2009). The value of using panel models in this case is in controlling for unobserved heterogeneity, in which omitted variable bias plays a role (Winship and Mare 1992). While fixed effects models do a better job of controlling for omitted variable bias, the trade-off in efficiency introduced by only estimating within-case variation reduces its explanatory power. The random effects model on the other hand, maximizes efficiency by assuming no correlation between unobserved and observed variables. This assumption reduces efficiency at the

risk of bias. The upshot for purposes here is that using random effects modeling may have introduced some degree of bias, but the Hausman specification test run on the presence of omitted variable bias noted, indicated that the correlation between unobserved and observed variables was insignificant. Omitted variable bias may have been present in this analysis, but its correlation with observed variables did not appear to interfere with model efficiency (Allison 2009).

Third, global obesity research has been done mostly at the national level. The present research, however, attempts to view obesity as a result of structural forces cross-nationally. The challenge of a taking a global view of obesity cuts two ways. Firstly, theorizing on the national level should generalize to global processes barring other intermediate processes. Secondly, processes at the global level may be wholly different from the national level when considering this sample. While both scenarios are possible, the range of countries and the use of multiple dependent variables favor the former – regional levels reflect intermediate processes that differentiate obesity trends at the country level rather than simply generalizing national trends to the global level. This analysis essentially risks ecological fallacy by positing links to lower-level obesity risks from using country level data. Still, results here suggest that people living in slums in various countries are less prone to obesity based on country level prevalences. Nor do the data reflect regional level extrapolations to country level processes. There may be some risk of comparing subpopulations of slum-dwellers to the average body mass that perhaps are not necessarily representative samples, but these levels of analysis inherent in the data are comparable. This study is actually more at risk of committing the reductionist fallacy – using country level data to make inferences about regions. However, the interpretations

of regional analyses are given for countries comprising their respective regions, and not made with respect to the regions themselves.

Conclusion

The picture emerging here suggests that the global obesity, while prevalent, is not occurring evenly. In many ways, this research shows that poverty does not carry substantial obesity risk, but that people living in urban slums must still deal with more pressing concerns like chronic hunger. Economic development appears to have an impact on obesity at the global and regional level, however, this relationship is not uniform among all global macroregions. Using three novel techniques in obesity research, I find that the global obesity epidemic is not uniformly predicted only by national level processes but also by regionally specific elements driving unique contributions of poverty cross-nationally. This nonuniformity across regions suggests that intervening structures influence obesity extending beyond dichotomous poverty-affluence paradigms.

The conclusion yields several further questions. Given the uniqueness of regional trajectories of obesity, how might obesity be influenced by global geopolitical structures? Is there a stronger effect of global integration not measured through economic development? Does the measure of indicator matter for these processes – BMI over obesity prevalence? There is an apparent need to clarify regional influences on obesity as well as exploring within and between relationships to further understand the global obesity epidemic.

Table 2
Adult Obesity Prevalence in Selected Countries

Country	Year of Data Collection	Age Category	Males		Females	
			Overweight	Obese	Overweight	Obese
			% BMI 25-29.9	% BMI 30+	% BMI 25-29.9	% BMI 30+
England	2004	16+	43.9	22.7	34.7	23.8
Germany	2002	25+	52.9	22.5	35.6	23.3
Poland	2002	18-94	39.0	19.0	29.0	20.0
Spain	1990-2000	25-60	45.0	13.4	32.2	15.8
Iran	2000	20+	42.0	10.0	45.0	30.0
Pakistan	N/A	18+	18.3	4.5	21.4	5.9
Saudi Arabia	1995-2000	30+	42.4	26.4	31.8	44.0
Australia	2000	25+	48.2	19.3	29.9	22.2
Congo (urban)	1996	15+	–	2.3	–	5.8
Mali	1996	15-49	–	–	7.2	1.2
South Africa	1998	15+	21.1	10.1	25.9	27.9
Argentina	2003	18-65	24.6	19.5	10.8	17.5
Mexico	2000	20-69	41.3	19.4	36.2	29.0
USA	2003-2004	20+	39.7	31.1	28.6	33.2
India	1998	18+	4.4	0.3	4.3	.6
Indonesia	2001	15+	7.3	1.1	14.2	3.6
Thailand	1997	20-59	15.7	3.5	25.1	8.8

(Lobstein and Leach 2006)

Table 3
Countries Included in Analysis Using Obesity
Prevalence as Dependent Variable

Region	Africa	Asia	Latin America
Country (years)	Egypt (2005-2006) Madagascar (2005) Morocco (1998-2000) South Africa (1998 Zimbabwe (2005)	China (1993-2002) Indonesia (2001) Mongolia (2004-2005) Pakistan (1990-1994) Philippines (1998-2003) Turkey (1997-2004) Vietnam (2000)	Brazil (2002-2003) Colombia (2004-2007) Dominican Republic (1996-1998) Mexico (2000) Peru (2000)

Table 4
Countries Included in Analysis Using Average
BMI as Dependent Variable

Region	Africa	Asia	Latin America
Country (years)	Angola (2005)	Bangladesh (1992-2007)	Argentina (1994-2005)
	Benin (1996-2006)	Cambodia (2005)	Bolivia (1990-2007)
	Burkina Faso (1993-2007)	China (1990-2007)	Brazil (1990-2007)
	Cameroon (1991-2006)	India (1990-2006)	Colombia (1990-2007)
	Central African Republic (1995-2000)	Indonesia (1995-2007)	Costa Rica (2005)
		Iraq (1991-2006)	Dominican Republic (1990-2007)
	Chad (1997-2004)	Jordan (2005)	
	Comoros (1992-2000)	Laos (2005)	El Salvador (2005)
	Congo DR(2005)	Mongolia (1992-2005)	Guatemala (1990-2007)
	Cote d'Ivoire (1990-2007)	Nepal (1995-2006)	Guyana (2005)
	Egypt (1998-2007)	Pakistan (1990-2001)	Haiti (1990-2006)
	Ethiopia (2000-2005)	Philippines (1990-2007)	Honduras (2005)
	Gambia (2005)	Saudi Arabia (2005)	Jamaica (2005)
	Ghana (1990-2007)	Syria (2005)	Mexico (1990-2006)
	Guinea (1995-2007)	Thailand (2005)	Nicaragua (1998-2007)
	Guinea Bissau (2005)	Turkey (1993-2004)	Paraguay (2005)
	Kenya (1990-2007)	Vietnam (1999-2007)	Peru (1990-2007)
	Lesotho (2005)		Suriname (2005)
	Madagascar (1990-2007)		Venezuela (2005)
	Malawi (1990-2006)		
	Mali (1996-2006)		
	Morocco (1990-2004)		
	Mozambique (1995-2003)		
	Namibia (1992-2007)		
	Niger (1990-2006)		
	Nigeria (1990-2007)		
	Rwanda (2000-2005)		
	Senegal (1990-2005)		
	Sierra Leone (2005)		
	Somalia (2005)		
	South Africa (1994-2007)		
	Sudan (2005)		
	Togo (2005)		
	Uganda (1990-2006)		
	Tanzania (1992-2005)		
	Zambia (1992-2007)		
	Zimbabwe (1990-2006)		

Table 5
Countries Included in Analysis Using Food
Consumption as Dependent Variable

Region	Africa	Asia	Latin America
Country (year)[a]	Angola (2005)	Bangladesh	Argentina
	Benin	Cambodia (2005)	Belize (2005)
	Burkina Faso	China	Bolivia
	Burundi (2005)	India	Brazil
	Cameroon	Indonesia	Chile (2005)
	Central African	Jordan (2005)	Colombia
	Republic	Laos (2005)	Costa Rica (2005)
	Chad	Lebanon (2005)	Dominican Republic
	Comoros	Mongolia	Ecuador (2005)
	Congo DR (2005)	Myanmar (2005)	El Salvador (2005)
	Cote d'Ivoire	Nepal	Grenada (2005)
	Egypt	Pakistan	Guatemala
	Ethiopia	Philippines	Guyana (2005)
	Gabon (2005)	Saudi Arabia (2005)	Haiti
	Gambia (2005)	Syria (2005)	Honduras (2005)
	Ghana	Thailand (2005)	Jamaica (2005)
	Guinea	Turkey	Mexico
	Guinea-Bissau (2005)	Vietnam	Nicaragua
	Kenya	Yemen (2005)	Panama (2005)
	Lesotho (2005)		Paraguay (2005)
	Madagascar		Peru
	Malawi		St. Lucia (2005)
	Mali		Suriname (2005)
	Morocco		Trinidad and Tobago
	Mozambique		(2005)
	Namibia		Venezuela (2005)
	Niger		
	Nigeria		
	Rwanda		
	Senegal		
	Sierra Leone (2005)		
	South Africa		
	Sudan (2005)		
	Togo (2005)		
	Uganda		
	Tanzania		
	Zambia		

[a]Countries include years 1990-2007 unless otherwise stated.

Table 6
Summary Statistics and Bivariate Correlations

Variable	N	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
BMI	5568																
Obesity Prevalence	648	1															
Food Consumption	4761	.354	1														
Urban Slum Prevalence	903	-.770	-.529	1													
Childhood Stunting	1914	-.566	-.622	.576	1												
Fat Consumption	4637	.062	-.062	.134	.101	1											
Sugar Consumption	4638	.029	-.097	.199	.202	.704	1										
GDP	5735	-.158	.563	-.492	-.366	-.096	-.105	1									
% Literate	3719	.036	.579	-.562	-.403	-.027	-.022	.383	1								
Population Size	6789	-.325	.054	-.179	.072	-.063	-.058	.758	-.027	1							
Year	8064	.263	.079	-.153	-.074	.113	.058	.097	.187	.057	1						
FDI	4119	.027	.006	-.160	-.173	.058	.032	-.146	.167	-.214	.363	1					
Food Import	4845	-.171	.216	-.426	-.294	-.101	-.036	.941	.297	.780	.050	-.159	1				
Health Care Expenditure	2935	.227	.320	.364	-.153	.065	-.075	.013	.212	-.097	-.116	-.015	.134	1			
Value-Added Agriculture	4803	-.098	-.638	.638	.645	.051	.055	-.497	-.436	.087	-.189	-.172	-.441	-.116	1		
Female Labor Force	5647	-.204	-.167	.332	.220	-.021	-.017	-.087	-.072	-.002	-.123	-.007	-.202	.498	.093	1	
Wealth Dummy	8064	.009	.232	-.409	-.279	-.057	-.069	.814	.339	.386	.003	-.101	.536	.011	-.382	-.128	

Table 7
Random Effects Analysis on Obesity Prevalence,
1990 – 2007

Variables	1	2	3	4	5
% in Urban Slum	-0.691***	-0.935***	-0.931***	-0.995***	-1.751***
	(0.250)	(0.313)	(0.332)	(0.308)	(0.164)
Childhood Stunting	0.560***	0.577***	0.607***	0.568***	-0.323
	(0.170)	(0.127)	(0.144)	(0.120)	(0.580)
Test Variables					
Sugar Consumption		-0.319*		-0.515***	-0.482
		(0.191)		(0.0943)	(0.595)
Fat Consumption			-0.189	0.303	0.751*
			(0.291)	(0.250)	(0.426)
Control Variables					
GDP					-0.371
					(0.841)
% Literate					1.111
					(0.696)
Population					-0.777
					(1.169)
Year					-0.197*
					(0.104)
Constant	-0.629*	-0.785**	-0.776**	-1.069***	394.4*
	(0.343)	(0.327)	(0.354)	(0.288)	(208.7)
Observations	51	37	36	36	25
Number of id	16	11	10	10	8
Rho	0.964	0.937	0.932	0.918	0
Between Rsq	0.119	0.283	0.267	0.467	0.860
Within Rsq	0.304	0.455	0.399	0.375	0.0924
Overall Rsq	0.257	0.314	0.259	0.540	0.862

Notes: Standardized regression coefficients. Robust standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 8
Random Effects Analysis on Average BMI,
1990 – 2007

Variables	1	2	3	4	5
% in Urban Slum	-0.209*	-0.264	-0.258	-0.256	-0.00931
	(0.126)	(0.170)	(0.172)	(0.169)	(0.0329)
Childhood Stunting	-0.357***	-0.259**	-0.247*	-0.233*	0.0110
	(0.0965)	(0.128)	(0.128)	(0.129)	(0.0285)
Test Variables					
Sugar Consumption		0.135*		0.104	0.0378
		(0.0700)		(0.0712)	(0.0339)
Fat Consumption			0.162*	0.129	0.0190
			(0.0952)	(0.0987)	(0.0578)
Control Variables					
GDP					0.266
					(0.169)
% Literate					0.0248
					(0.0310)
Population					-0.339
					(0.284)
Year					0.0329***
					(0.00346)
Constant	-0.331***	-0.346***	-0.390***	-0.396***	-66.01***
	(0.0907)	(0.113)	(0.115)	(0.120)	(6.913)
Observations	713	470	470	470	372
Number of id	71	46	46	46	42
Rho	0.949	0.944	0.946	0.947	0.991
Between Rsq	0.630	0.657	0.616	0.575	0.440
Within Rsq	0.338	0.349	0.352	0.371	0.891
Overall Rsq	0.529	0.597	0.534	0.491	0.361

Notes: Standardized regression coefficients. Robust standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 9
Random Effects Analysis on Obesity Prevalence,
1990 – 2007

Variables	1	2	3	4	5	6	7
% in Urban Slum	-0.925***	-0.968***	-0.751***	-0.763***	-0.908***	-0.976***	-0.961***
	(0.279)	(0.348)	(0.291)	(0.294)	(0.281)	(0.340)	(0.345)
Childhood Stunting	0.761***	0.760**	0.601**	0.676***	0.762**	0.783**	0.756**
	(0.295)	(0.324)	(0.275)	(0.253)	(0.303)	(0.314)	(0.299)
Control Variables							
GDP	1.664*	1.227*	1.627*	1.790*	1.446	1.648*	1.900*
	(0.951)	(0.657)	(0.902)	(0.923)	(0.981)	(0.983)	(1.046)
% Literate	0.0989	0.0306	0.242	0.112	-0.00487	0.0849	0.0893
	(0.285)	(0.318)	(0.336)	(0.358)	(0.300)	(0.292)	(0.308)
Population	-2.433*	-1.675***	-2.533**	-2.609**	-2.228*	-2.430*	-2.496**
	(1.275)	(0.625)	(1.254)	(1.238)	(1.303)	(1.315)	(1.241)
Food Consumption	-0.0542	-0.201	0.296	0.268	0.0117	-0.0502	-0.0819
	(0.485)	(0.616)	(0.590)	(0.573)	(0.494)	(0.480)	(0.567)
Year	-0.0339*	-0.0296	-0.0406*	-0.0337	-0.0364*	-0.0346*	-0.0394**
	(0.0191)	(0.0204)	(0.0236)	(0.0214)	(0.0211)	(0.0194)	(0.0198)
Test Variables							
Foreign Investment		0.0271					
		(0.0992)					
Food Imports			0.0763				
			(0.434)				
Public Health Expenditure				0.0879			
				(0.150)			
Value-Added Agriculture					-0.252		
					(0.195)		
Female Labor Force						0.128	
						(0.377)	
Wealthy Status Dummy							-0.650
							(0.844)
Constant	68.68*	59.62	82.12*	68.44	73.61*	69.93*	80.09**
	(37.78)	(40.61)	(47.02)	(42.44)	(41.70)	(38.53)	(39.35)
Observations	37	34	36	35	37	37	37
Number of id	13	10	12	13	13	13	13
Rho	0.973	0.954	0.927	0.952	0.974	0.976	0.968
Between Rsq	0.520	0.669	0.592	0.644	0.534	0.513	0.539
Within Rsq	0.500	0.528	0.388	0.411	0.504	0.502	0.488
Overall Rsq	0.638	0.693	0.687	0.708	0.643	0.636	0.666

Notes: Standardized regression coefficients. Robust standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 10
Random Effects Analysis on Average BMI,
1990 – 2007

Variables	1	2	3	4	5	6	7
% in Urban Slum	-0.0787*	-0.0584	-0.0702	-0.0533	-0.0805*	-0.0780*	-0.0859*
	(0.0476)	(0.0464)	(0.0458)	(0.0342)	(0.0471)	(0.0457)	(0.0472)
Childhood Stunting	0.00156	0.00120	0.00130	0.0177	0.0144	0.00152	-0.00110
	(0.0349)	(0.0393)	(0.0338)	(0.0275)	(0.0337)	(0.0321)	(0.0341)
Control Variables							
GDP	0.502***	0.516***	0.362**	0.472***	0.394***	0.599***	0.484***
	(0.144)	(0.168)	(0.144)	(0.132)	(0.150)	(0.169)	(0.142)
% Literate	-0.00320	0.00522	-0.00174	-0.0259	-0.00466	-0.00506	-0.00401
	(0.0175)	(0.0169)	(0.0179)	(0.0173)	(0.0208)	(0.0167)	(0.0175)
Population	-0.615***	-0.595**	-0.555***	-0.558***	-0.564***	-0.678***	-0.610***
	(0.206)	(0.234)	(0.211)	(0.171)	(0.211)	(0.218)	(0.202)
Food Consumption	0.00332	0.0262	-0.0133	0.102*	0.0520	-0.0154	0.00657
	(0.0771)	(0.0734)	(0.0705)	(0.0532)	(0.0723)	(0.0759)	(0.0773)
Year	0.0312***	0.0308***	0.0337***	0.0341***	0.0301***	0.0290***	0.0308***
	(0.00300)	(0.00300)	(0.00292)	(0.00319)	(0.00329)	(0.00322)	(0.00296)
Test Variables							
Foreign Investment		-0.0208					
		(0.0160)					
Food Imports			0.130***				
			(0.0388)				
Public Health Expenditure				0.0181			
				(0.0200)			
Value-Added Agriculture					-0.0639		
					(0.0462)		
Female Labor Force						0.174*	
						(0.105)	
Wealthy Status Dummy							0.0307
							(0.0264)
Constant	-62.55***	-61.71***	-67.37***	-68.20***	-60.24***	-58.11***	-61.69***
	(5.995)	(5.995)	(5.851)	(6.368)	(6.574)	(6.444)	(5.916)
Observations	555	510	533	463	530	555	555
Number of id	61	57	59	60	61	61	61
Rho	0.989	0.990	0.990	0.993	0.991	0.989	0.989
Between Rsq	0.739	0.767	0.751	0.780	0.735	0.641	0.742
Within Rsq	0.907	0.909	0.914	0.922	0.905	0.911	0.907
Overall Rsq	0.667	0.687	0.644	0.695	0.667	0.612	0.672

Notes: Standardized regression coefficients. Robust standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 11
Random Effects of Key Slope-Dummy Variables
on Obesity Prevalence, 1990 – 2007

Variables	1	2
% in Urban Slums	-0.238	-0.944**
	(0.612)	(0.392)
Childhood Stunting	0.546***	0.299
	(0.144)	(0.514)
Regional Dummies		
Africa	0.871	1.020
	(0.642)	(0.846)
Latin America	1.627***	1.609***
	(0.364)	(0.268)
Interactions		
Slums X Africa	-0.714	-0.150
	(0.662)	(0.510)
Stunting X Africa	-1.434**	0.425
	(0.660)	(0.873)
Slums X Latin	0.0414	0.445
America	(0.645)	(0.479)
Stunting X Latin	-0.351	-0.00293
America	(0.228)	(0.616)
Control Variables		
GDP		-0.403*
		(0.241)
% Literate		0.0750
		(0.401)
Year		-0.0397
		(0.0300)
Constant	-1.193***	78.47
	(0.350)	(59.92)
Observations	51	37
Number of id	16	13
Rho	0.934	0.928
Between Rsq	0.569	0.607
Within Rsq	0.452	0.420
Overall Rsq	0.345	0.641

Notes: Standardized regression coefficients.
Robust standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 12
Random Effects of GDP Slope-Dummy Variables
on Obesity Prevalence, 1990 – 2007

Variables	1	2
% in Urban Slums	-0.683***	-0.803***
	(0.241)	(0.306)
Childhood Stunting	0.379**	0.113
	(0.181)	(0.364)
Control Variables		
GDP	-1.387***	-0.600***
	(0.522)	(0.167)
% Literate		0.241
		(0.329)
Year		-0.0550**
		(0.0247)
Region Dummy		
Africa	-0.665	-0.504
	(0.814)	(0.891)
Latin America	0.497	0.545
	(0.810)	(0.692)
GDPX Africa	2.691***	1.983***
	(0.645)	(0.496)
GDP X Latin America	1.327***	0.476
	(0.509)	(0.306)
Constant	-0.365	109.2**
	(0.861)	-49.35
Observations	52	37
Number of id	17	13
Rho	0.975	0.848
Between Rsq	0.601	0.706
Within Rsq	0.506	0.262
Overall Rsq	0.384	0.665

Notes: Standardized regression coefficients.
Robust standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 13
Random Effects Analysis on Food Consumption, 1990-2007

Variables	1	2	3	4	5	6	7	8	9
Urban slum Controls	-0.266*** (0.0535)	0.0129 (0.0629)	-0.0125 (0.0648)	0.0231 (0.0623)	0.0194 (0.0523)	0.0329 (0.0496)	0.0118 (0.0612)	0.0216 (0.0610)	0.0115 (0.0631)
GDP		0.796*** (0.153)	0.813*** (0.163)	0.751*** (0.171)	0.672*** (0.127)	0.841*** (0.165)	0.868*** (0.154)	0.829*** (0.150)	0.776*** (0.153)
% Literate		0.0379 (0.0323)	0.0116 (0.0333)	0.0138 (0.0304)	0.0228 (0.0291)	0.0351 (0.0352)	0.0360 (0.0318)	0.0407 (0.0328)	0.0403 (0.0315)
Population		-0.657*** (0.143)	-0.649*** (0.157)	-0.660*** (0.144)	-0.500*** (0.120)	-0.692*** (0.150)	-0.703*** (0.144)	-0.664*** (0.139)	-0.638*** (0.151)
Year		0.0069** (0.00326)	0.00731* (0.00377)	0.00945*** (0.00310)	0.00987*** (0.00298)	0.00662** (0.00320)	0.00534 (0.00375)	0.00722** (0.00328)	0.00868** (0.00358)
Test Variables									
Foreign Investment			-0.00292 (0.0270)						
Food imports				0.0645 (0.0633)					
Public Health expenditure					-0.00849 (0.0338)				
Value-Added Agriculture						-0.0220 (0.0748)			
Female labor force							0.143 (0.143)		
Wealthy Status Dummy								-0.0569 (0.0668)	
Political Regime									-0.00632** (0.00261)
Constant	-0.258*** (0.0435)	-13.75** (6.513)	-14.46* (7.534)	-18.74*** (6.199)	-19.67*** (5.952)	-13.04** (6.396)	-10.50 (7.485)	-14.24** (6.575)	-17.18** (7.152)
Obs	862	705	632	671	539	672	704	705	702
No. of id	80	69	66	67	70	67	68	69	66
Rho	0.884	0.902	0.911	0.906	0.947	0.910	0.905	0.902	0.902
Btw Rsq	0.389	0.519	0.523	0.527	0.511	0.515	0.447	0.518	0.546
W/in Rsq	0.185	0.429	0.423	0.422	0.472	0.464	0.445	0.433	0.440
Total Rsq	0.518	0.522	0.533	0.513	0.523	0.538	0.487	0.522	0.54

Notes: Standardized regression coefficients. Robust standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Figure 1. Obesity Prevalence by Geographical Region, 1980 – 2009
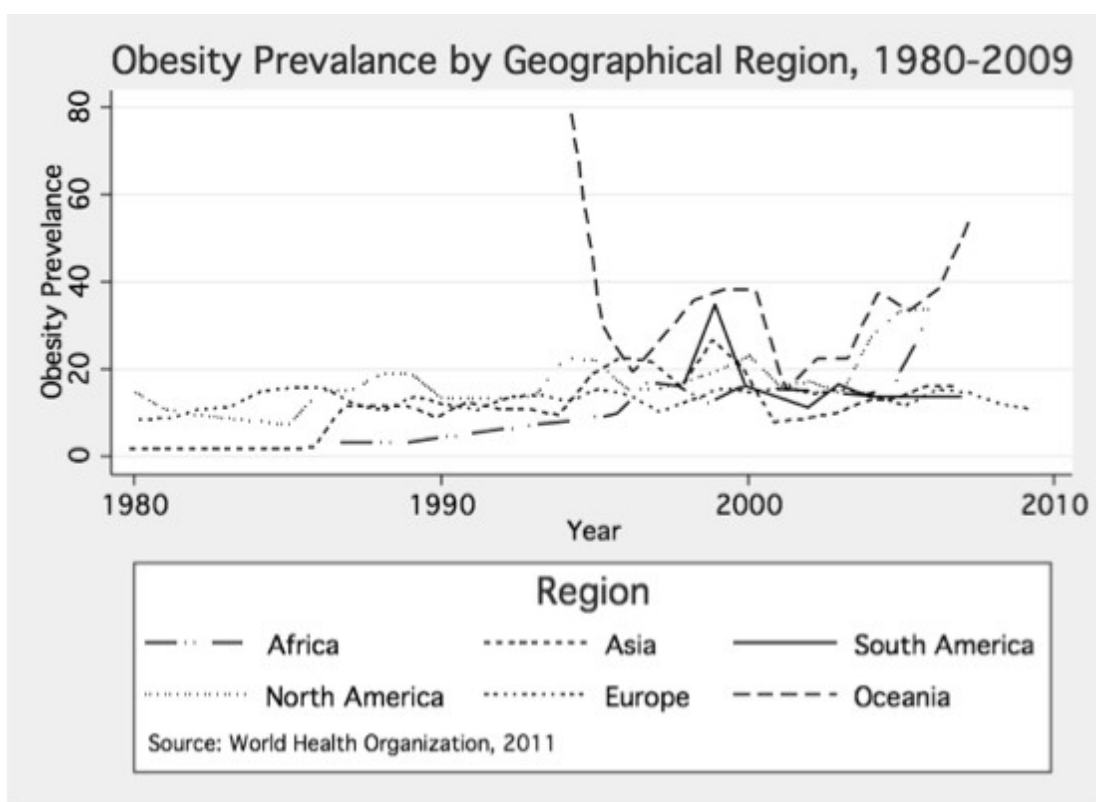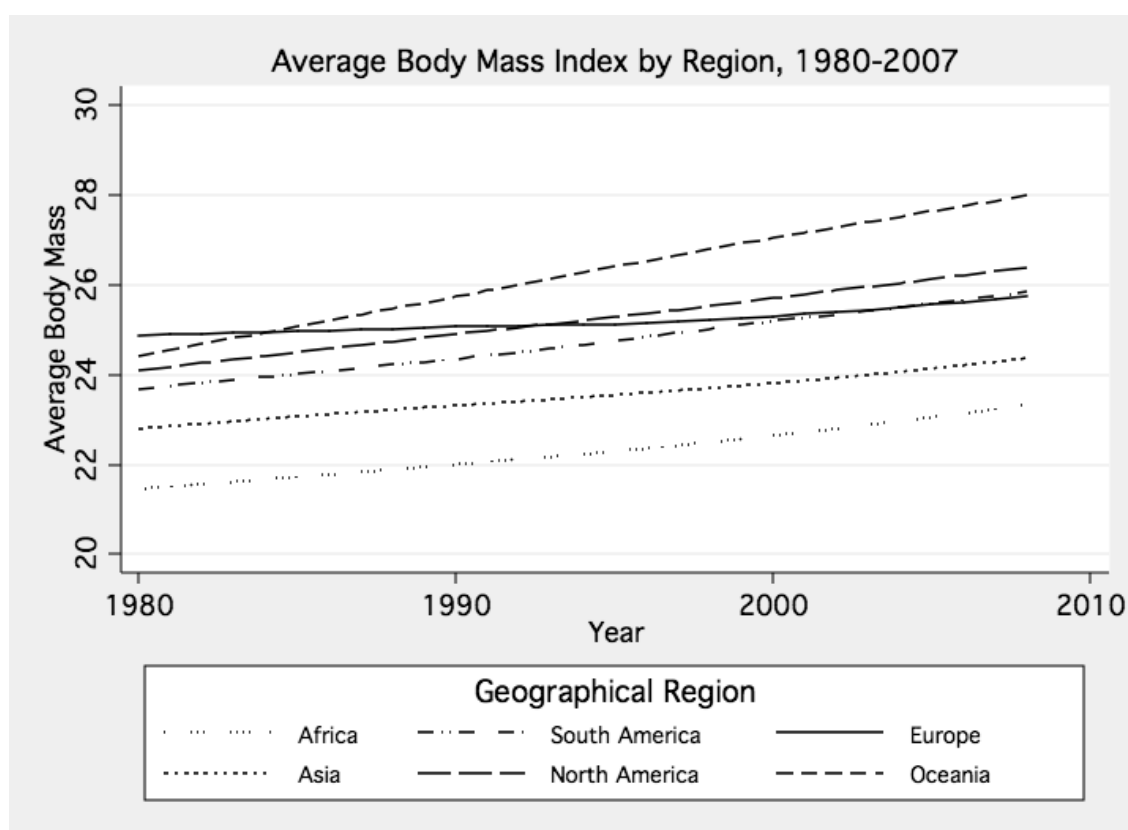
Figure 2. Average Body Mass Index by Region, 1980 – 2007
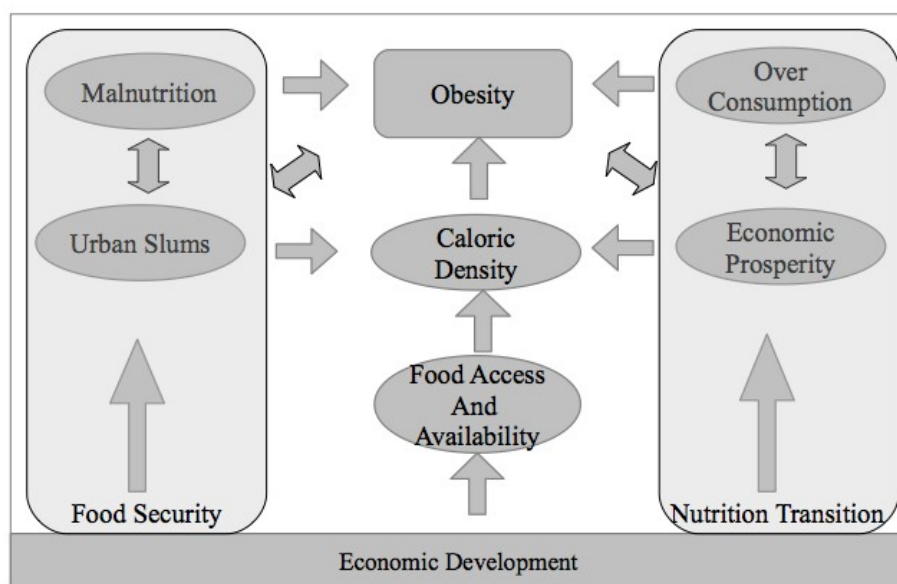
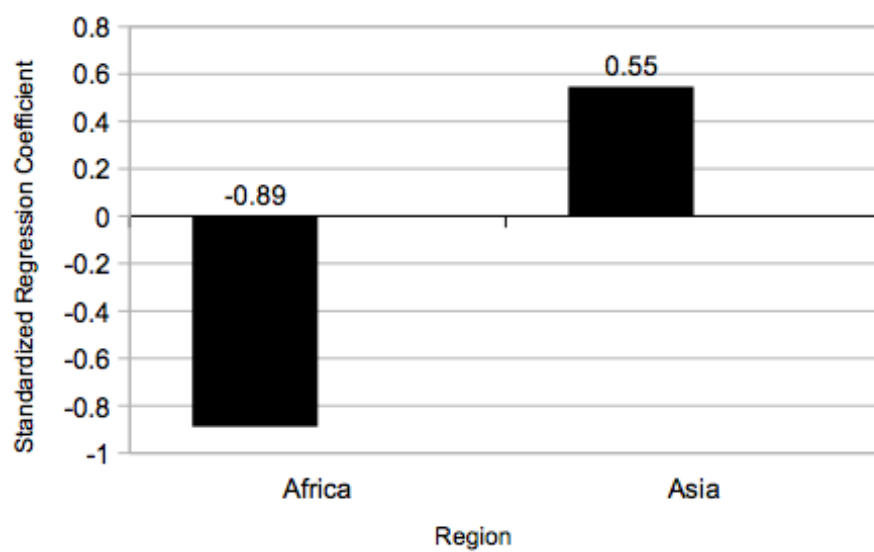Figure 3. Hypothesized Routes to Obesity by Nutrition and Food Security

Figure 4. Effect of Childhood Stunting on Obesity Prevalence, 1990 – 2007; Regression Coefficients by Region, Asia Reference
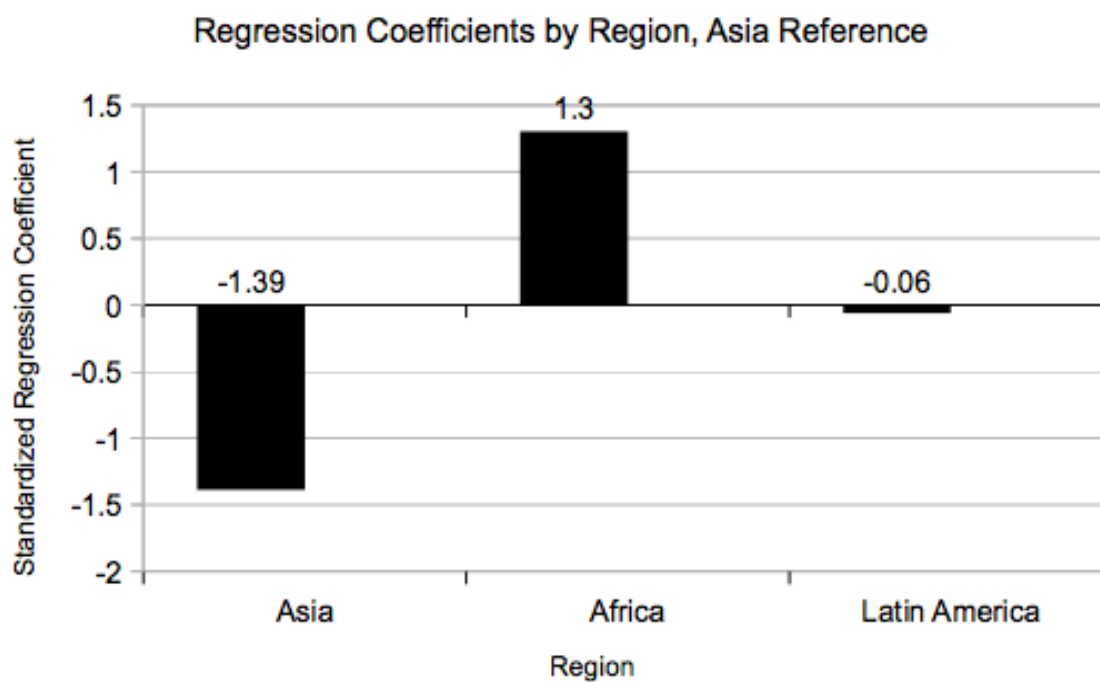
Figure 5. Effect of GDP on Obesity Prevalence, 1990 – 2007; Regression Coefficients by Region, Asia Reference

CHAPTER 2

GLOBALIZATION AND OBESITY: TESTING MECHANISMS OF

GLOBAL CONVERGENCE THROUGH BODY MASS

Introduction

In 2008, 1.4 billion adults were overweight and 65 percent of the world's population lives in countries where being overweight kills more people than being underweight (WHO 2012). While the absolute number of obese and overweight was greater among upper-middle and high-income countries, the relative growth of overweight prevalence was greater among lower-middle and low-income countries (WHO 2011).

Termed "globesity" (Dietel 2002), global obesity has formally been referred to as the result of global organizations which, intentional or not, influences body size (Sobal and McIntosh 2009). The growth of social, political and economic global interconnectivity has increased the ability to market food products around the world in ways that were hitherto impossible. Essentially, the expanse of the global marketplace that has integrated the world economy has provided excess calories, inexpensively, to a wider array of people than ever before. In this light, economic influences play important roles in "globesity" showing how developing countries are facing emerging obesity

epidemics while also explaining the persistence of obesity among wealthy ones. Behavioral and economic interventions such as reducing fast food consumption among children or soft drink taxes have shown limited progress in curtailing obesity (Bluford, Sherry and Scanlon 2007; Kropski, Keckley and Jensen 2008) prompting the development of costly anti-obesity medications.

However, civil society organizations – which are known as nonstate, not for profit, voluntary organizations – have offered a compelling means to combat social problems throughout the world (CSI 2002b). Social advocacy such as policy strategy research and analysis proposed by civil society organizations increases problem visibility and is argued to prompt action from states and corporate entities to formalize coherent problem resolution strategies. Civil society can be instrumental in combatting obesity as it is able to influence "upstream" factors like dietary information or food policy rather than concentrating on individual "downstream" factors, such as medical intervention or behavior modification.

Much of globalization literature seeks to explain economic, cultural and social convergence, in which obesity has arguably been included. However, cross-national obesity scholarship has not been strong in applying formal globalization theory. For this reason, I attempt to view the rising obesity epidemic as a means of exploring the value, if any, of using competing explanations of global convergence to explore emerging trends in rising body mass. Economic integration and world society theories of globalization can be useful in understanding "globesity." These views of structural convergence posit the nature of obesity differently. First, the expanse of economic interconnectedness provides a clear explanation for common obesogenic environments. The influx of inexpensive

processed, energy dense foods as a result of economic development is argued to promote obesity. The world society perspective, though, takes the view that structural convergence occurs through labeling social problems brought on by a more densely integrated world culture. Addressing social problems then comes with other sets of converging factors. This view suggests that civil society is in position to prevent obesity from "spreading" among vulnerable populations. These theories posit different obesity trajectories based on their respective explanations of global convergence. In this paper, I essentially ask which type of organization – McDonald's or Doctors Without Borders – has a greater role in body mass. Body mass is a new facet of global convergence that has garnered little attention in this regard. Findings here stand to inform our perspectives of both globalization theory and global obesity trends.

Conceptualizing globalization has been extremely diverse in research literature. I concentrate on economic and cultural globalization as a means of exploring pathways of the global obesity epidemic. Though these two broad conceptualizations of globalization are not presumed to be independent, studying them separately provides a useful means of identifying processes of convergence among them and how they may have influenced each other (Chase-Dunn and Jorgenson 2007; Jorgenson and Kick 2003). Comparing different conceptualizations will aide in exploring how these influences may have complementary or competing effects on body mass.

Economic Globalization

Broadly speaking, economic globalization can be conceptualized by economic exchange and flows of goods, services, people, information, and capital across national

boundaries. Empirically, it has been operationalized as the sum of exports, imports, and inward and outward (direct portfolio) investment, which are themselves indicators of networked flows of goods and capital (Brady, Beckfield and Zhao 2007). Other authors have described globalization as the proportion of all invested capital in the world that is owned by nonnationals (Chase-Dunn and Jorgenson 2007), or similarly, the interrelationships of markets, finance, goods and services, and networks created by transnational corporations (Chase-Dunn 1999). Though somewhat different, these conceptualizations emphasize the overall increase in breadth and depth of economic endeavors and the capitalization of production and mechanization among all nations (Chase-Dunn 1989). The aim of this approach is to specify the processes by which world networks are empirically growing more interconnected in ways reaching new levels not seen in this century (Brady, Beckfield and Zhao 2007).

The current wave of economic globalization usually describes the processes of neoliberalism that shrinks obstacles to trade which encourages the assumption that free exchange of goods brings with it a host of social and economic benefits (Chase-Dunn 1999). As Arrighi (2007) explains, the advent of the most modern iteration of structural globalization was brought on by two relatively simultaneous ideological transitions. First, the welfare systems such as New Deal era initiatives in wealthy nations were divested to free up domestic capital. By diverting funds to foreign countries, capital was invested abroad in similar privatization programs through FDI which increased the volume and scope of international financial instruments and investments. The second transition, Arrighi argues, was the liquidation of state-sponsored industrialization among the global south. As part of the privatization goals of modern nations, state sponsored industry was

argued to hinder economic development. It was reasoned that in dismantling these sectors, private capital could then provide labor and production without increasing state financial insolvency to borrow funds for industrialization (Gilpin 2001; Stiglitz 2003). With a similar rationale, global economic emphases shifted from developing strong national industrialization to strong international trade.

The overall outcomes of the neoliberal shift in economic policy throughout the globe have been widely debated (Gilpin 2001), though, arguably the most profound change throughout the global economy has been the proliferation of FDI and trade openness. From 1960 to 2000 trade openness measured by exports and imports as a percentage of GDP grew from 43.8 to 80.5 percent (Brady, Beckfield and Zhao 2007). By another measure, the spread of transnational corporations grew from 7,000 in 1970 to 600,000 in 2000 (Robinson 2004). Foreign investment has grown to be the hallmark of economic globalization after 1970 (Chase-Dunn 1975; Chase-Dunn and Jorgenson 2007; Kentor 2005).

The political reality of the 1980s was dictated by Margaret Thatcher's thesis that in the global economy, the only alternative was economic liberalization (Arrighi 2007). The logic of capital liberalization dictated "Washington Consensus" economic policies for an export oriented, privatized, and deregulated economy enforced by international lenders that are largely supported by wealthy countries (Arrighi 2007; Babb 2005; Chase-Dunn 1999). The general argument goes, that in terms of health and welfare, trade liberalization – including membership in trade federations and FDI– has served to constrain the power of the nation-state in meeting the needs of the public (McMichael 2003; Shaffer 2005; Stiglitz 2003).

Trade Liberalization and Health

Global economic liberalism does not have uniform effects on population health. A major focus in literature emphasizes the role of globalization on economic inequality as a means of explaining the consistent relationship between income and health with evidence showing both positive and negative outcomes (Salomon and Murray 2002). For instance, The Millennium Development Fund is ahead of schedule by a factor of 2 in achieving its goal on reducing urban slums (though the absolute number of slum dwellers has increased by 58 million people a year between 2000 and 2010, with concentrated urban slum growth across Africa and Asia) (UNHABITAT 2010). Global average life expectancy and child mortality have improved over time, but those gains have also not been consistent particularly among Sub-Saharan Africa and post-Soviet Russia (Bonita, Irwin and Beaglehole 2007; McMichael et al. 2004). The means through which liberalization affects health – primarily among lower-income countries – is in access to and quality of care, and in affecting the price of drugs and equipment (Breman and Shelton 2007; Schrecker and Labonte 2007). Breman and Shelton point out that it is important to consider the individual features of liberalization to see its overall effects on national health.

For instance, FDI is one area that has a diverse track record. The evidence on the effects of FDI is varied, though several points of consensus have emerged in recent years. FDI generally has a stagnating effect on long-term domestic economic growth in low-income countries and plays an important role in generating income inequality (Alderson and Nielsen 1999; Kentor and Boswell 2003). Though with respect to health, FDI increases commercialization of the health care sector, and the attendant risks that

accompany commercialization (Smith 2005). For example, the disadvantages of privatized health care delivery include a number of negative externalities such as higher financial burden of healthcare among the poor, uncoordinated services across areas, higher concentration of delivery in higher-income areas. Negative attitudes towards privatized health care is most salient if the delivery is perceived to be through a large transnational corporation (Hampton and Higman 2009). Still, more importantly, the extent to which a country's health system is commercial rather than foreign plays a larger role than FDI in health outcomes. The power imbalance introduced through the health sector financing involves the relative strength of national regulatory systems and potential investors. "For-profit" and "not-for-profit" care can be equally damaging if regulations regarding care are absent. Through this lens, health service itself is relatively unaffected by globalization, while the economic conditions that promote a healthy environment and healthcare access tends to benefit the wealthy (Smith 2005).

The other area where liberalization has influenced population health is nutrition, but not uniformly. Primarily during the later decades of the 20th century, international lending organizations like the World Bank used carrot and stick approaches to economic development among borrowing countries requiring macro-economic policy revision. These "adjustments" typically involved revising public spending on a wide variety of services from state-sponsored industry to public development projects. Most importantly it required opening state borders to private investment which has had a myriad effects. One consequence has arguably been an increase in poverty (Gilpin 2001; McMichael 1996; McMichael 2003; Stiglitz 2003). Malnutrition among developing countries generally increased over the short-term of liberalization, exacerbated by fewer social

safety nets from structural adjustment, whereas in the long-term, malnutrition outcomes have improved by some measures (Breman and Shelton 2007). However, in recent years, with energy crises looming, higher proportions of grains dedicated to biofuel production have increased food price volatility. Reports suggest that more people suffer from chronic hunger than ever before, not from lower food availability but because of lower food access from price increases (Allen and Wilson 2008; FAO 2009). The relationship of caloric intake to the price of food is generally inverted (Evenson and Gollin 2003; Miller and Coble 2008). Bleich et al. (2007) point out that among OECD countries, the 12 percent decrease in food prices from 1980-2002 was associated with an additional 40 calories per person. The issue here is not that the price of food is going up or down but the relative changes in price for whom. In the well-developed OECD compared to the less developed global south, there are very different hunger outcomes based on the relative pace and expanse of a nation's economic development. Hence, FDI has contributed to over-nutrition in some areas and under-nutrition in others.

Calories, overall though, have become more widely available over the 20th century. Improvements in agricultural production efficiency and increases in the market share of agricultural products have reduced wastage through several economic practices. First, historically, agricultural production was subject to boom and bust years based on the growing season. To control price fluctuations, farm policy since the 1930s in the US, (and to a lesser extent in Europe) has provided income supplements to growers for storing commodities rather than selling them (Guthman 2011). Production controls to reduce agricultural production on suboptimal land also serve to lower food costs (Allen and Wilson 2008). Subsequently, controls for production, storing, and marketing basic

commodities lowered the cost of cattle feed making animal products more affordable (Tillotson 2004). Second, "The Green Revolution" also characterized higher foodstuff output through farm mechanization, pesticides, fertilizers, and agricultural bioengineering. Research indicates the accelerated use of pesticides has contributed to insect tolerance leading to what some authors have termed the "pesticide treadmill" (Jorgenson and Kuykendall 2008). Pesticides have been linked to a wide array of non-communicable diseases from neurodegenerative diseases to various forms of cancer (Alavanja, Hoppin and Kamel 2004; Giordano and Costa 2012). Some estimates suggest that worldwide caloric intake would have been roughly 14 percent lower had the Green Revolution not taken place (Evenson and Gollin 2003). Improved agricultural efficiency has been a hallmark of economic development for low-income countries in providing sufficient food for its public (UN 2005).

Higher productivity and efficiency led to other means to put excess food production to use. The "Food for Peace" program implemented for a time during the Cold War exported excess US grains to politically unaligned countries to expand US agricultural markets and decrease sympathy towards the Soviet Union (Nestle 2002). It also served to provide for the world's hungry (Critser 2003; Guthman 2011). Improvements in efficiency have also contributed to wider market niches for crop-based sweeteners like High Fructose Corn Syrup (HFCS) which have largely supplanted cane sugar in soft drinks, thus lowering production costs and increasing consumption (McCrory, Suen and Roberts 2002; Popkin 2006). Food additives more generally have become commonplace in a variety of foods and have lowered production costs but also increased caloric density. With more efficient production, more bread for instance, can be produced at a lower cost,

but with food additives, foods can also be made more palatable which increases purchases. In particular, the trend of "value" meals at fast food restaurants arguably has taken advantage of satiability paradoxes – more palatable foods accelerate the return of hunger and increase overall energy intake (McCrory, Suen and Roberts 2002). The logic of "supersizing" exploits the relative cost of food production by matching low profit foods like beef with higher profit foods such as soft drinks. For the consumer, one can add more quantity for relatively little cost, while for the producer, cheap foods subsidize more expensive foods, thus increasing the meal's "value" (Critser 2003). Indeed, fast food and soft drink brand recognition is increasing worldwide as more urban populations seek out Western style foods high in fat and sugar because they are associated with being more "modern" (Chopra 2002).

Cheaper calories available on the world market represents a major contributor to global obesity. The amount of total foreign exchange concerned with manufacture, service or sales of food products among the top 100 transnational corporations from 1990-2001 has reportedly increased by 263 percent (Hawkes 2005; Rayner et al. 2006). Initial results of research into FDI penetration suggested a loss of 650 calories in penetrated countries due to the harmful effects of liberalization (Wimberly and Bello 1992). However, after the 1980s, the degree of investment increased caloric intake in host countries through wider processed food availability (Hawkes 2005; Rayner et al. 2006). US FDI in food processing grew from $9 billion in 1980 to $36 billion in 2000, where sales in those companies grew from $39.2 billion in 1982 to $150 billion in 2000 (Boling and Somwaru 2001; Hawkes 2006). Increases in FDI amount and concentration have increased the number total calories available to the average world citizen.

In addition to FDI, the global trade environment has introduced conditions favorable to rising obesity rates. Reductions in agricultural tariffs as targeted by the World Trade Organization in 1994 have encouraged domestic producer subsidies in highly developed nations and disincentivised agricultural producers in poorer countries (Rayner et al. 2006). For instance, in Central America and Mexico, Thow and colleagues (2009) have argued that the signing of the 1994 North American Free Trade Agreement and the 2004 US-Central American Free Trade Agreement, facilitated rising availability and consumption of US meat, dairy products, processed foods, as well as imported fruits and snack foods despite better growing conditions among Central American countries (Hawkes 2006; Rayner et al. 2006; Thow and Hawkes 2009). In the case of public health, trade agreements are more focused on property rights, patents and sanitation than nutrition (Rayner et al. 2006). As it stands, Word Trade Organization (WTO) agreements are currently based on relevant international standards of food trade set by the Food and Agriculture Organization of the UN and World Health Organization (FAO/WHO) Codex Alimentarius – the legal precedent of the FAO/WHO Food Standards Program protecting the health of consumers and ensuring fair practices of food trade (FAO/WHO 2007). Under the provision of "like products," it was reasoned that a country cannot exclude a good produced in a foreign country even if they deem the production of that good involves a risk to health or society (Chopra 2002). In dispute settlements, the WTO body prioritizes trade interests at the expense of what is a "necessary health measure" so much so that "like" foods may be those higher in salt, fats or sugars (Chopra 2002).

These global trade practices have had a number of consequences. First, they have increased food availability and lowered costs. They have served to find market niches for

extra calories that would otherwise not be consumed. Further, they have provided the world with new diets. Essentially, international trade and food production has created a fix for the basic problem of under-consumption (Guthman and DuPuis 2006). Inelastic demand suggests that food consumption cannot be overproduced because consumption is essentially fixed and excess food will only be wasted. However, economic liberalism responded by creating processed foods ready for the world market higher in calories than more "natural" foods. For example, fast food is 65 percent more energy dense than the average diet (Cummins and Macintyre 2006). Furthermore, energy dense diets are increasingly marketed to world consumers in ways that put individual health at risk. Overall, global food consumption has risen by an average 400 kcalorie/person/day from 1969-2001 (Kearney 2010). Foods with the greatest increases in consumption are vegetable oils, animal products, and sugars (Chopra 2002; Tullao 2002).

The above discussion has addressed how neoliberalism contributes a number of features to obesity risk. First, the rise of FDI has increased disproportionate health burdens to lower incomes through lower social welfare and higher health care delivery costs. Second, the evolution of food processing has served to disproportionately favor wealthy nation's agricultural production through subsidy and market access while simultaneously lowering costs of energy dense foods for sale. This process is known as "Coca-colonization," or "McDonaldization," and is used to describe economic convergence and homogenization (Ritzer 1983). Still, other evidence indicates that local contexts have incorporated global foods through a "glocal" transformation (Matejowsky 2007; Ritzer 2003). As Hawkes (2006) demonstrates, a convergence-divergence duality is likely occurring among developing nations exacerbating an uneven dietary transition

between the rich and poor: high-income groups may afford novel, yet healthy foods while income constraints produce dietary convergences coinciding with obesogenic foods.

In this light, a precipitating cause of the global obesity epidemic is leveled at neoliberalism in that it has provided the marketability of additional calories, but also through increasing vulnerability among different income strata. In the next section, I discuss how the global culture among civil society organizations stands to mediate obesity outcomes by increasing advocacy and health as a human right. The main focus of world society theory has focused on building international legitimacy through membership in the world society. This membership has recently emphasized improving advocacy among vulnerable groups. Civil society networks have been viewed as the premier means of improving global health in nations of all economic strata and by international organizations like the WHO and UN. I argue that civil society networks can play a vital role in alleviating obesogenic environments through membership in the world society.

<center>World Society</center>

One surprising aspect of globalization has been the rise of obesity across diverse nations. It is well known that the United States has a growing obesity epidemic, but unexpectedly, so too does Ghana, Malaysia, Brazil and many other developing nations (Bezerra and Sichieri 2009; Drake, Tawiah and Badasu 2010; Khor 2012). Globalization literature contains strong themes about the forces which unify the world's diversity. Indeed, many theories of globalization ask the question, "What, if any, are the channels through which homogenization occur?" Meyer et al. (1997) suggest that using global

culture to explain institutional and societal similarities best illustrates how societies can be structurally similar on unexpected dimensions and how change can occur in unexpectedly similar ways across them. Using this lens, I examine the effects of civil society networks in the global obesity epidemic.

Civil Society Organizations (CSOs) have been a long-term presence on the global scene – as early as 1839 with the British and Foreign Anti-Slavery Society – but their proliferation has accelerated since the early 1990s (Anheier, Glasius and Kaldor 2001). Subgroups of CSOs such as International Nongovernmental Organizations (INGOs) and Nongovernmental Organizations (NGOS) have grown a great deal throughout the 20th century illustrating vast diversity in focii. Some examples of CSOs include organizations as expansive as the WHO or the more narrowed European Group of Research on Elderly Physical Activity. As of 2004, the number of international INGOs had grown 20 fold since 1950, and 65 percent of NGOs had formed since 1960 (Salamon 1994; Smith and Wiest 2005). The wide expanse of CSO membership and its relative diffusion of Western culture over the last century has concentrated and diffused from wealthy nations (Beckfield 2003). Central to world polity theory, this diffusion has been the primary medium in transmitting bureaucratic structures infused with the values of wealthy nations. Presumably, civil society membership is directly correlated with integration in the world society acting through the number of CSO networks functioning in a given country.

Neo-institutional views of globalization have used international culture to assert how the influence of global actors can drive similarities between societies. In this view, normative functions of the nation state are institutionalized such that action and discourse

are based on recipes, scripts and rules inherited from modes of behavior that have been perceived to be effective by global actors translated across a variety of national contexts (Chabbot 1997). Over time, international actors are legitimized by the international community through their support of the rule of law, frames of reference based in scientific rationality and goals of improving basic human rights. State functions are essentially propped up by the self-reinforced values of international models permeating the national arena in ways that reflect principles common to the international order. Thus, to maintain legitimate international sovereign identities, actors must comply with models of legitimate actions which in themselves entail adhering to world values (Meyer et al. 1997). Legitimate global actors are supported and reinforced by other legitimizing actors. Meyer (2000) argues

> the drive to creation of common globalized models of instrumental culture, in other words, is produced by a system that defines actorhood as a core principle. It feeds on itself: each step forward in globalization produces more and more definitions of the requirements and responsibilities of effective actorhood. (p 238)

The roles of international actors, then, are legitimized by their adherence to organizational, structural, and ideological norms of the international community (Meyer et al. 1997).

According to this view, in the current era of globalization, the expanding nature of the organizational and institutional structure of the world polity intensifies the influence of global discourse on nations (Schofer and Hironaka 2005). World polity scholars often downplay hierarchical polity membership structures, arguing that because of their decentralized "stateless" influence, transmitters of cultural policy scripts are distributed through the world polity of Intergovernmental Organizations (IGOs) like the UN and

through diffuse civil society membership in NGOs and INGOs (Beckfield 2003; Beckfield 2008). As Cole (2011) argues, early world polity work formed out of a need to respond to the economistic terms of global hierarchical structuring; it therefore preserved the state-centric theorizing, but as decentralized forms of "world society" have become more prolific, the theoretical emphasis has shifted from processes of state actors to processes of nonstate actors to accomplish cultural diffusion independent of the state, primarily through IGOs and INGOs. In many respects, today the theoretical emphasis of the world society perspective includes examining explicitly nonstate actors and their impacts on outcomes independent of the state itself. Though CSOs typically lack the coercive authority of the state, the authority of the world polity derives primarily from its emphasis on scientific expertise and the common good (Boli and Thomas 1997). The International Obesity Task Force (IOTF) and the WHO are currently important voices on global obesity. NGOs have become the contractor of choice to provide aid, information, and services, rather than cash donations to individual states (Reimann 2006).

Smith (1997) reports transnational social movement organizations have been increasingly linked with world polity and civil society networks. However, few researchers have offered coherent distinctions between civil society organizations and formal world polity bodies, though some research has indicated differences exist. For example, with respect to human rights abuses, Tsutsui and Wotipka (2004) found that IGOs and NGOs played different roles: NGOs primarily drove global human rights expansion through flexibility and lack of concern over state sovereignty, whereas IGOs legitimize efforts by the state. Wealthy Western countries are more integrated into IGOs and INGOs than are poorer, non-Western states, though IGO membership shows a more

equal membership distribution (Beckfield 2008). Cole (2011) argues, in essence, IGO membership indicates integration in the world polity and represents the interests of states, while INGO membership shows the extent of embeddedness in the world society and represents non-statist interests.

Much of the rise of CSOs has been associated with several interrelated global developments that have broadened the space for CSO action. First, in general, neoliberal pressures on national budgets has led to increased reliance on independent organizations in light of the financial weakening of the welfare state (Anheier, Glasius and Kaldor 2001; Cohen, Küpçü and Khana 2008; CSI 2002a; Salamon 1994). For example, with shortfalls resulting from budget cuts in the 1980s and 90s, the WHO increasingly turned to public-private partnerships with NGOs to accomplish mission goals (Brown, Cueto and Fee 2006). Second, CSOs have increased where centralized authority of the state produces dissatisfaction in timeliness, cost, or representativeness in policy prescriptions. Third, the demand for public accountability of the state has contributed to the increase in awareness of and memberships in CSOs (CSI 2002a).

These developments have allowed CSOs to carve out particular service niches. As Reimann (2006) puts it, there exists pressure from both the "top down" and "bottom up" for their services. CSOs rely on national governments and IGOs for material and political resources which enable them to function in their respective subject areas. Conversely, intergovernmental organizations and state governments rely on CSOs to fill particular roles such as agenda setting, data acquisition, mobilization of public opinion, and watchdog functions on behalf of the public. A large area recently receiving scholarly attention in the realm of CSO advocacy has been health as a human right (Kickbush

2002; Lee, Sridhar and Patel 2009; London and Schneider 2012; Smith and Wiest 2005).

Viewing health care issues through the lens of human rights provides a counterbalance to economistic or political views which posit health as a matter of meeting welfare needs or national security (Chinkin 2006). By orienting around health as a human right, the determinants of health are seen as a public good, and as such, advocates can concentrate efforts on reducing health inequality among vulnerable groups. Indeed, a major function of CSOs associated with the WHO is in advocacy, lobbying, and disseminating information to build larger alliances for accomplishing health goals (CSI 2002b). While the decoupling of human rights policy enactment and enforcement is relatively common (Cole 2005; Hafner-Burton and Tsutsui 2005), advocacy groups' main avenue of advocacy is in improving human rights outcomes through strong legal accountability (Chapman 2009; Cole 2006; London 2007; London and Schneider 2012). In this case, the mechanisms through which CSOs function presumably holds the state accountable for outcomes in its purview. Civil society performs "watchdog" functions to pressure the state to increase representation, reduce injustices, uphold international treaties or act in some way that behooves a state seeking social, political, or bureaucratic legitimacy in the eyes of its global peers.

The upshot is that civil society networks have gained traction from both national states as well as intergovernmental agencies as a means of combating social problems. International civil society can play a key role in combating worldwide obesity through increasing state accountability, providing technical support, influencing policy responses, or disseminating information to the public (Blas et al. 2008; Bozorgmher 2010). However, up to now, the role of civil society networks in obesity advocacy has been not

been examined despite a growing research body addressing the role of civil society in improving global health.

One potential reason for the lack of social and political advocacy on behalf of the obese is the common notion that obesity is an individual problem. This argument claims obesity is a condition wherein individuals are essentially exercising the right to overeat (DeBoy and Monsilovich 2012). In this light, obesity is reframed as a condition of choice, rather than the result of financial, health, or other constraints. As such, the politics of individual responsibility are used to obscure the intricate relationship of individual-environment interactions of obesity wherein biology permits weight gain, but it is the environment that provokes it (Brownell 2005; Swinburn et al. 2011). Research in the US and Europe shows two varying political responses. In the US, policy positions have become retrenched in the debate outlined above leading to relatively few improvements on the national level but more promising initiatives by individual states and cities (e.g., the proposed New York City ban on soft drinks larger than 16 ounces). The European response has, with limited success, included proposals to limit food advertising to minors and "Eurodiet" initiatives calling for more food labeling information available to consumers (Lang and Rayner 2005). Still, in both instances, Kersh (2009) notes there is virtually no probability of introducing any law that will slow the advance of obesity within the next 10 years. As such, advocacy groups are turning toward judiciary rather than legislative action to represent their interests and they are drawing on civil society to call attention to obesity. For example, the International Size Acceptance Association has, in the past, spoken out against discrimination against the obese and has acted as a forum for attorneys challenging size discrimination (Saguy and Riley 2005). Other judiciary

action has included bringing lawsuits against fast food establishments. This has resulted in publishing nutritional information and other self-imposed industry regulations (Brownell and Warner 2009). In both instances, pressure has come to bear to bring attention to obesity as a societal problem and introduce legal action which codify public policy. Theoretically, world society has operationally posited that CSO connectivity reduces the risk of social problems through the mechanisms of the state. However, change essentially occurs at various levels in society – at the individual, community, or state level based on the effectiveness of advocacy from CSOs.

Social advocacy can be a powerful means to reduce the spread of obesity especially when considering how the state can be mired in political inaction. As Meyer and Jepperson (2000) conclude, the Western emphasis on individualism and personal choice provides expanded space for specialized identities, particularly when traditional forms of individual expression are stymied by the nationstate. This particular brand of individualism lies contrary to that of the individualized focus of obesity discussed above given that the reasons for obesity rarely come in the form of seeking to be a global actor. For anti-obesity advocates, political gains are made in bypassing domestic political stalemates in favor of wider political attention to the problem. For example, in having a presence in the United States, the International Task Force on Obesity, which has been a major voice in the WHO anti-obesity program, can appeal to scientific consensus of the obesity epidemic to make recommendations on policy implementation to reduce obesity. Policy recommendations are wide ranging from enhanced food labeling, prohibitions on particular food additives, subsidies for local farmer's market organizations or incentives for healthy living by insurance companies (Guthman 2011; Nestle 2002). Furthermore,

the world society pits structural actors like the nation state against the relative agency of individuals, particularly with its emphasis on authoritative sciences (Meyer and Jepperson 2000). Nation states are pressured to enact policies based on established scientific consensus. However, nonstate groups can seek organizational legitimacy through the world society thus freeing them from constraints in and from the state. In this way, both actors seek legitimization though nonstate actors are more able to influence perceptions of their behavior because of their more relative successes (Frank and Meyer 2002). In the US, the food industry has used several tactics to avoid adhering to recommendations from nutritional scientists (Brownell and Warner 2009). So far, the most promising efforts to reduce caloric consumption are not from economic incentives, as some argue, but through nutrition education and calorie information (Seiders and Petty 2004; Senauer and Gemma 2006), which in large measure have been brought about by the negative attention garnered to dense calorie suppliers.

I argue that the role of CSOs stands to impact the global obesity epidemic. First, CSOs are argued to function independently of economic markets freeing them of financial incentives to promote cheap calories, calorie substitute products, or with other mixed motives in food information. Second, CSOs generally operate independently of the state, and as such, outside the formal stalemates of the state polity where they can avoid ideological commitments or other attached strings inhibiting anti-obesity information dissemination. Third, CSOs rely on authority derived from scientific research. There is wide empirical consensus indicating lower overall health and quality of life for the obese (Campos et al. 2006b; Kim and Popkin 2006; Lobstein 2006; Rigby 2006). Fourth, the emphasis by CSOs on social advocacy provides a means of improving global obesity

because of the long tradition of performing watchdog functions of both the state and the market in order to reduce disproportionate burdens. As obesity epidemics go, the wealthy are the first to "contract" obesity due to affluence, but later, the poor are more susceptible because of limited resources, and excess calorie availability. The bifurcation of the calorie distribution shows that the poor, over the long-term, are at highest risk of obesity in wealthy, developing and low-income countries (Hawkes 2006). As Lobstein (2006) has argued, obesity intervention is a step in the process of social justice.

<div align="center">Global Pathways of Obesity</div>

As the nutrition transition theory posits, food consumption and available calories increase as economic development pushes up income. In essence, economic development allows for greater food production that increases food supply, but economic integration can also increase food access to internationally produced foods. Together, greater food supply and accessibility are theorized to be accompanied by higher obesity risk. Accordingly, rising obesity rates among developing countries such as Brazil, Egypt, Mexico, and South Africa are assumed to be the result of wider global and regional economic integration and greater caloric access from domestic and foreign producers (Popkin and Gordon-Larsen 2004).

Nutritional change is accelerating for many developing nations. While being overweight is traditionally characterized as a condition of affluence, recently that association has drifted to lower-income countries (Pickett et al. 2009, Ezzati, 2005). In analyses of Food Balance Sheets from the FAO, evidence suggests that fat intake and income have decoupled in wealthy and middle-income countries, meaning the poor are

more able to afford high calorie foods (Drewnowski and Popkin 1997; Ezzati et al. 2005; Popkin 2003). Other trends include the influence of rapid urbanization occurring in many low-income countries as rising markets for globally traded foods (Caballero 2007; Popkin 2003; Popkin 2001a). These trends suggest that low-income countries are most vulnerable to obesity risk and its negative health outcomes.

Few researchers in global obesity would disagree that obesity is merely coincidentally related to globalization. However, research in global obesity has rarely sought an explanation grounded in the process of globalization itself. The proliferation of being overweight offers a unique opportunity to examine two major contrasting theories of globalization because of the varied nature of globalization "transmission." On the one hand, the influence of global economic restructuring in the era of cheap calories is presumed to be increasing the amount of calories available on the world stage. On the other hand, CSOs work as independent state and market arbiters of healthy living in legal and scientific terms working towards political influence among societies struggling to confront their own obesity epidemics.

In this paper, I test disparate theories of convergence globalization to ascertain how the spread of the obesity epidemic is occurring cross-nationally. Neoliberal globalization and world society approaches posit that isomorphic forces act on diverse societies, however, they do so through different channels. These channels are important because of the opposing obesogenic effects each element of globalization is presumed to take. For example, from a neoliberal economic globalization perspective obesity is driven by consuming Western, salty, fatty foods, and cheaply processed grains. Conversely, from the world society view, civil society creates and legitimizes pressures for healthy eating

and responsible food policy thereby potentially contravening the rising body mass trend. Both perspectives explain global convergence in their own way, but using obesity as an outcome allows us to examine the relative strength of their divergent obesogenic pathways. Economic integration presumably contributes to higher body mass through its key mechanisms, but obesity is potentially obstructed through world society mechanisms. An important piece of the theoretical development in obesity research is the degree to which these influences are mutually exclusive or coconstitutive in obesity onset. It would be helpful to future research to identify whether a traditional globalization approach suffices in explaining rising obesity rates.

I examine the relative influences in global obesity based on theoretically important variables derived from two main perspectives on global convergence. No research to date has explored the influence of CSOs on obesity, nor have any perspectives emerged formally testing obesity through the lens of globalization. I explore the roles of Obesity Oriented INGOs (OINGOs) and IGOs (OIGOs, defined below) as well as economic integration measures to examine their relative contributions on body mass. Given the current amount of foreign dollars invested globally, the expanse of food marketing and agricultural integration across the world, I hypothesize that economic integration will have a positive influence on BMI. With obesity gaining greater attention at the state, regional and cross-national level, the rationale for anti-obesity research and information has expanded a great deal, the outlet for which has traditionally been through civil society. I hypothesize that CSOs have a negative effect on BMI. In addition, I disaggregate CSOs into OIGO and OINGO membership and test their influence separately. IGOs traditionally function through the formal organizations of the world

polity, thus their influences are more prone to be encumbered by that polity. However INGOs enable more direct influence while operating outside of such formal encumbrances. I hypothesize that OIGOs will have lesser effects than OINGOs. Hypotheses are summarized in Table 14.

One aim of this study is to test the relative role of economic globalization and world culture, but also to test the pathways of rising body mass. Accordingly, in case of obesity, civil society memberships would be negatively associated with BMI because they are effectively working towards reducing obesity. Unfortunately, the pathway through which these mechanisms may work is more difficult to uncover. For example, longitudinal data do not sufficiently exist for countries that have enacted policy to confront obesity or its common comorbidities. According to theory, civil society memberships disseminate calorie information, work toward legislation to protect consumers from food additives, or improve dietary options in public schools. However, data about the presence of anti-obesity initiatives, or a national health strategy confronting obesity is not representative of global breadth or chronological depth. Nor, unfortunately, are data more closely related to BMI such as physical activity or energy expenditure. Some proxies have been used – such as controls for employment sector – but have questionable validity (Bleich et al. 2007). The distribution of sampled INGOs and IGOs by type are shown in Figure 6. Table 15 indicates the number of obesity oriented civil society organizations in each region while Tables 16 and 17 show the countries included in the analysis, including the number of OINGO and OIGO memberships of each region in parentheses. The fewest OIGOs are located in South America and the most are in Africa; the fewest OINGOs exist in Oceania while Europe

contains the most. It is important to note, however, that a typical civil society organization membership variable often concatenates all types of civil society organizations regardless of their focus. As shown here, however, obesity-IGOS and obesity-INGOs represent very different conceptual spheres. For example, most obesity-INGOs are related to cardiovascular health, while most obesity-IGOs are concerned with nutrition. Obesity-IGOs and obesity-INGOs were theorized to behave differently because of their different spheres of influence and origin. However, this issue then presents conceptually interesting questions – for example, what can these differences tell us regarding the specific function of IGOs versus INGOs – given that they are often 1) not disaggregated in world society research and 2) reasoned to be functioning through the same mechanisms? One issue for world society theory is whether all the different types of civil society organization memberships share the same characteristics given the very different emphases of those memberships.

Because all regions contain both obesity oriented IGOs and INGOs, statistical associations can be interpreted as having an influence on BMI. In particular, South America and Asia are areas of emerging obesity epidemics, but Africa is still an area of emerging development. Comparing the relative pace of economic and cultural influence on body mass across regions would be a fruitful addition to international obesity research as well as an insightful view into the relative influence of OINGOs regionally. I also include tests assessing the strength of CSOs compared to economic inputs on BMI across geographic regions.

Data

As a dependent variable, I use average BMI[21] from 1980 to 2007 as measured by the World Health Organization. This measure is age-standardized population average, 20 years of age and above. Figure 1 illustrates trending BMI data from the World Health Organization from 1980 – 2007 separated by geographical region.

Key independent variables considered by world polity literature includes membership in the global society. Membership data were collected from the *Yearbook of International Organizations* published by the Union of International Associations – the most common measure of INGO membership.

Organizations were found through the Yearbook index which compiles listings of organizations based on keywords in an organization's mission statement or name. Country level membership was collected at 5 year intervals staring in 1981 until the most recent year available (2007). Indicators of membership included various types of CSO membership. I coded CSO membership across six broad "classes" of organizations as listed by the *Yearbook* specifically relevant to obesity: Public Health, Nutrition, Physical Activity, Cardiology, Diabetes, and Obesity.[22] Each class of organization includes both OIGO[23] and OINGO memberships used in disaggregating world society membership. For

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[21] Obesity prevalence would be a considerably better measure of obesity, however, because of data restrictions both from limited obesity prevalence and CSO membership data, average BMI is considered a reasonable proxy. Pearson product moment correlation between obesity prevalence and average BMI was measured, .86. Furthermore, there exists a strong research precedent for using BMI as a proxy for obesity prevalence (Monteiro, et al. 2004).

[22] Search terms used in locating CSO membership included terms specific to each category including: "medicine/cardiology," "heart disease," "medicine/diabetes," "physical activity," "exercise," "obesity," "BMI," "health care/nutrition," and "public health." Because of coding changes across *Yearbook* editions, the 1984-85 editions and prior, search terms for cardiology related CSOs included "cardiologie," "Societe/ cardiovascular surgery," "physical fitness," and "physical education."

[23] IGO memberships were substantially lower than INGO memberships, particularly in

example, the International Diabetes Association started in 1949, registerd in 145 countries, has the widest and longest standing membership than any other diabetes association. Research conducted in other areas of world society literature have used similar techniques in compiling CSO data (Schofer 2012).

Key independent variables for economic globalization include common measures of marketplace integration. A measure of FDI stocks as a percentage of GDP was used to measure global investment (UNCTAD 2012). Domestic investment stocks were used as a control measure with analyses of FDI available from the World Bank. The use of overall FDI, is consistent within the tradition of political-economic sociology asserting that the accumulation of foreign investment increases social and economic vulnerabilities among less-developed nations (Amin 1976; Chase-Dunn 1975; Jorgenson and Kuykendall 2008). A persistent theme of this literature examines the role of foreign investment in well-being among highly penetrated countries. Furthermore, the limited availability of more direct measures of agricultural or food production FDI warranted using a wider measure of FDI. To mediate this measure of FDI, I use value-added agriculture output as a percent of GDP to assess the strength of the food processing sector. This measure corresponds to net outputs from hunting, fishing, crop cultivation and livestock production without including depreciation or depletion of assets. An ideal measure would have been more closely related to food processing. However, because of its generality, it is also able to assess the wide variety of production available as a country develops. Processed foods are referred to as "value-added" in that a raw agricultural product is enhanced for additional marketing – sugar, eggs, or butter, for instance – which is a large aspect of obesity (Tullao

"diabetes," "obesity," and "cardiology" organizations where no IGO organizations were categorized. While OINGO memberships included six types, OIGO memberships included only three.

2002). As a measure of agricultural trade openness, I use the ratio of agricultural imports to exports (current value base price in US$1000).

Control variables include GDP (in constant 2000 US$), which measures absolute economic status. Total population was used as a country size control. Also, productive age population growth contributes indirectly to industrialization, so population size acts as a means of controlling for economic productivity trajectories, enabling countries to be more closely comparable. These data came from the World Bank.[24] Time was also introduced as a control including years from 1980 – 2007. Data except time were logged to reduce skew. Table 18 shows descriptive statistics and serial correlations of all model variables.

Methods

I employ repeated measures data on 142 countries from 1980-2008 using fixed effects modeling techniques. The main benefit in using fixed effects modeling for cross-national studies is how it treats unobserved variation that can be partialled out (Allison 2009). Fixed effects estimation is a conservative test of model predictors as it assumes unobserved variables vary at random with model predictors. In so doing, this assumption allows the possibility that data may be correlated across time. It uses change over time to control for this bias. Fixed parameters that are constant over time are differenced out of the model equation; thus, reducing heterogeneity bias. Functionally, this assumption "discards" between-case variation in order to reduce model biases (Alderson and Nielsen

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[24] Human capital measures presented a limiting variable. A conservative imputation method included averaging within-country data points for time 2 based on time 1 and time 3. Following this method increased observations from 2792 to 3719 across 130 countries. Even with imputation, including an education measure imposed, there were too many limitations on suitable cases for its inclusion.

1999). While the assumptions made in random effects modeling preserve the use of within- and between-case variance, it assumes unobserved variables to be uncorrelated with model predictors thus allowing variables to be correlated over time. However, by including between-case variation, random effects models introduce bias which leads to a less conservative test of estimation parameters. In order to reduce the likelihood of committing a Type I error and improving model validity, I use fixed effects estimation[25]. I test the roles of key variables on BMI controlling for time, and with and without control variables. These tests enable a direct comparison of economic and CSO mechanisms on BMI that constitute contributing evidence for hypotheses regarding the magnitude and direction of economic input relative to CSO membership. Robust standard errors are used to control for collinearity among indicators of economic development. Outliers were excluded based on outstanding Cooks distance scores. Power calculations were performed using PASS 11 software (Hintze, 2013) and indicated that sample size and model parameters were appropriately specified. Additional Two Stage Least Regression (2SLS) techniques were used to control for endogeneity bias. This method uses instrumental variables to eliminate potentially reciprocal relationships between dependent and independent variables. I discuss this issue along with diagnostic tests using 2SLS in Appendix A.

For a second analysis, I examine the relative influence of OINGOs, OIGOs and economic development tests variables cross-regionally through the use of slope-dummy variables. One method to do this would be testing models on cases within individual regions, but doing so is infeasible given data limitations. Another method includes using

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[25] Analyses using random effects modeling techniques provided substantively similar results. Hausman tests of estimation efficiency indicated fixed effects models were more appropriate over random effects.

categorical dummy variables coded by region to test differences between intercepts. However, neither of these methods allows one to view the factors involved in processes of how OINGOs operate cross-regionally (Jorgenson, Rice and Clark 2012). Slope-dummies allow for examining differences between slopes among categories by testing main effects and interactions between continuous and dichotomous variables (Jorgenson and Clark 2011). Tests of this nature allow a comparative view of key variables on BMI, facilitating a closer view of the influence of obesogenic mechanisms that potentially operate in different ways depending on development or CSO membership.

A slope-dummy is an interaction term wherein a dichotomous variable, $x1$ (Africa), is multiplied by a continuous variable, $x2$ (OINGO membership), which creates a new variable, $x1x2$ (OINGO membership*Africa). This new variable has the values of $x2$ where $x1 = $ "1" (OINGO membership) and "0" for all remaining cases (Allison 2009; Hamilton 1992; Jorgenson, Rice and Clark 2012). Homogeneity of slopes can be tested by entering into the regression equation the main effect (OINGOs) and the slope-dummy variables created by the process above. Constructing variables in this manner primarily allows us to view how OINGO membership affects BMI in specific regions without sacrificing test cases. In this instance, the time-invariant main effects of region are perfectly correlated with the fixed effects and thus accounted for by within-case estimation used in fixed effects modeling (Allison 2009). Regional status is essentially differenced out of the regression equation while the main effect of OINGO membership and the interaction term, "region*OINGO," remain as model predictors. This technique in fixed effects model estimation was used to assess the comparative influence of key variables on BMI across six geographic macroregions: Africa, Asia, South America,

North America, Europe and Oceania. Control variables included logged GDP and population size to limit collinearity.[26] As such, areas of higher CSO concentration – like Africa – should have a stronger influence of CSOs on body mass than areas of low CSO concentration like Oceania. However, specifically related to obesity, South America and Asia should show greater influence by CSOs on BMI relative to other regions given both their emerging developmental status and obesity growth.

Results

In Tables 19 and 20, I show the results of the fixed effects estimation of key world society and global economic integration variables regressed on BMI controlling for time. Models 1 through 5 show key variables tested on the influence of BMI alone, and models 6 and 7 include the influence OIGO and OINGO membership, respectively, with economic integration variables. Most notably, the influence of OIGOs and OINGOs exert relatively weak negative influences on body mass, consistent with the stated hypotheses. Foreign investment shows a negative association when controlling for domestic investment whose effects vary by world society membership type. Agricultural variables are not statistically significant when tested alone, but value-added agricultural output does show a net negative influence of OIGOs. Time shows a consistent positive effect on BMI.

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[26] As I show in Appendix A, using GDP as a control measure introduces methodological and theoretical challenges to analyses using OINGOs. GDP and population size were selected in order to minimize the interferences by additional measures that are also incorporated into economic and social development along the lines of GDP and CSOs. The most effective control for economic development is arguably GDP. Other measures may also be appropriate in these analyses but R-squared values without controls are upwards of .80, indicating that the introduction of additional controls would not likely contribute to the explantory nature of the analyses in any substantive way.

Table 20 shows the results of fixed effects estimation of key variables on BMI including control variables. Statistical significance for OINGOs increases when including statistical controls but drops completely for OIGOs. The effect of foreign investment is consistently negative, but value-added agricultural output shows an influence that switches directions depending on the world society membership inclusion – negative for OIGOs, positive for OINGOs.

Regional Influences on BMI

Tables 21 through 25 show the fixed effects estimation of key independent variables on BMI using regional slope-dummy variables. These tables show the relative contributions to BMI across geographic regions[27]. Figure 7 illustrates the effect sizes of independent variables net of controls. It was reasoned that BMI is subject to variation depending on regional location, thus these tests are intended to show that influences to BMI are not uniform. Table 21 indicates a negative effect of OIGOs across regions relative to European countries, where the degree of influence is greatest. It is noteworthy that the net effect of OIGOs in North America is zero not because of null findings, but due to a negative effect counteracting the overall main effect. Using other reference

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[27] Robustness checks using more closely aligned sample sizes and control variables to the general effects models of Table 18 are included for slope-dummy analyses across key independent variables shown in Appendix B. Results of these additional checks indicate that the effects of key variables are largely due to independent effects and not due to sampling differences or the inclusion of control variables. Two notable exceptions exist. The influence of value-added agriculture on BMI is likely based on sample size. With a reduced sample frame, the main effects of value-added agriculture are not significant. However, the effects of FDI on BMI is likely a function of OIGO membership. The main and regional effects of FDI are altered by OIGO membership differently than OINGOs, indicating the state-oriented nature of OIGOs in some measure contributes to the identification and adjustment to BMI in ways OINGOs do not. Further development of this finding is addressed in Appendix B.

categories, though, did not change the substantive findings, but when references groups changed, it became clear that OIGOS showed a minimal net effect in North America across models. Obesity oriented IGOs in North America consistently ranked among the lowest negative influence from OIGOs, or even a positive effect when used as the reference group. Overall, BMI in European countries was negatively associated with OIGOs as well in countries in Africa, Asia, and South America. Table 20 showed no statistically significant differences in Oceania.

Table 22 includes the effects of INGOs on BMI by region. Unfortunately, not all regions showed statistical significance, but using Europe as the reference region enabled the most statistically significant comparisons. Compared to Europe, all other regions except North America showed negative associations between OINGOs and BMI net of control variables. The effect was strongest in Europe itself.

Contrary to hypothesis 1, only one economic integration variable increases BMI. Next, I report the effects of economic integration through FDI, value-added agricultural output and agricultural trade openness in Tables 22 through 24. Table 22 shows the influence of foreign investment stocks controlling for domestic investment. The main effects of foreign investment are not statistically significant when using Europe as the reference category. However, additional analyses using South America, as shown in Table 23, and Asia, and North America (not shown) as references groups, respectively, show consistent negative effects for all regions. These models show that the strongest negative effects between FDI and BMI exist in Africa also, with South America, Asia, and North America with consistent moderately negative associations. The least negative associations existed in Oceania and Europe.

Regional analyses of value-added agriculture on BMI, shown in Table 24, also indicated regional effects. Consistent effects were seen across models of different reference regions. Relative to other regions, Africa and Asia show the strongest associations between value-added output and BMI while North and South America and Oceania show moderately positive associations. In fact, all countries showed positive associations between value-added output and BMI except when using Europe as the reference region, indicating that value-added agricultural output is not as closely associated with BMI in Europe as it is in other regions.

Lastly, the effects of agricultural openness on BMI are displayed in Table 25. Model 2 shows that agricultural openness is more closely associated with BMI in African countries than among Oceanic countries, relative to Europe. In comparing analysis using different reference groups, BMI in Oceanic and Asian countries is also much less associated with agricultural openness than in Africa. These models indicate that African BMI is subject to more influence than other regions likely due to the overall lower BMI starting point relative to other regions.

Discussion

Scholars have debated the process and mechanisms of globalization. In this paper, I examine the mechanisms of economic globalization and world society perspectives to elucidate the process of a globally growing obesity trend. The purposes of this paper are two fold: first, develop more theoretically cogent claims of international obesity to be compatible with a broader, more generalized explanation of globalization. Second, test the applicability of competing mechanisms of globalization to explain a relatively recent

global phenomenon. These purposes guided the main hypotheses tested in this analysis: first, that economic globalization would be strongly positively associated with BMI, and second, that civil society organizations would be more weakly negatively associated. Furthermore, I hypothesized that areas of greater obesity risk would have higher associations of civil society influence. My findings suggest that there are competing global influences on BMI and that these influences are not uniform. In this section, I evaluate these claims.

<div align="center">Global Economy</div>

The hypothesis asserting that economic integration's positive association with BMI were not wholly supported. FDI and value-added agricultural output were negatively associated with BMI, while agricultural trade openness showed areas of positive associations.

FDI has been the center of a long-standing debate surrounding its role in well-being among less developed countries. The negative influence of FDI on body mass found here holds a number of implications. Prior research has asserted a negative association between FDI and well-being (Brady, Beckfield and Zhao 2007). Only one study has attempted to use BMI as a means of well-being. Wimberly and Bello (1992) use first differencing in Ordinary Least Squares (OLS) regression to assess transnational corporate investment on calories per day from 1967 to 1985. The analytical structure of the first difference estimation equation is virtually equivalent to fixed effects estimation performed here with similar results (Allison 2009). They argued that lower caloric intake primarily occurs through lower economic growth – because FDI is associated with lower

economic growth and income inequality, the inverse relationship they found is a reflection of those detrimental effects. It is noteworthy that the persistent negative effects of FDI are corroborated in my study, and that the swelling of FDI monies of recent decades has not changed its overall effects in host nations. The rationale for outcomes of value-added agriculture could be considered similar. Value-Added agriculture includes food processing facilitated by foreign investment. For that reason, the effects of the value-added agricultural investment mirror closely the effects of FDI.

On the other hand, one of the purposes of this study was to test the effect of a wide array of economic influence measures. FDI was chosen as the broadest measure, while value-added agricultural output was intended as the most specific. My findings bear out this potential mismatch in measurements. The effect of FDI was resoundingly negative, and as addressed earlier, provides particular context to the global health-investment relationship – that FDI used broadly is perhaps an inappropriate predictor for particular health outcomes such as obesity. However, agricultural openness, as a more narrowed measure shares very little overlap with all sector FDI, and as such shows that global food trade may contribute to the global increase in body mass.

These two measures may also provide methodological insight into the study of global obesity. Foreign direct investment's effect, here, may be showing the suppressing effect FDI has on economic growth as is typically argued. The effect of agricultural openness on BMI as shown here may be illustrating how food traded on the global scale may increase BMI because of the greater caloric access the global food trade provides. The issue is likely to be BMI. Often BMI is associated with economic development, but it is also conceptualized as a health outcome. This dual nature may in fact be working at

cross-purposes among these variables – BMI may stand as a development proxy when applied to the effects of FDI, but it may also be functioning as a health outcome measure when applied to the effects of agricultural openness. Using BMI may be an ambiguous outcome when testing on conceptually different predictors. Greater care must be used to hone in on global health indicators rather than let different conceptualizations of development interfere.

The attention from global obesity literature has focused on the influx of FDI as partly responsible for higher obesity prevalence (Hawkes 2006; Kearney 2010; Popkin 2006). My findings do not support this view. In addition, more specific measures of agricultural production reproduced similar effects as FDI. Past research has argued that the inverse relationship of agricultural production and processing and obesity came about because of manual labor inputs (Bleich et al. 2007; Popkin and Gordon-Larsen 2004). This could likely be occurring here as well. When comparing the effects of value-added agriculture across regions, Africa and Asia show much stronger effects than do more developed regions, suggesting that the amount of labor in agricultural production in these areas outweighs the overall benefit from supplying additional calories. Agricultural trade openness does show a positive association with BMI, particularly in regions with lower average BMI like Africa and Asia and the effects among higher BMI regions is relatively low.

Contemporary explanations of global obesity have characteristically lacked a strong theoretical undercurrent though its emergence could be greatly enhanced by theoretical development. I have used research methods from contemporary globalization literature to assess the applicability of obesity within a broader globalization framework

to varying degrees of success. Obesity is robust to interventions of various stripes but this research establishes the importance of education, advocacy and legal accountability accomplished through civil society. As London and Schneider (2012) argue, one reason for success by health-as-a-human-right advocacy is through the codification of human rights into national and international law granting the underserved a political voice across settings. They add that political rights often stand parallel with socio-economic rights. In the obesity dialogue, these different voices can often present competing claims about the causes of obesity. Public opinion in the United States, though, has come to view obesity as partly a function of environmental influences which undermine the personal effort directed against it (Brownell 2005). Advocacy would appear to play a key role in facilitating both personal and environmental efforts, particularly when state-level political and economic efforts have not been effective.

The performance of traditional economic globalization indicators, on the other hand, have provided limited conclusions in part because of correlation with other economic functions. For example, foreign investment's association with economic stagnation requires a more carefully refined measure of food related investment and processing to assess their relative influence for obesity outcomes. These data are not widely available. Subsequently, as presented here, it would appear that there are few economic influences on obesity despite a growing literature body suggesting the contrary. Offering a unified theory of obesity globalization shows obvious challenges because of the intricate nature of the global economy compounded by the wide array of inputs on body mass.

World Society

Results indicate that international obesity trends are compatible with a world society perspective of globalization. As the world society position argues, one of the primary functions of civil society memberships is to act as advocates for those affected by salient global problems. In most regions, world society measures were negatively associated with BMI with the exception of North America and Oceania. It is noteworthy that the net effect of OIGOs in North America from Table 20 was zero not because of null findings, but due to a negative interaction effect counteracting the overall main effect. These results could actually be reflective of a recent BMI plateau trend in the United States given its high proportion of overweight relative to other North American countries (Ogden et al. 2012). It may be that the effects of OIGO membership in North America are just keeping pace with obesity, but not enough to reduce it. Likewise, Oceania reflects overweight prevalence at a much higher proportion than the average population. The effect of IGOs in these areas may simply be overwhelmed by the disproportionate overweight populations. Hypotheses suggested that, if CSOs were to have any effect, it would be most present in areas of higher CSO concentration. Civil society memberships are lower in Oceania which may explain its relative associations with body mass.

IGOs versus INGOs

In the past, the world society emphasis on state isomorphism has conceptualized state transformation as acting through political and legal IGO ties. INGOs, on the other hand, as more autonomous actors, are more closely aligned with the social capital through which policy scripts influence the state (Beckfield 2003). One limitation of the

world society perspective is the degree to which it postulates similar effects for a multiplicity of organizations of varying kinds. A major drawback to operationalizing CSO influence has been in combining both IGOs and INGOs of a certain kind, but also pooling all CSO memberships regardless of their intended target area. This study attempted to differentiate the influence of CSOs in two ways. First, I allow for OIGOs and OINGOs to have separate effects. Second, the organizations themselves were thought of as being instrumental in mediating body mass. Conceptualizing the CSO sample in this way allowed me to test the mechanisms of world society through a specific type of organization rather than pooling the effects of diverse types of organizations. Studies engaging this approach are limited, and no study has yet engaged obesity using these methods.

One goal of separating OIGO and OINGO measures is to corroborate with Beckfield's (2008) findings of a more equal effect of IGOs to assess the salience of IGO versus INGO influence. According to hypotheses, IGOs correlated with lower magnitudes or nonsignificant differences between regions because of their distribution or concentration among developing nations. However, this deserves some qualification. At the global level net of controls, OIGOs were not statistically significant but showed opposite effects from OINGOs. Two possibilities exist for these disparate effects. First, obesity oriented IGOs may be underrepresented relative to OINGOs. For example, no IGOs existed explicitly oriented towards diabetes or obesity. The incompleteness of the data then may explain important differences in their BMI outcomes. Second, this pattern exhibits abnormalities compared to world society literature which asserts that IGOs and INGOs effects have a consistent similarity. Two questions arise: Why are obesity related

IGOs and INGOs different than total IGO and INGO comparisons, and what are the substantive reasons for this divergence? Assuming these are not artifacts of incomplete membership data, the answer to both these questions could be the same. Tsutsui (2004) suggests similar results found in her own analysis. She found that for ethnic mobilization, civil society played a role in fomenting problem salience and contributed to political mobilization of IGOs and INGOs. OIGOs, in this case, concentrate their efforts among countries where BMI poses less of a relative problem and political action is easily justified. Body mass, among OIGOs, represents not obesity, but malnutrition – hence the positive, albeit nonsignificant effect. In this application, OIGOs and OINGOs may have different foci by the nature of the outcome measure – hence the low serial correlation. Body mass has different practical meanings in different contexts, thus these divergent effects likely reflect the political functionality of IGOs where it is needed most. Other world society outcomes like human rights violations, for example, may not share the same contextual ambiguity.

However, at the regional level, Table 20 indicates that among four of the six macrogeographical regions, relative to Europe, the effects of OIGOs were consistently negative on BMI. Still, despite the unequal distribution of IGO membership among regions, the effects were largely similar. In this case, other regions relative to Europe showed similar effects of OIGOs despite their differences in discrete CSO membership. Africa, where OIGO membership outstripped Europe by as many as 3 fold, and South America by nearly 8, showed similar effects from OIGOs as these other less connected regions. However, OIGO influences on body mass were less there than in Europe. Obesity oriented INGOs, though, showed similar cross-regional effects as did OIGOs.

The effects of OINGOs were much stronger than OIGOs globally, but when disaggregated by region, the effects of OINGOs were actually lower than OIGOs. Like Table 7, Table 8 showed that the effect of OINGOs was greatest in Europe. While OIGO and OINGO membership is influential, their effects were not regionally uniform.

Both types of CSOs predominant functionality in Europe compared to other areas could arise from a number of sources, but let us consider the following: first, in particular, Africa and Asia both have lower than average BMI. The association between OIGOs and BMI are negative in those regions because there, obesity is not yet a widespread phenomenon. The negative associations between OIGOs and BMI likely reflect "room to grow" in the effectiveness of CSOs among these regions. Additional analyses using percent change scores among all variables were used to examine if the persistent negative effect of OINGOs could be attributed to program ineffectiveness or simply due to a lower overall BMI starting point among African and Asian countries. Insufficient data existed for most tests, but the negative effect of OINGOs on BMI among African nations changed to positive and statistically significant at the 0.01 level. These results suggest that OINGOs could actually be contributing to rising obesity or malnutrition alleviation in these circumstances. The majority of organizations in Africa and Asia are oriented around nutrition and heart health. Both these types of topics are directly related to obesity, and as such, the relatively low associations between CSOs and BMI in these regions compared to Europe may reflect successful interventions where CSOs, OIGOs in particular, have prevented obesity from fully emerging.[28]

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[28] Additional analyses using BMI compared to obesity prevalence as an outcome measure produced two main conclusions. First, analyses of OIGOs on obesity prevalence did not have sufficient cases for output. Among the "general effects" models corresponding to Table 18, where differences did emerge, the number of cases differed by as much as a factor of 10. Second, further regional analyses were run

Also, unfortunately, this type of model also exemplifies a major problem inherent to analyses of economic development, BMI and INGO linkages. Essentially, as poor countries develop, both INGO memberships, economic development and BMI all stand to increase because of their habit of having low levels to begin with, thus making a positive association inherent to the model prediction on the basis of correlation alone. Relying on change models therefore introduces substantial collinearity and highlights challenges implicit in the nature of INGO linkages. Regardless, regional context plays an important role in how OINGO influence should be interpreted.

Second, decoupling has been noted as a persistent problem for the world society perspective because of the disconnect between theory and practice. As Meyer (2010) explains, world actor standards exceed the capacities they possess, resulting in program ineffectiveness. In this case, obesity oriented civil society organizations presume an adherence to the virtues of reducing obesity risk factors, but face localized limitations in their ability to attain their goals (Meyer 2000). The relative effectiveness by CSOs among different regions corroborates with decoupling among lesser developed countries theorized by world society literature (Meyer et al. 1997). More broadly, this finding also suggests that health as a human right has yet to gain relevance in these areas. As Palmer (2009) argues, the link between treaty ratification and health improvement is a weak one. It stands to reason that based on the association between civil society membership and social advocacy, the traction required for legal accountability and health improvement

corresponding to Tables 20-24 reported above. The effects of the five key independent variables on obesity prevalence, again, produced similar effects. Among the three economic integration variables tested here, their effects on obesity prevalence were enhanced consistent with findings reported above among African and Asian countries. As such, BMI, while not ideal, did not produce substantially different results from a more refined measure.

may need substantial improvement.

This study attempted to use common world society measures to assess a theoretically derived function of global civil society theorizing different results than commonly assumed. Discrepancies between theoretical predictions and empirical outcomes are likely due to the practical operationalizing of civil society and economic development. Beckfeild's work illustrates the inequality of the world society acts to concentrate INGO functionality to higher areas of development. One problem this has introduced is parsing out the effects of social and economic development in civil society relative to the effects of the civil society itself. Even in this study, economic development and world culture operationalized by civil society organizations are so highly related, in relevant tests, the influence of economic development overshadowed effects of civil society. The influence of civil society is so closely tied to economic development that operationalizing world culture as a function of civil society membership inhibits the ability to find mutually exclusive effects. This would in part help explain null findings in my hypotheses of civil society – there may be effects but these measures may not be sufficiently specific to uncover them.

This research also hold implications for public health policy by putting together several of the main findings. First, one of the main findings of this research suggests that among poor countries, malnutrition creates a substantial obesity risk among growth stunted children and that malnutrition tends to contribute to obesity more so in Asian countries than in other regions. Of course, on this finding alone, it could be recommended to improve access to nutritional programs among developing nations. Doing so would improve malnourishment in children, but it would also appear to improve obesity risk

later. Also, another main finding is that obesity oriented IGO membership in developing regions tended to concentrate among nutritional organizations, and that IGO memberships were associated with lower BMI independently of economic development and market forces. Though the effect of obesity-IGOs was small, in a majority of regions, the effect was greater than that of obesity-INGO memberships. Intergovernmental organizations appear to be the most appropriate vehicle for improving nutrition, and obesity outcomes across countries.

Improving IGO access to a country makes both theoretical and practical sense. CSOs are guided by a sense of social welfare; perhaps where they differ most is in their funding, organizational structure, and perceptions of their global legitimacy. Because of their associations with formal global political bodies, IGOs stand to be more consistently funded, staffed by professionals from around the world, and are likely to have doors opened to them based on the perceived need by local officials. In a sense, IGOs may be able to use the name of their parent organization – the WHO for example – to permit them to work in a given location unavailable to INGOs with fewer resources. In addition, because IGOs tend to act independently of market forces, they stand to provide services more widely to countries where markets are yet to emerge and bring INGO attention.

Limitations

This study has a number of limitations. First, BMI is often used as an outcome measure in global obesity research. Obesity prevalence and BMI are not synonymous, though in many respects they are intended to be. One problem in using BMI is that it often masks subgroup variation of those at lower or higher risk of obesity as BMI is a

measure of central tendency. These problems are amplified by the study's level of analysis. BMI is a poor proxy for obesity in countries like Vietnam or Ethiopia where average body weight is still quite low. However, it performs well in countries with widespread overweight populations such as Samoa or Tonga whose BMI scores are 2 to 3 standard deviations higher than the world average. A common problem for this research, where data are limited, is that there is a trade-off in sensitivity for specificity, and data would be too restricted if limiting analyses to countries with higher than average BMI, or when using a more refined obesity measure such as obesity prevalence. For better or worse, BMI remains the most commonly used indicator of obesity in related literature.

Second, collinearity among cross-national indicators introduces methodological challenges which may overwhelm common methods of analysis. The largest of which is separating the effects of economic development from culture, which may not be operationally possible. Linear fixed effects estimation was used here because it represents a suitable method for holding constant implicit cultural fixed effects without introducing unwarranted regression assumptions. Using alternative methods were explored to overcome this problem but the related nature of these test variables inhibited any ability to obtain defensible results. An exploration of these issues is dealt with in Appendix A.

Third, OINGOs and OIGOs as reported by the *Yearbook of International Associations* are incomplete. Tracking methodologies by the *Yearbook* rely on periodic self-reporting by organization staffers, which, in many cases are lost to follow up. Membership records often lag behind the ability for the *Yearbook* to accurately keep up with the organizations. Also, the *Yearbook* record may not reflect accurate data when individuals constituting membership do not operate within a formal international

organization. However, these are common problems subject for scholarship using civil society membership counts and represent the best data options available for INGO research.

Last, this study emphasizes the function of the world society by using explicit types of world society organizations to identify substantive effects of civil society influence. An important criticism of world society theory is that while civil society organizations are presumed to transmit patterns of influence, all civil society organizations are also presumed have the same effects. In the case of this study, for example, coding obesity-related INGOs labeled "the European Groups for Research into Elderly and Physical Activity" as having the same scope and function as "the European Association for the Study of Diabetes" may not be a reasonable assumption to make. Putting the function of all types of INGOs on the same footing makes too many assumptions about their influence on the state, as well as the influence of their staffers. Differentiating OIGO from OINGO membership represents a step in the right direction. Theoretical refinement of the mechanisms, functions, and influence of INGOs may be warranted for further application of world society scholarship.

Conclusion

Approaches to the global obesity epidemic have lacked formal inputs from globalization theory. This study attempts to make the connections between rising BMI and economic and cultural connectivity in the modern era more explicit. This approach intimates a number of directions for future international obesity research. For countries where data are more rich, is the effect of CSOs consistent? Given that body mass across

the globe is increasing and the direct inputs on this trend are economic, at what point can we expect body mass to level out and what are the inputs precipitating that plateau? As observers of global environmentalism note, the power for change lies with an informed populace, and according to these data, the mechanisms through which information is disseminated has contributed to some noticeable results. Thus what is the point at which body mass may begin to decline particularly when the influences are strongly related to economic and social development? Can more specific measures of global trade be linked to faster trajectories of obesity or body mass? With a clearer sense of how body mass is growing, these questions would be especially relevant to emerging economies into the future.

Table 14
Proposed Hypotheses on the Role of CSOs and Body Mass

1	Economic integration will have a positive influence on BMI.
2	OIGOs will demonstrate a lesser effect than OINGOs.
3	Higher CSO concentration have a stronger influence on body mass than areas of low CSO concentration.
4	South America and Asia should have a higher influence by CSOs on BMI relative to other regions.

Table 15
Percent of Sampled OINGO and OIGO Types by Region

Region	Percent of OINGO Memberships (OIGO)					
	Cardiology	Diabetes	Obesity	Nutrition	Physical Activity	Public Health
Africa	49.3	20.9	11.6	2.8 (99.4)	14.4 (0)	.9 (.6)
Asia	63.3	16.7	7.2	8.1 (93.9)	4.2 (0)	.4 (6)
South America	65.2	12.7	7.6	5.1 (54.2)	8.5 (45.8)	.9 (0)
North America	43.8	32.9	5.1	8.8 (90.5)	7.3 (9.5)	2.2 (0)
Europe	72.6	2.3	13.7	7.8 (73.5)	1.6 (8.8)	2 (17.7)
Oceania	25.5	19.6	7.8	31.4 (97.8)	2 (0)	13.7 (2.2)

Table 16
Countries with OINGO Membership by Region

Africa (216)	Asia (234)	South America (120)	North America (137)	Europe (250)	Oceania (51)
Algeria	Armenia	Argentina	Antigua and Barbuda	Austria	Australia
Benin	Azerbaijan	Bolivia	Bahamas	Belarus	Fiji
Botswana	Bangladesh	Brazil	Barbados	Bulgaria	New Zealand
Brunei Darusaalam	China	Chile	Belize	Cyprus	Papua New
Burkina Faso	Hong Kong	Colombia	Bermuda	Denmark	Guinea
Burundi	India	Ecuador	Canada	Estonia	Tonga
Cameroon	Indonesia	El Salvador	Costa Rica	Finland	
Central African Republic	Iran	Grenada	Cuba	France	
Chad	Iraq	Guatemala	Dominica	Georgia	
Congo DR	Israel	Guyana	Dominican Republic	Germany	
Cote d'Ivoire	Japan	Nicaragua	Haiti	Greece	
Egypt	Jordan	Panama	Honduras	Hungary	
Ethiopia	Kazakhstan	Paraguay	Jamaica	Iceland	
Gabon	Korea DR	Peru	Mexico	Ireland	
Gambia	Korea Rep	Suriname	St. Kitts and Nevis	Italy	
Ghana	Kuwait	Uruguay	St. Vincent	Latvia	
Guinea	Kyrgyzstan	Venezuela	and Grenadines	Lithuania	
Kenya	Lebanon		USA	Malta	
Lesotho	Malaysia			Moldova	
Madagascar	Nepal			Netherlands	
Mali	Pakistan			Norway	
Mauritania	Philippines			Poland	
Mauritius	Saudi Arabia			Portugal	
Mozambique	Singapore			Romania	
Namibia	Sri Lanka			Russian Federation	
Niger	Syria			Spain	
Nigeria	Tajikistan			Sweden	
Rwanda	Thailand			Switzerland	
Senegal	Turkey			UK	
Seychelles	Turkmenistan				
Sierra Leone	United Arab Emirates				
South Africa	Ukraine				
Sudan	Uzbekistan				
Swaziland	Vietnam				
Tanzania					
Togo					
Tunisia					
Uganda					
Zimbabwe					

Table 17
Countries with OIGO membership by Region

Africa (159)	Asia (68)	South America (24)	North America (63)	Europe (47)	Oceania (46)
Angola Benin Botswana Brunei Darusaalam Burkina Faso Burundi Cameroon Central African Republic Chad Comoros Cote d'Ivoire Djibouti Ethiopia Gabon Gambia Ghana Guinea Guinea Bissau Lesotho Madagascar Malawi Mali Mauritania Mozambique Namibia Niger Rwanda Sao Tome and Principe Sierra Leone Seychelles Sudan Swaziland Tanzania Togo Uganda Zimbabwe	Armenia China Japan Korea DR Kyrgyzstan Laos Malaysia Mongolia Mongolia Nepal Philippines Syria Tajikistan Thailand United Arab Emirates Uzbekistan Vietnam	Argentina Bolivia Brazil Chile Colombia Ecuador El Salvador Grenada Guatemala Guyana Nicaragua Panama Suriname Uruguay Venezuela	Antigua and Barbuda Bahamas Barbados Belize Canada Costa Rica Cuba Dominica Dominican Republic Haiti Honduras Jamaica St. Kitts and Nevis St. Lucia St. Vincent and Grenadines St. Lucia Trinidad and Tobago USA	Austria Belarus Bulgaria Denmark Finland France Greece Iceland Ireland Italy Netherlands Norway Poland Portugal romania Spain Sweden Switzerland United Kingdom	Papua New Guinea Samoa Solomon Islands Tonga Vanuatu

Table 18
Bivariate Correlations of Variables and Summary Statistics

Variable	N	M	SD		1	2	3	4	5	6	7	8	9	10
BMI	5568	0	1	1										
OIGO	407	0	1	2	-0.28*									
OINGO	1008	0	1	3	0.25*	-0.39*								
FDI	4857	0	1	4	0.16*	0.18*	-0.12*							
Domestic Investment	5156	0	1	5	0.20*	-.072	0.133*	0.10*						
Value-Added Agriculture	4803	0	1	6	-0.56*	0.27*	-0.44*	-0.17*	-0.16*					
Agricultural Openness	4731	0	1	7	0.11*	0.16*	-0.12*	0.04*	0.08*	-0.25*				
GDP	5733	0	1	8	0.16*	-0.24*	0.73*	-0.15*	0.03*	-0.50*	-0.11*			
Population size	6787	0	1	9	-.30*	0.12*	0.40*	-0.21*	-0.12*	0.09*	-0.28*	.76*		
Year	8119	–	–	10	0.26*	0.27*	0.06*	0.36*	0.03	-0.19*	0.10*	0.10*	0.06*	

Notes: * $|p| > .05$

Table 19
Fixed Effects Analyses on Average BMI, 1981– 2007

Table 18: Fixed Effects Models of Key Variables 1981-2007
Dependent Variable: BMI

Variables	1	2	3	4	5	6	7
OIGO	-0.0203*					0.00853	
	(0.0121)					(0.00829)	
OINGO		-0.0197*					-0.0189
		(0.0107)					(0.0117)
FDI Stocks			-0.0447***			-0.0312**	-0.0597***
			(0.0119)			(0.0130)	(0.0173)
Domestic Investment Stocks			0.0112			0.0137	0.00396
			(0.00704)			(0.0126)	(0.0119)
Value-Added Agricultural Output				-0.0341		-0.151***	0.0499
				(0.0404)		(0.0413)	(0.0367)
Agricultural Openness					0.00747	-0.0197	0.0143
					(0.0234)	(0.0145)	(0.0281)
Time	0.0365***	0.0337***	0.0366***	0.0314***	0.0333***	0.0349***	0.0403***
	(0.00266)	(0.00126)	(0.00135)	(0.00167)	(0.00123)	(0.00216)	(0.00224)
Constant	-73.00***	-67.16***	-73.10***	-62.70***	-66.38***	-69.79***	-80.48***
	(5.303)	(2.507)	(2.697)	(3.326)	(2.453)	(4.317)	(4.476)
Observations	329	884	3,483	4,486	4,277	172	536
Number of id	135	172	165	182	155	76	126
Rho	0.982	0.979	0.986	0.980	0.978	0.992	0.986
Within Rsq	0.816	0.846	0.858	0.786	0.812	0.929	0.890

Notes: Standardized regression coefficients. Robust standard errors in parentheses.
*** $p<0.01$, ** $p<0.05$, * $p<0.1$

Table 20
Fixed Effects Analysis on Average BMI with Control Variables, 1981 – 2007

Variables	1	2	3	4	5	6	7
OIGO	-0.00935					0.00598	
	(0.00923)					(0.00916)	
OINGO		-0.0212**					-0.0222*
		(0.00963)					(0.0121)
FDI			-0.0389***			-0.0352**	-0.0553***
			(0.0115)			(0.0144)	(0.0170)
Domestic Investment Stocks			0.00155			0.0116	0.00200
			(0.00773)			(0.0127)	(0.0113)
Value-Added Agricultural Output				0.0429		-0.0829*	0.114*
				(0.0390)		(0.0491)	(0.0630)
Agricultural Openness					0.0228	-0.0226	0.0188
					(0.0150)	(0.0139)	(0.0259)
Time	0.0355***	0.0242***	0.0291***	0.0215***	0.0236***	0.0344***	0.0372***
	(0.00348)	(0.00294)	(0.00296)	(0.00245)	(0.00294)	(0.00337)	(0.00536)
GDP	0.520***	0.354***	0.407***	0.641***	0.521***	0.351*	0.236
	(0.0952)	(0.134)	(0.132)	(0.109)	(0.144)	(0.184)	(0.203)
Population size	-0.617**	0.703***	0.152	0.453*	0.349	-0.460	0.0817
	(0.274)	(0.248)	(0.241)	(0.258)	(0.241)	(0.310)	(0.369)
	(7.006)	(5.763)	(5.868)	(4.844)	(5.799)	(6.845)	(10.54)
Observations	292	851	3,429	4,399	4,078	179	538
Number of id	122	168	163	178	153	77	126
Rho	0.982	0.991	0.988	0.992	0.989	0.987	0.985
Within	0.899	0.862	0.869	0.828	0.838	0.919	0.887

Notes: Standardized regression coefficients. Robust standard errors in parentheses.
*** $p<0.01$, ** $p<0.05$, * $p<0.1$

Table 21
Fixed Effects Slope-Dummy Analysis of OIGO
Membership on Average BMI, 1980 – 2007
Europe Reference

Variables	1	2
OIGO	-0.125***	-0.113***
	(0.0108)	(0.0106)
Regional Interaction		
Africa	0.0732***	0.0837***
	(0.0112)	(0.0146)
Asia	0.0480	0.0730***
	(0.0358)	(0.0130)
South America	0.0883***	0.0767***
	(0.0277)	(0.0112)
North America	0.155***	0.113***
	(0.00795)	(0.0120)
Oceania	0.120*	0.104
	(0.0674)	(0.0736)
GDP		0.524***
		(0.0945)
Population size		-0.491
		(0.313)
Time	0.0366***	0.0339***
	(0.00269)	(0.00395)
Constant	-73.26***	-67.67***
	(5.349)	(7.951)
Observations	327	290
Number of id	135	122
Rho	0.981	0.982
Within Rsq	0.829	0.89

Notes: Standardized regression coefficients.
Robust standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 22
Fixed Effects Slope-Dummy Analysis of OINGO
Membership on Average BMI, 1980 – 2007,
Europe Reference

Variables	1	2
OINGO	-0.110***	-0.0871***
	(0.0230)	(0.0194)
Regional Interaction		
Africa	0.0979***	0.0813***
	(0.0193)	(0.0159)
Asia	0.0880***	0.0442*
	(0.0269)	(0.0243)
South	0.104***	0.0802***
	(0.0204)	(0.0173)
North	0.124***	0.106***
	(0.0279)	(0.0244)
Oceania	0.0583	0.0324
	(0.0542)	(0.0549)
GDP		0.354***
		(0.129)
Population		0.583**
		(0.253)
Time	0.0342***	0.0256***
	(0.00124)	(0.00287)
Constant	-68.21***	-51.32***
	(2.484)	(5.635)
Observations	883	847
Number of id	171	167
Rho	0.981	0.990
Within Rsq	0.853	0.867

Notes: Standardized regression coefficients.
Robust standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 23
Fixed Effects Slope-Dummy Analysis of FDI 1981 – 2007
on Average BMI, Reference Regions: South America and Europe

Variables	1	2	3	4
FDI stocks	-0.296*	-0.453***	0.0291	0.0326
	(0.156)	(0.157)	(0.0250)	(0.0256)
Regional Interaction				
Africa	-0.653	-1.055**	0.181***	0.189***
	(0.402)	(0.405)	(0.0572)	(0.0601)
Asia	-0.0772	-0.133**	0.0624***	0.0750***
	(0.0598)	(0.0599)	(0.0150)	(0.0152)
North America	0.0355	0.0765**	-0.0417***	-0.0388***
	(0.0375)	(0.0373)	(0.00674)	(0.00656)
South America			-0.0230**	-0.0344***
			(0.0111)	(0.0110)
Europe	0.104**	0.156***		
	(0.0504)	(0.0499)		
Oceania	0.0909	0.149**	-0.0298**	-0.0310*
	(0.0615)	(0.0629)	(0.0144)	(0.0160)
Controls				
GDP		0.461***		0.461***
		(0.121)		(0.121)
Population		0.275		0.275
		(0.231)		(0.231)
Domestic investment	0.0134*	0.00305	0.0134*	0.00305
	(0.00682)	(0.00703)	(0.00682)	(0.00703)
Time	0.0358***	0.0265***	0.0358***	0.0265***
	(0.00141)	(0.00300)	(0.00141)	(0.00300)
Constant	-71.57***	-52.99***	-71.57***	-52.99***
	(2.811)	(5.947)	(2.811)	(5.947)
Observations	3,473	3,437	3,473	3,437
Number of id	165	162	165	162
Rho	0.987	0.990	0.987	0.990
Within Rsq	0.869	0.883	0.869	0.883

Notes: Standardized regression coefficients. Robust standard errors in parentheses.
*** $p<0.01$, ** $p<0.05$, * $p<0.1$

Table 24
Fixed Effects Slope-Dummy Analysis of
Value-Added Agricultural Output
on Average BMI, 1981 – 2007;
Reference Europe

Variables	1	2
Value-Added Agricultural Output	-0.338***	-0.133**
	(0.0676)	(0.0575)
Regional Interaction		
Africa	-1.107***	-0.927***
	(0.167)	(0.149)
Asia	-0.216***	-0.259***
	(0.0402)	(0.0326)
South	0.0463*	0.0791***
	(0.0263)	(0.0201)
North	0.0961***	0.0984***
	(0.0164)	(0.0153)
Oceania	0.212***	0.169***
	(0.0485)	(0.0400)
GDP		0.648***
		(0.104)
Population		0.215
		(0.249)
Time	0.0317***	0.0234***
	(0.00156)	(0.00261)
Constant	-63.27***	-46.82***
	(3.119)	(5.163)
Observations	4,412	4,401
Number of id	178	178
Rho	0.984	0.992
Within Rsq	0.835	0.860

Notes: Standardized regression coefficients.
Robust standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table 25
Fixed Effects Slope-Dummy Analysis of Agricultural
Openness on Average BMI, 1981 – 2007
Reference Region: Europe

Variables	1	2
Agricultural Openness	0.0383	0.107**
	(0.0583)	(0.0473)
Regional Interaction		
Africa	0.240**	0.296***
	(0.118)	(0.106)
Asia	0.102***	0.0980***
	(0.0321)	(0.0275)
South	0.00153	0.00384
	(0.0200)	(0.0194)
North	-0.00101	-0.0150
	(0.0187)	(0.0170)
Oceania	-0.0854***	-0.0998***
	(0.0261)	(0.0273)
GDP		0.520***
		(0.137)
Population		0.435*
		(0.226)
Time	0.0327***	0.0225***
	(0.00119)	(0.00274)
Constant	-65.18***	-45.05***
	(2.380)	(5.420)
Observations	4,273	4,069
Number of id	155	153
Rho	0.981	0.991
Within Rsq	0.833	0.857

Notes: Standardized regression coefficients.
Robust standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1
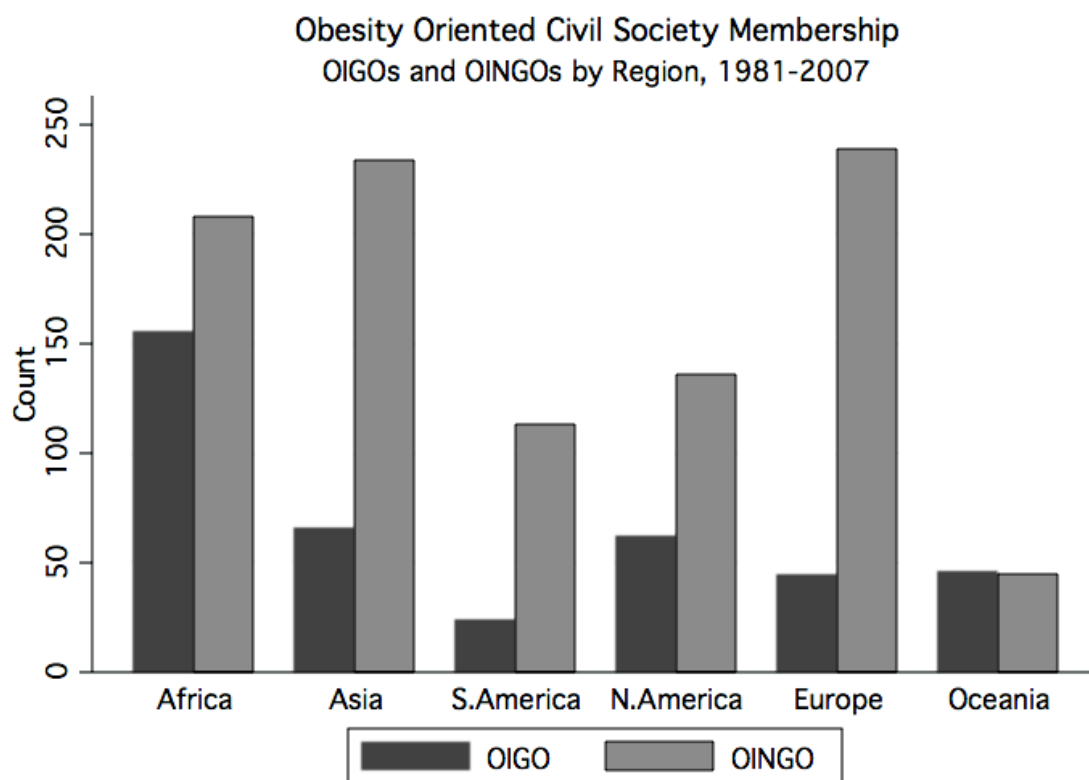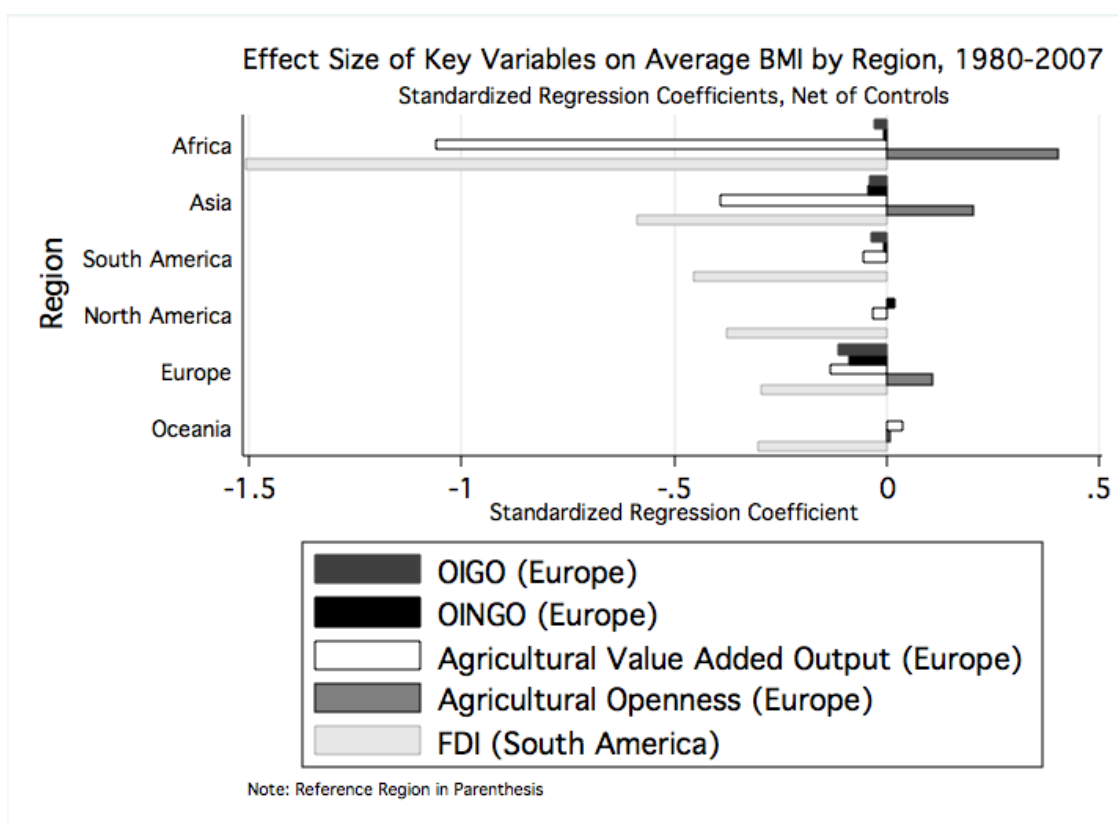
Figure 6. Obesity Oriented Civil Society Membership

Figure 7. Effect Size of Key Variables on Average BMI by Region, 1980 – 2007

CONCLUSIONS

Findings and Contributions

One of the guiding aims of this project is to examine competing explanations for the global obesity epidemic from theoretically informed views of global structure. I examine how economic development and global market integration are associated with obesity. This project aims to refine several theoretical frameworks which are useful, but have proved incomplete in framing international obesity scholarship. Following, I review the main findings of each chapter, and discuss their implications. In Chapter 1, I examine the role of malnutrition and urban poverty on obesity prevalence, body mass index (BMI), and caloric density and access. In Chapter 2, I examine how perspectives of global convergence influence rising body mass outcomes through two channels: economic means through foreign investment, agricultural production and trade, and the prosocial advocacy encouraged from civil society membership. To conclude, I review how typical obesity scholarship underemphasizes the role of malnutrition among the poor as a driving force of obesity in poor countries, and how obesity studies, in incorporating the effects of economic development may benefit from global convergence perspectives. I discuss the contribution of this research by arguing that my findings indicate, international obesity research has accepted incorrect assumptions of obesity as a condition of overabundance rather than malnourishment. I offer thoughts on the

limitations of this research, and explain the strategies I use to overcome many of those challenges. I end with additional questions for international obesity research and concluding comments.

Obesogenic Effects of Malnutrition

and Urban Poverty

In Chapter 1, I concentrate on what effects urban poverty have on body weight using two main perspectives emphasizing the role of inexpensive calories and food access. In this chapter, I assess obesity among developing nations to address a puzzle in the literature that finds faster growth rate of obesity among poor nations. Several conclusions can be made from this analysis. First, the nutrition transition perspective does not effectively posit the role of the economically marginalized in the onset of obesity itself. Nutrition transition argues that dietary shifts are likely responsible for higher obesity rates. What is more likely is that nutrition transition stratifies the nutritional access across the population. The urban poor are last to benefit from it. I find that those living in urban poverty, are less likely to be obese regardless of the nutritionally empty calories consumed. My findings also show malnutrition playing an important role over and above dietary composition, international food trade, and political regime. Diagrams of the nutrition transition have posited that the primary inputs to obesity are increased fat, sugar and processed foods (Popkin and Gordon-Larsen 2004). According to nutrition transition, calorie dense foods drive obesity and diet-related non-communicable diseases, but my findings show that fat consumption among malnourished children actually lowers obesity risk.

At first sweep, these findings appear to contradict assertions of the nutrition transition perspective, but in light of uneven economic development they follow a certain logic. My findings suggest the stages of nutrition transition may be overlaid within a country based on economic vulnerability. Essentially, obesity risk is accelerated in poor countries from obesity increases at both ends of the economic spectrum – lower physical activity and higher caloric intake may be occurring among the wealthy (Chopra 2002), and my findings indicate growth stunting increases risk among the economically vulnerable.

The empirical and theoretical puzzle introduced in Chapter 1 indicates that much of the thinking about global obesity posits the importance of caloric availability – people are gaining weight because of rising economic power, wider caloric marketing, and higher dietary access. My results would suggest that this may only be a selective reading of global obesity literature. I find that many of the commonly intuited factors of weight gain play smaller roles compared to nutritional deficit. Hence, climbing obesity requires viewing obesity through the lens of nutritional impoverishment rather than mere abundance.

Obesogenic Effects of Globalization

Another main purpose of this project is to verify whether or not obesity scholarship can be enhanced by macrolevel structural perspectives of globalization. Globalization is often cited as having a disproportionately negative impact for developing countries (Stiglitz 2003). The analyses presented here corroborate that reading. As argued here, obesity may primarily afflict those at the top and bottom of the economic ladder.

Perhaps more importantly, the view of obesity presented in Chapter 1 challenges the traditional notions of its onset. In Chapter 2, the guiding question is whether or not convergence theories of globalization can effectively describe global obesity trends. I find that the effect of indicators of globalization on BMI reflect disparate outcomes depending on the region. In Africa, the most starkly contrasted region, FDI has a strongly negative influence on BMI, while agricultural trade openness has a substantially positive one. Those differences are minimized substantively in North America and Europe. Also, body mass convergence may be occurring in counter-intuitive ways. For example, my findings indicate FDI growth among lower-income regions suppress body mass rather than increase it.

To corroborate this reading, in Chapter 2, I further test the proposition that economic integration is responsible for increasing BMI. There, my purposes include integrating obesity scholarship into broader sociological theories of global structure, and testing the applicability of mechanisms of global convergence which presume the opposite obesogenic effects. On the one hand, economic globalization is presumed to stimulate body mass convergence through providing technological innovations to lower manual labor in food production, produce goods for the world market, and supply additional cheap, dense calories. However, existing civil society also compels states to conform to pressures regarding the science of unhealthy body mass resulting in isomorphic state-enforced action to curb obesity.

My findings indicate that, for most countries, economic integration reduces body mass, though agricultural trade openness was instrumental in increasing body mass across most regions, Africa and Asia in particular. Overwhelmingly foreign investment

decreased body mass especially in lower-developed regions. For example, in regional analyses, Africa and Asia experience much stronger negative effects of FDI and value-added agricultural output than other regions, particularly North America and Europe. Also, economic integration for the most part complements the effects of civil society. Overall, these effects are much smaller, but I argue that civil society is a reasonable means to implement change in rising body mass.

Civil society memberships are theoretically presumed to commence independently of the state and market in influencing population-level health. However, the results shown in Appendix B reveal a different story. The analysis described there produced theoretically interesting results even if only originally designed as a check on sample size. The presence of certain types of civil society organizations may indicate a niche for profitable investments. For example, OIGOs appear to influence the effect of FDI on body mass. I find that when comparing regional analyses, controlling for OIGO membership changes a negative main effect of FDI on BMI to a positive one, but OINGOs wholly nullify the significance of the main effect of FDI. Similar results exist for value-added agriculture. However, in the only instance of a positive influence of economic integration on BMI, OINGOs do not substantively affect the input of trade openness. OIGO controls, on the other hand, convert a positive main effect of trade openness on BMI to a negative one. In this way, IGOs may act as a signal for the need for investment opportunities where body mass may be problematic. OINGOs do not appear to substantively influence the effect of economic integration. This may be indicative of a nonindependent relationship with market forces. In other words, OINGOs may be present because of newly opened market niches for investment. This type of interaction, while

not tested explicitly, has been insinuated elsewhere (Beckfield 2003; Beckfield 2008).

The differential effects of OIGOs and OINGOs lead to other conclusions. In particular, the effects of OIGOs are more pronounced in Europe and North America than Africa and South America despite having greater memberships in the latter regions. Unfortunately, these data do not speak clearly as to why IGOs and INGOs related to obesity perform differently by region, though there might be some hints. Practically speaking, world society is West-centric in ways that reproduce material inequality, though its theoretical claims tend to be egalitarian (Beckfield 2008). Europe and North America contain many of the global political bodies of which OIGOs make part. For example, headquarters for the World Bank and the International Monetary Fund are located in Washington, D.C. Many critiques of these two organizations in particular involve how the financial policies prescribed to poor nations reflect the interests of wealthy donor states (Stiglitz 2003). Furthermore, the world society presumes to impact states, in particular, various states that take part in IGO programs to convey their legitimacy and willingness to comply with international norms. This contributes to disproportionate action in two ways. First, the proximity to international bodies located in Europe and North America contribute to greater accountability in these regions (Meyer and Jepperson 2000; Smith and Wiest 2005). Second, civil society functions best through well running social democracies (Anheier, Glasius and Kaldor 2001). This would explain why civil society favors Europe and North America. In addition, the decoupling of policy goals and responses is relatively common among areas lacking sufficient resources (Cole 2006). Within the developing world, it is likely that insufficient state resources prohibit OIGO efforts to effectively deal with local obesogenic influences such as implementing policy

to alleviate heart disease or diabetes. These disproportionate effects could be reflective of the type of state. Unfortunately, data for testing the influence of state policies on BMI is poor, though Tsutsui et al. (2004), among other world society scholars, indicate differential effects of INGOs and IGOs on local politics. Furthermore, more sense could be made from North America's zero net effect of OIGOs. The contentious debates regarding obesity's framing and definition have contributed to political stalemates about the proper role of the state in personal behavior (Brownell and Warner 2009; Nestle 2002). As such, this net effect of IGOs in North America could reflect political reactance against OIGO advocacy efforts. Whatever the case, the point here is that these data show differential effects of IGOs and INGOs – a scenario which has not been seriously engaged in world society literature to date (Cole 2011).

The results of Chapter 2 produce a few general conclusions. First, one of the aims of this chapter was to assess the assertion among global obesity scholars that the current obesity epidemic is a manifestation of global convergence (Drewnowski and Popkin 1997; Hu 2008; Iqbal et al. 2008). As my research shows, body mass convergence occurs in ways that challenge typical thinking in cross-national obesity research. Investment actually contributes to lower body mass the world over but mostly in Africa, Asia, and South America – precisely where many countries are facing rising obesity risk. This finding places the obesity literature in the intersection of two trajectories. On the one hand, the FDI literature – particularly in the dependency school – has a long track record of documenting negative outcomes across a variety of settings, with lower body mass now more firmly among them (Alderson and Nielsen 1999; Chase-Dunn 1975; Kentor and Boswell 2003). On the other hand, rapid and expansive investment has produced

weight gain in many countries (Hawkes 2005; Khor 2012; Rayner et al. 2006). It should be noted that in my project, FDI was a broad measure of total investment, and that food trade specific measures, in two instances, positively contributed to higher body mass. The issue for international obesity scholarship, then, is how to frame obesity as the product of multiple global convergences when broad measures of convergence (FDI, food trade, and even food consumption) indicate divergence. Put another way, if only refined measures of investment produce favorable results for the obesity-convergence hypothesis, in order for obesity scholarship to take globalization theories more seriously – or vice versa – some kind of reconciliation must occur; e.g., investment, does or does not produce obesogenic environments. Second, Chapter 2 also showed that BMI is consistent with other well-being outcomes in cross-national studies like income inequality (Chase-Dunn 1975), class formation (Boswell and Dixon 1990), over-urbanization (Timberlake and Kentor 1983), mortality, and infant mortality (Volker and Chase-Dunn 1985). In this case, FDI produces lower body mass disproportionately and presumed egalitarian measures of convergence such as the influence of civil society that lowers body mass, are not egalitarian in practice. Thus, as an outcome of global convergence, body mass appears to function compatibly with findings in other studies of global convergence, it just so happens that body mass does not appear to be a good measure of convergence in the way typically articulated by international obesity scholarship.

This project also aims to more clearly articulate where obesity scholarship can benefit from theoretical redefinition. The focus of each chapter intended to target typical thinking about global obesity and explain how those assumptions are insufficient. In Chapter 1, I describe the process of obesity among the urban poor, while in Chapter 2, I

attempted to refine thinking about global obesity convergence mechanisms. Together, these studies inform global obesity scholarship in two particular ways: first, clearer implementation of how globalization actually occurs can improve the study of obesity, and second, an appreciation of the uneven nature of global economic development leading to obesity convergence could improve analysis on obesity pathways.

The scholarship of global economic development has long identified contradictions. National development and industrialization has been argued to have originated out of the colonial era, idealistically, to improve humankind. According to one view, the push by wealthy nations to shift economic specialization from agricultural to industrial products would infuse technology into poor countries and improve their overall well-being (McMichael 2003). However, through the 1980s, the ideological shift towards neoliberal economic policy directed poor countries to integrate into the world marketplace through economic privatization and liberalization. These policy orientations were not without their problems as they have been shown to increase urban impoverishment and economic inequality among developing nations (Breman and Shelton 2007; Gilpin 2001; McMichael 2003; Schrecker and Labonte 2007; Stiglitz 2003). The trajectory of economic development is not even, but the main assumptions used in explaining the global obesity epidemic have primarily been linear – more agriculture, more food, more trade, more investment, more obesity. For example, the rise of FDI as a means of increasing global production has contributed to more goods, but in terms of obesity, it serves to decrease body mass in some regions. This essentially indicates what the dependency school has argued all along – FDI contributes to sub-optimal social and economic outcomes among developing countries. However, because

of the obesity literature's epidemiological myopia, it has posited that obesity is the result of additional calories. The contribution FDI mainly makes to obesity is indirectly through malnutrition (Breman and Shelton 2007; Smith 2005). My results show that not only does FDI not increase total food consumption, but that it decreases BMI in every geographic region. My results also show an increased risk of obesity prevalence among malnourished children. In that sense, FDI is driving the global obesity epidemic through social and economic inequality, not the way obesity scholars typically argue. Hence, if obesity is expected to be the direct outgrowth of economic development, we have started off on the wrong assumption.

Increased hunger across the world, particularly in poor areas, has been well documented (UN 2012; UNHABITAT 2010). Economic liberalism has been suggested as one of the main causes of that hunger primarily through the inequality typically associated with its initial phases and its subsequent economic bifurcation (Breman and Shelton 2007; Kuznets 1955; McMichael 1996; McMichael 2003). However, because obesity is traditionally conceptualized as an illness of overabundance, few researchers have considered that obesity may actually be the product of under-abundance such as the case of malnourished children, as I demonstrate. This challenges our understanding of how to define obesity risk. If obesity risk among poor countries actually begins with malnutrition but is primarily conceptualized as *excess* weight negatively affecting health, then our notion is already misleading. Clearly more theoretical rigor is needed.

Limitations

There are various limitations in this study. In this section, I discuss the various levels at which this study confronts challenges and how I address their impact.

Unfortunately, international attention to obesity has not been accompanied by appropriate, consistent data collection. As such, even the best data often under-represent the extent of the obesity problem. In every analysis, the issue of sufficient data introduces questions about the extent to which findings could be artifacts of mere sample size. I deal with this problem in several ways. First, the emphasis of Chapter 1 is on the role of the key independent variables urban slum prevalence and childhood stunting. By focusing on independent rather than dependent variables, the emphasis is on showing consistent theoretical predictions across multiple outcomes to ascertain the extent to which the findings based on one theoretical interpretation could be considered replicated on another outcome. For example, one research question in Chapter 1 asks whether or not malnutrition plays a role in the obesity outcomes of poor countries. In using obesity prevalence, the number of cases on this dependent variable is commonly under 40 – often too close to the acceptable limits of normality assumptions. The same analytical design is therefore applied to average BMI, in many instances increasing cases by tenfold. The goal of introducing comparable dependent variables for this question, however, requires re-interpreting how each theory of obesity onset would explain outcomes on each respective dependent variable. This type of inference, then requires additional analyses to ascertain from a third dependent variable the validity of the inferred theoretical claims – hence the use of food consumption as a robustness check. In that analysis, I find no statistical significance on consumption by urban slum prevalence per se, hence, I rule out

the predictive claim of the food security hypothesis that slum dwellers drive obesity epidemics in poor countries. Relying on additional hypotheses introduces its own problems, but in using "triangulating" dependent variables, the importance of my findings becomes clear – poverty primarily contributes to global obesity through childhood malnutrition by predisposing the undernourished living in poverty to obesity as they grow older. Using multiple dependent variables, then serves to strengthen my interpretations as they are corroborated across all outcome measures. This approach is admittedly nontraditional but the novelty of the question and the data problems certainly justify using unconventional methodology.

The second way in which I deal with a restricted sample size is in comparing models of low sample size to those where higher sample sizes are available. In Chapter 2, I use slope-dummy variables to test the influence of independent variable "classes" which differ in sample frames. International nongovernmental organization and IGO memberships limit the number of cases included in my analysis by as much as a factor of 19 relative to FDI, value-added agriculture, and agricultural trade openness. Given the research question of this analysis – which class of variables plays a stronger role in obesity – the differential in sample size alone confounds the validity of the results. In Appendix B, I limit samples of economic integration to a similar frame as OIGO membership. In this instance, I find that except for only one test variable, the regional effects of economic integration on average body mass are consistent net of sample size. In other words, the significance of FDI and agricultural trade openness on BMI is not based on sample size, but true effects. Using a more detailed civil society measure thus is appropriate in two ways. First, few world society analyses attempt to conceptualize the

mechanisms of civil society occurring differently. This disaggregation shows that each type of civil society network is functioning through different mechanisms. In the case of my analyses, IGOs appear to be paving the way for market entry. Doing so opens questions about the applicability of treating all types of civil society organizations the same. Second, my robustness checks validate this method as an approach that produces theoretically relevant results.

This project is limited in other ways. Average BMI used at the national level also masks a great deal of heterogeneity among the populace. Some controversy has arisen in recent years regarding the applicability of BMI cut-off scores to all populations, notably Asian populations. According to a WHO Expert Consultation (2004), evidence suggested that cardiovascular and diabetes risk was lower among Asian populations compared to the typical "overweight" classification of 25 kilograms/meter^2 or higher, but that also, no clear cut-off score was necessarily more appropriate. Thus, as they argued, WHO classifications for overweight and obese should be used for international classifications. However, using BMI may mask pockets of wealth where BMI may be disproportionately high or low. Having a broad measure in itself protects against the effects of outliers. BMI can be a cumbersome measure of body mass, but it is best suited as an indicator of trends given that few people or countries, for that matter, suddenly find themselves overweight. Using BMI longitudinally is one way which I control for the nature of BMI in the modeling rather than the variable itself. Perhaps the biggest limitation is that BMI may be a faulty measure, nonetheless, it is the basis for most studies in obesity.

More fundamentally, the level of analysis is not commensurate with the level of measurement of the variables. Obesity on the global level is prone to this problem

because of the ways in which obesity is measured. On the individual level, BMI can be a poor proxy for obesity because of the wide array of variations in body type, racial and gender differences, and fat density all of which can interfere with obesity classification. Still, BMI is also highly correlated with anthropocentric measures of body fat and is valid at group-level aggregations (Khosla and Lowe 1967). Together with the close association with more direct measures of obesity, at the population level, regression to the mean evens out those cases where BMI is unrepresentative of true obesity.

Because BMI is the primary method of diagnosing obesity, problems further escalate when considering population measures of obesity. Obesity prevalence is based on the number of persons with BMI of 30 or over. Relying on obesity prevalence as a national level variable makes sense, but only if we can assume that national obesity is comparable. In other words, using obesity prevalence makes assumptions regarding the racial composition of a country's population. When Pacific Islanders are compared with the Japanese, illustrating the extremes of the BMI spectrum, this includes a host of factors which BMI does not necessarily control for. At the global level, in using BMI or obesity prevalence, we are assuming too much commonality across national, cultural, genetic contexts. Fortunately, fixed effects estimation capitalizes on differencing out the constant, fixed effects. One might assume that the national, genetic and cultural variations which introduce problems surrounding BMI at a high level of aggregation, while not controlled for in the actual variable itself, are accounted for through the mode of estimation.

Other data limitations exist in finding suitable measures of body mass influence at all levels of analysis. This is one problem that makes studying obesity particularly challenging. Unfortunately, data such as physical activity, meals eaten outside the home,

or even income inequality are not widely available to complement analyses above the national level. Paired with equally problematic challenges of obesity measurement and aggregation, parsimony is perhaps the best solution for this phase of theoretical clarification.

I also confront a common problem in cross-national research involving economic development such as collinearity among predictor variables and endogeneity, but separating the effects of economic development from those of the world society may not be operationally feasible. In Appendix A, I explore acceptable means to overcome these limitations through the use of instrumental variables and alternative development measures. There, I use robustness checks to verify that the strength of my main development measure – GDP in this case – may be overshadowing the more nuanced effects of civil society. Results of those tests indicate that treating endogeneity as a source of statistical interference distracts away from the more fundamental problem of collinearity. Suitable instrumental variables are so highly correlated with economic development that introducing the subsequent autocorrelation interferes with efficient regression estimation. For instance, diagnostic tests show that instrumental variables produced unstable standard errors commonly associated with collinearity. Also, the effects of OIGOs and OINGOs varied by the strength of economic development controls indicating that there were effects independent of development. However, GDP as an indicator of economic development has been firmly established as a staple of cross-national research and removing it due to the collinearity it reflected with civil society membership variables is not possible. Furthermore, I find that linear fixed effects modeling was the most efficient technique to control for collinearity. Ultimately then, the

key problem lies in operationalizing key concepts to the world society in ways that clearly delineate the roles of civil society independent of economic development. I use established world society measures in this project, albeit to a more specific degree than commonly attempted. Thus any challenges these analyses expose will ideally contribute to refining the operationalization of civil society concepts as a whole.

Future Research

One of the main conclusions from this project has been that clearer theoretical articulation is needed in obesity scholarship to integrate how global obesity researchers and public health officials can make better use of the economic development and globalization literature to improve obesity trajectories in high risk countries. A somewhat surprising outcome of this research was that obesity is connected with more nuanced aspects of global economic development such as the persistent negative effects of FDI. This unexpected turn prompts other questions about the assumptions of the global obesity epidemic in other similar areas. For example, one the drivers of obesity has been leveled at urbanization where international goods and services are most available, but also where economic marginalization is likely to occur. Still, rural poverty and obesity are also becoming more likely. What differences exist between rural and urban obesity? Does malnutrition in rural settings predispose the poor to obesity in the same way as in urban settings?

In another vein, the concept of food deserts illustrates how low quality food options have increased obesity rates among the urban poor, in the United States and Europe, where marketing limitations relegate food choice to the most inexpensive and

calorie dense. My findings indicate that among the urban poor in poor countries, malnutrition predisposes obesity despite no differences among actual caloric consumption. Can food deserts exist in entire cities, or countries because of the overall lower ability to market and sell foods? This particular application might examine how poor countries may or may not have been "peripheralized" in the food economy because of their economic status. The burden of global obesity-related illnesses is only expected to increase. Thus, identifying other social and economic contexts for its growth would be a beneficial avenue to explore.

One valuable contribution this project has made to the wider globalization literature has been the use of category specific IGO and INGO membership as a means of testing their relative impacts. I find that IGOs have had different results from INGOs, particularly among lesser developed regions which challenges the majority of findings regarding civil society among world society literature. As I argued in Chapter 2, contextual factors that define the scope of category-specific civil society organizations appear to matter, such that organizations differ in their goals depending on their country of operation. Allowing room for differences in the type of organization and how it operates locally is a departure from how these measures are typically used and brings several related questions. First, how much can intergovernmental organizations be tied to prosocial outcomes across the spectrum of state strength? My findings indicate IGOs' effectiveness is loosely tied to lower developed regions, but this is based on the way IGOs in these regions are associated with improving social problems. Second, what is their role in more developed countries where infrastructure exists more explicitly for alleviating social problems? The premise of this question rests on how a problem can be

experienced differently across countries. Illness, for example, has a wide variety of outcomes depending on where a person lives. In all contexts, it represents a challenge, but it can be a matter of survival among many poor nations. Thus, in the case of obesity, the public response has been shaped so heavily by political interpretations of responsibility, the state appears to be playing a very different role mediating its effects in wealthy versus poor countries. It stands to reason that not all countries interpret obesity in this way, and there may be differences in health policy responses among states. Third, in the context of "decoupling" where the capacities of civil society are incompatible with its standards, inaction by the state may be the result of the inability to enforce civil society recommendations, or may also stem from an unwillingness to accept particular types of civil society influence due to a problem of framing or political will.

More broadly, the associations between income inequality and health are diverse. In particular, the classic Whitehall study in social epidemiology posited that health differentials functioned primarily through individuals' sense of autonomy (Marmot 2006). In addition, findings on the effects of stress indicate that high efforts and high rewards are generally health promoting while high effort with low or no rewards are health discouraging (Bosma et al. 1997; Siegrist 1996). Individuals in circumstances where they are unable to succeed or advance for long periods of time can precipitate a deterioration in health. Is the growth of obesity another notable outcome of perceived powerlessness and stress? The idea here is that because obesity is so closely tied to economic status, a link between even the perception of economic mobility and health may be playing a role in obesity onset through the psychological nature food plays in dealing with stress. This question may expose more fully how symbols of success are changing in society.

The burden of pursuing this research lies in strengthening the connection between the personal to the global. More creative modeling may be a fruitful avenue of research. For example, multilevel modeling may placate skeptics who argue against the applicability of using an international theory of obesity to elucidate individual level obesity outcomes. As Luke (2004) explains, society comprises of both collectives and members, and information regarding the higher level collective is obtained from lower-level members. Still, these properties do not prohibit them from being analyzed at the same time. Hence the problems of ecological fallacy – the inappropriate inference at the group-level to individuals, and atomistic fallacy – the inappropriate inference at the individual-level to groups – is a problem of inference rather than measurement. Multilevel modeling utilizes data collected at multiple levels in order to highlight relationships between those levels (Luke 2004). It capitalizes on context which has been illustrated as a key component in international obesity studies. One question in particular that arises from this analysis is the relationship of food access to obesity. Are there personal factors that interact with the nutritional environment that contribute to obesity resilience among the nutritionally disadvantaged? What role does geographic location play in providing food access given that FDI shows such a compelling negative effect on average BMI? Perhaps the most broad application of this research serves to anchor obesity literature to macrostructural global research. It has been said that Wallerstein's world systems theory originally grew out of a need to advocate for the underprivileged (Tilly 1981). This research situates itself among literature connecting health problems of today with economic hardships of yesteryear.

Concluding Comments

In this project I test competing narratives of the global obesity epidemic using cross-national longitudinal methods. In Chapter 1, I show how malnutrition plays a stronger role than economic development in growing obesity epidemics in poor countries. In Chapter 2, I evaluate obesity as the outcome of global convergence and find that contrary to assumptions in obesity literature, economic development has a negative effect on body mass, while civil society also appears to have negative effects.

In many ways, the search for the mechanisms that have triggered the increase in global obesity underscores the context of bleak health prospects for millions of people. I may have identified possible sources of obesity, but discovered them to be among a host of other serious problems. This project shows that the machine in which obesity functions is as big as the global economy. One positive note, however, is that social advocacy is likely to produce change. In the end, one might be hopeful that despite the host of negative externalities produced by global economic development, action taken by people on behalf of others leads to positive results.

APPENDIX A

EVALUATING TWO-STAGE LEAST

SQUARES MODELING AS MEANS

OF CONTROLLING

ENDOGENEITY

A notable concern in asking whether NGO membership plays a role in increasing body mass is the problem of endogeneity – the potential reciprocal relationship between the dependent and independent variables. Do NGOs in a country increase because of obesity, or does obesity follow from NGO membership itself? Furthermore, additional confounding exists because of unobserved variables correlated with fixed effects. In the case of unobserved heterogeneity bias, OLS assumptions are violated and regression estimates are biased. However, in using instrumental variables, one can use variables that are correlated with the endogenous variable but leaves the unobserved variance in the error term.

Endogeneity can be expressed by the following two equations; by expressing Y – endogenous variables – in terms of exogenous variables (z), and the error term (u)

$$Y_1 = \alpha_1 \, Y_2 + \beta_1 \, z_1 + u_1 \tag{1}$$

$$Y_2 = \alpha_2 \, Y_1 + \beta_2 \, z_2 + u_2. \tag{2}$$

If we assume $\alpha 2$ and $\alpha 1$ are not correlated, then the reduced form – called the first-stage regression – of (2) yields

$$Y_2 = {}_{\Pi 21} + {}_{\Pi 22} + v_2 \tag{3}$$

where

$$\Pi_{21} = (\alpha_2 \beta_1/(1-\alpha_2 \alpha_1)z_1 \tag{4}$$

$$\Pi_{22} = (\beta_2/1-\alpha_2 \alpha_1)z_2 \tag{5}$$

and

$$v_2 = (\alpha_2 u_1 + u_2)/(1-\alpha_2 \alpha_1). \tag{6}$$

Because u_1 and u_2 are uncorrelated with z_1 and z_2, v_2 is also uncorrelated with z_1 and z_2, thus one can consistently estimate $_{\Pi 21}$ and $_{\Pi 22}$ by OLS (Wooldridge 2002). Two Stage Least Squares (2SLS) makes use of instrumental variables through its correlation with a causal variable of interest, and its uncorrelated relationship with unobservable determinants of the dependent variables. Capitalizing on these respective associations is used to estimate unbiased beta coefficients. It is widely used in econometrics as a means to estimating parameters of linear simultaneous equations and solving omitted-variable bias (Angrist and Imbens 1995). 2SLS uses strictly exogenous covariates and predetermined endogenous covariates to eliminate the unit effect by first differencing variables. It then applies the instrumental variable estimation for the parameters of the lagged endogenous variable. However, using 2SLS introduces large standard errors, increasing the chance of making a Type I error. Using 2SLS requires at least one instrument for every endogenous variable included in the model. When doing so, the model is referred to as exactly identified. As Murray explains (2006), having at least as many valid instruments as endogenous variables is a necessary condition for identification. Over-identification increases R^2 in the first stage of 2SLS and thus produces standard errors closer to those of ordinary least squares.

I conducted a test for endogeneity for OINGOs and BMI by 1) treating OINGO and OIGO membership as a dependent variable and regressing it on control variables, 2) saving residuals, 3) entering the residuals for the original dependent variable (Brady, Beckfield and Zhao 2007; Wooldridge 2002). Results are presented in Table 26. Significant regression coefficients indicated positive endogeneity bias for OINGO membership, negative for OIGO membership and intermittent positive and negative bias for economic integration variables. These endogenous biases would suggest that not only are OINGOs and BMI potentially mutually causational, but that the bias indicates that NGO membership and economic integration skew towards higher BMI[29]. Results of endogeneity tests using fixed effects modeling techniques are reported here, in Appendix A. Thus for consistency, I use Two Stage Least Squares (2SLS) with fixed effects estimation as a means of correcting for endogeneity bias across all key test variables. Endogeneity test results are presented in Appendix A, Tables 26 and 27.

There are a number of potential problems in operationalizing the link between OINGO membership and body mass. Most importantly, the problem of collinearity among predictors introduces complications to unbiased estimates when using multivariate regression methodologies. Collinearity occurs when two or more variables are closely correlated. During estimation, this redundancy produces large standard errors that lower the likelihood of statistical significance. Bivariate correlations between variables above 70 create problems. Bivariate correlations between BMI and OINGO and OIGOs are reported as -.28 and .25, respectively. Instrumental variables, though, are required to be strongly correlated with independent variables, and weakly correlated with dependent

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[29] For consistency, I include both OIGO and OINGO in 2SLS models because of their theoretical similarity. Likewise, not all economic integration variables showed positive endogeneity bias, but for I use 2SLS for all tests to regardless of the direction of bias.

variables.

The problem these data present is in operationalizing key variables in such a way as to capture unique contributions of each concept that are not mutually exclusive. While predictors and outcomes may not be collinear, instruments that are used to control for endogeneity bias – used in estimating the effects of key predictor variables – are. For example, I use food as a percent of total merchandise imports as an instrument variable to endogenous variables – OINGO and OIGO. It was selected on the basis of a strong to moderate correlation with OINGO membership and a relatively low correlation with BMI. A common test of suitability of the instrumental variable is the Sargan-Hansen test of over-identification which essentially asks if any of the instruments are invalid. Food imports passed all over-identification tests indicating that it was a suitable instrument even in the case of its relatively low relationship with OIGO memberships. In this case though, the Sargan-Hansen statistic indicates that among the models where food imports is a weak instrument, identification does not pose a sufficient problem to invalidate the models. Still, passing diagnostic tests for the key predictor variable does not isolate the problem of collinearity among control variables. Food imports, for example, may have passed common diagnostics for suitable instrumentation, but its bivariate correlation with GDP is .93. In this case, using an endogeneity control in 2SLS actually introduces more problems than it solves because of noncollinearity requirements among all model predictors. GDP is the best measure of economic development available and controlling for its pervasive influence on other indicators challenges the degree to which any model can isolate its explicit or implicit influence on an outcome

To show how operationalizing development has introduced problems to this

particular analysis, I implement 2SLS using the -xtivreg- suite of commands in STATA and present first-stage, second-stage results of economic integration, OINGO and OIGO analyses in Appendix A, Tables 26 through 28, respectively. I focus on CSOs to illustrate the constrictive nature 2SLS imposes on these data. I also include less stringent linear fixed effects models to test the relative influence of different operationalizations of development. These models are intended to show that because development is a multifaceted concept, parsing out its effects poses substantial methodological challenges when attempting to isolate the effects of apparently more sensitive civil society influences.

Appendix A, Table 27 shows the results of first-stage models using instrumental variables in estimation. Models 1 and 2 test the role of OIGOs adding control variables. In models 3 and 4, I test the influence of OINGOs on BMI. For OIGOs the effect of food imports is nonsignificant and net of controls it is likewise nonsignificant for OINGOs. Adding the instrument to account for the role of endogeneity does not substantively contribute to model estimation.

Table 28 shows the results of second stage fixed effects regression of OINGOs and OIGOs on BMI. OIGOs are not significant with or without control variables. OINGOs show strong positive associations, but when including control variables the effect drops to null because of a substantially higher standard error, and a much lower sample size. One possible reason for why OIGOs and OINGOs are showing such differences is because of the distribution of memberships according to development status. Beckfield (2003) found that Western states have increased their ties to IGOs and INGOs more than poorer non-Western states, and that these relationships have strengthened for INGOs but

weakened for IGOs. OIGOs being more egalitarian, and less associated with development are showing no significant effects, whereas INGOs, which are more associated with development, do show positive significance.

In this case, 2SLS is a cure for endogeneity that is worse than the illness. It seems that 2SLS is overly restrictive because of the use of instrumental variables. As indicated by endogeneity tests, the problem of mutual causation is present among these tests. However, the relationship of the variables themselves plays a role in how endogeneity is affecting the outcome of these tests. Both first and second stage result models of OINGO influence on BMI indicate that model parameters are estimated properly. However, when including economic development controls, shown in Table 28, the effect of OINGOs drops from a relatively large magnitude and highly significant, to a relatively low magnitude and nonsignificant. Using the instrumental variable to to control for endogeneity appears to be obscuring the more pressing issue that GDP is confounding the effects of OINGOs.

To test this possibility, I tested the same models using linear fixed effects estimation without the inclusion of an instrumental variable. I tested whether the effect of "development" was masking the influence of CSOs on BMI in 2 ways: first, running all models without controls; second, with GDP as the only control. Results indicated that GDP was overwhelming the effect CSOs as Appendix A, Table 29 indicates. First, in Appendix A, Table 29 shows GDP accounted for 38 percent of the effect on BMI but left the effect of IGOs largely unchanged. This corroborates evidence presented by Beckfield regarding the more equal distribution of OIGOs across the developmental spectrum. Second, among OINGOs, GDP accounted for about 50 percent of the effect BMI and

reduced a substantial effect from OINGOs to nonsignificance. In both cases, adding additional controls substantially reduced the sample size which likewise plays a role in these changing effects.

The upshot of these diagnostic tests indicate that when using a less defined measure of economic development as a control, the influence of other variables is present. When GDP is included, it is the strongest influence – particularly in the INGO analysis, but also to a lesser degree in the IGO analysis. In addition, 2SLS does not add additional specificity to model parameters over a linear fixed effect model. Essentially, it is operationalization of "development" which is contributing, first to collinearity problems rather than endogeneity problems, and subsequently, second, Type I errors about the nature of civil society influence.

Table 26
Endogeneity Tests using Fixed Effects Modeling
of Keys Independent Variables, 1981 – 2007
Dependent Variable: BMI

Variables	1	2	3	4	5	6
Food Imports	-0.00713					
	(0.0251)					
Food Imports Residual	1.591***					
	(0.0628)					
FDI		0.0430				
		(0.0298)				
FDI residual		1.766**				
		(0.551)				
Value-Added Agricultural Output			0.0213			
			(0.0354)			
Value-Added Agricultural Output Residual			-0.872***			
			(0.0567)			
Agricultural Openness				0.0135		
				(0.0248)		
Agricultural Openness Residual				1.678***		
				(0.334)		
OIGO membership					-0.230***	
					(0.0698)	
OIGO membership residual					-0.164	
					(0.300)	
OINGO membership						0.0289
						(0.0235)
OINGO membership residual						1.149***
						(0.0923)
Constant	-0.157***	-0.196*	0.0730***	0.250***	-0.127	0.102***
	(0.0149)	(0.0999)	(0.00638)	(6.87e-05)	(0.120)	(0.0174)
Observations	1,710	1,869	1,851	1,708	94	469
R-squared	0.852	0.398	0.730	0.152	0.453	0.541
Number of id	145	168	165	145	41	150
Rho	0.999	0.996	0.987	0.995	0.972	0.989
Btween	0.0154	0.00213	0.474	0.370	0.172	0.0695
Within	0.852	0.398	0.730	0.152	0.453	0.541
Overall Rsq	0.00508	0.0127	0.449	0.331	0.170	0.0851

Notes: Standardized regression coefficients. Robust standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 27
First Stage Results of Fixed Effects Two Stage
Least Squares Model, Obesity oriented
CSO Membership, 1981 – 2007.
Dependent Variable:
OIGO (1 and 2)
and OINGO
(3 and 4)
membership

Variables	1	2	3	4
GDP		0.976		1.111
		(0.976)		(0.698)
Population		1.617		-1.556
		(3.277)		(1.565)
Time		-0.102***		0.0253*
		(0.0360)		(0.0128)
Food import	0.449	-0.666	0.607***	-0.165
	(0.302)	(0.489)	(0.105)	(0.289)
Constant	0.374**	206.5***	-0.103***	-50.10**
	(0.163)	(73.01)	(0.0361)	(25.11)
Observations	253	67	777	405
R-squared	0.014	0.594	0.080	0.168
Number of id	108	26	144	128
Rho	0.609	0.889	0.484	0.919
Btween	0.209	0.0349	0.697	0.0184
Within	0.0139	0.594	0.0801	0.168
Overall Rsq	0.0903	0.102	0.502	0.0350

Notes: Standardized regression coefficients.
Robust standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 28
Second Stage Results of Fixed Effects Two Stage
Least Squares Model, Obesity Oriented CSO
Membership, 1981 − 2007.
Dependent Variable: BMI

Variables	1	2	3	4
OIGO	1.908	0.0251		
	(1.308)	(0.115)		
OINGO			1.599***	0.283
			(0.270)	(0.510)
GDP		0.605**		-0.0517
		(0.279)		(0.532)
Population		-0.457		1.542
		(0.507)		(1.067)
Time		0.0341***		0.0193
		(0.0130)		(0.0142)
Observations	198	60	772	393
R-squared	-29.577	0.932	-5.653	0.609
Number of id	53	19	139	116

Notes: Standardized regression coefficients. Robust standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 29
Sensitivity Analysis on the effects of Development
Fixed Effects Model, Obesity Oriented CSO
Membership, 1981 – 2007.
Dependent Variable: BMI

Variables	1	2	3	4	5	6
OIGO	0.0552**	0.0513**	-0.00774			
	(0.0255)	(0.0222)	(0.0168)			
OINGO				0.161***	-0.0146	0.00390
				(0.0201)	(0.0151)	(0.00969)
GDP		1.397***	0.695***		1.888***	0.206*
		(0.221)	(0.0719)		(0.140)	(0.106)
Population			-0.491			0.901***
			(0.591)			(0.217)
Time			0.0310***			0.0267***
			(0.00483)			(0.00265)
Constant	-0.343***	0.590***	-61.96***	0.119***	-0.511***	-53.60***
	(0.00421)	(0.141)	(9.811)	(0.00161)	(0.0487)	(5.226)
Observations	325	289	83	878	849	456
R-squared	0.024	0.615	0.949	0.074	0.691	0.913
Number of id	133	122	33	172	168	146
Rho	0.904	0.985	0.994	0.882	0.989	0.998
Within	0.0239	0.615	0.949	0.0740	0.691	0.913

Notes: Standardized regression coefficients. Robust standard errors in parentheses.
*** $p<0.01$, ** $p<0.05$, * $p<0.1$

APPENDIX B

ROBUSTNESS CHECK ON OIGNO

AND OIGO SAMPLE SIZES

A robustness check of the slope-dummy analyses were run in order to compare the effects of sample size and control variables on BMI across the various key independent variables. In the following appendix, additional analyses were included which examined the role of key independent variables in slope-dummy analyses including a similar sample from the original "general effects" models shown in Table 17. It was reasoned that without such tests, the regional findings from slope-dummy analyses had limited generalizability given the distinct differences in sample sizes across each test. I tested how each key independent variable interacted by region, controlling for other variables of interest. OIGOs and OINGOs were run in separate models because of limitations in sample size by attempting to include them in the same model.

Results

Table 30 shows the results of OIGOs on BMI net of control variables. Remarkably, given the additional statistical economic integration controls, the results are quite similar except for slight variations in beta coefficient magnitudes.

Table 31 shows the effect of OINGOs on BMI net of economic development

controls with comparable samples as the general effects model. Again, the effects of OINGOs on BMI are markedly similar with and without control variables.

Table 32 contains the effect of FDI on BMI using four models to compare the effects of sample size. Models 1 and 2 are repeated from Tables 19 and 22, respectively, including a full sample. Models 3 and 4 show models with similar samples from analyses of OIGO and OINGO "general effects" models. In particular, models 2 and 4 indicate the greatest amount of similarity despite having a sample about 6 times smaller. However, the main effect of FDI is significant in the reduced sample (model 3) and so are the interaction effects. In model 3, the magnitude and direction of effects are roughly the same except in the case of South America where the direction changes from negative to positive and statistical significance reduces somewhat.

A similar approach of comparing the effect of sample size on value-added agricultural in BMI was followed in Appendix B, Table 33. This Table shows four models-- two repeated with full samples (models 1 and 2) and two models with reduced samples corresponding to controls for OIGO and OINGO membership. The main difference of the effect of sample size is that the main effect of value-added agriculture is not statistically significant for models net of controls.

In model 2, without additional economic and world society integration controls, the main and interaction effects are significant. Interestingly, without regional effects (model 1), the effect of value-added agriculture is not significant.

In the last Appendix B, Table 34, I show the effect of agricultural trade openness on BMI in the same method as above. The differences between these models is more

detailed than those above. First, controlling for OIGOs and other variables in model 3, the total effects of trade openness is overwhelmingly negative in all statistically significant regions (Africa, South America, North America and the reference region – Europe). This stands in direct contrast to a mostly positive total influence of openness in most regions. However, in model 4, controlling for OINGOs and other economic control variables, the effects are similar as those reported though only three regions show statistical significance – Europe, Asia, and North America.

Discussion

The additional models were included as a robustness check in two ways. First, countries included in these analyses were limited to those from the "general effects models." In so doing, I was able to compare the effects of key variables on a similar sample – making a comparison of the effects of key variables in the slope-dummy analyses more directly with "main effect" models. Second, these tests re-analyzed slope-dummy analyses using controls for the other key independent variables used in the analyses. In using control variables, the effects seen are more comparable to prior, more saturated models.

Tests of the effects of world society variables on BMI shown here are consistently similar to prior models in spite of additional control variables. These results are expected given the similar sample size and R squared values across the models. However, it is instructive to see the analogous total effects of OIGO and OINGO in more rigorous models. It appears that GDP is an adequate control variable for additional economic integration not explicitly captured in other control variables.

The robustness check of FDI, however, indicates that the regional effects of FDI on BMI are exposing true effects, shown in Table 19. The difference between the two analyses indicates that the effect of OIGOs is an important factor in influencing FDI which, when uncontrolled for, otherwise masks an important element of FDI. Controlling for OIGOs in this case, may be indicative of a more concentrated effect of investment in areas where BMI is considered a problem. In other words, OIGOs provide the incentive for FDI with respect to BMI. Furthermore, a related outcome is apparent in (Table 34) modeling the effects of trade openness on BMI. The fact that controlling for OINGOs across models 2 and 4 does not influence the main test variable indicates that OINGOs may not be acting independently of the global marketplace. It could be that with respect to these models, GDP in effect offers similar controls as does OINGOs because of incentives exposed through economic growth. In either case, controlling for CSOs – specifically OIGO memberships – appears to offer the cogent reason for the discrepancy.

Lastly, the effects of value-added agriculture are likely due to sample size. In models controlling for OIGO and OINGO memberships, the sample size is reduced substantially. Though the directions of individual regional effects do not substantively vary, the absence of a significant main effect nullifies any generalizability.

Together, the results of robustness checks are insightful as to the consistency of key variables on BMI. The only variable where the findings of regional analyses are suspect due to sample size are value-added agriculture. The effect of this variable may be subject to statistical distortion from large sample sizes. Additional control variables contribute to a more nuanced view of FDI net of sample size. This finding offers supplementary evidence to assertions made previously in this paper regarding the role of OIGOs. The

association between FDI and BMI is inverse, as reported in the "general effects model," but that effect also happens to be influenced by more state-oriented OIGOs, unlike OINGOs, when examined regionally.

Table 30
Robustness Check of Fixed Effects Slope-Dummy
Analysis of OIGO Membership on BMI
Reference: Europe

Variables	1	2
OIGO	-0.101***	-0.121***
	(0.0111)	(0.0125)
Region Interaction		
Africa	0.0672***	0.0846***
	(0.0168)	(0.0162)
Asia	0.181***	0.175***
	(0.0470)	(0.0296)
South America	0.0830***	0.113***
	(0.00361)	(0.0122)
North America	0.109***	0.135***
	(0.0149)	(0.0167)
Oceania	0.132***	0.144***
	(0.00800)	(0.00979)
Control Variables		
Year	0.0286***	0.0327***
	(0.00330)	(0.00314)
FDI		-0.0334***
		(0.0111)
Domestic investment		0.00853
		(0.0118)
Value-Added Agriculture		-0.0797**
		(0.0394)
Agricultural Openness		-0.0128
		(0.0115)
GDP	0.591***	0.383*
	(0.166)	(0.193)
Population	-0.128	-0.253
	(0.381)	(0.301)
Constant	-56.98***	-65.31***
	(6.657)	(6.341)
Observations	171	171
R-squared	0.933	0.942
Number of id	75	75
Rho	0.992	0.989

Notes: Standardized regression coefficients. Robust standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 31
Robustness Check of Fixed Effects Estimation
of OINGO Membership on BMI
Reference: Europe

Variables	1	2
OINGO	-0.0918***	-0.0650**
	(0.0326)	(0.0303)
Region Interaction		
Africa	0.0713***	0.0505**
	(0.0233)	(0.0226)
Asia	0.0276	0.00573
	(0.0347)	(0.0324)
South America	0.0937***	0.0750***
	(0.0259)	(0.0252)
North America	0.0886***	0.0676**
	(0.0296)	(0.0333)
Oceania	0.0334	0.00825
	(0.0546)	(0.0502)
Control Variables		
Year	0.0316***	0.0346***
	(0.00481)	(0.00523)
FDI		-0.0552***
		(0.0174)
Domestic investment		-0.00202
		(0.0118)
Value-Added Agriculture		0.0510
		(0.0465)
Agricultural Openness		0.0121
		(0.0264)
GDP	0.174	0.258
	(0.181)	(0.196)
Population	0.445	0.293
	(0.335)	(0.367)
Constant	-63.31***	-69.24***
	(9.458)	(10.29)
Observations	533	533
R-squared	0.889	0.896
Number of id	126	126
Rho	0.989	0.989

Notes: Standardized regression coefficients. Robust standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 32
Fixed Effects Estimation of FDI on BMI
Reference: Europe

Variables	1	2	3	4
FDI	-0.0389***	0.0333	0.0613***	-0.0496
	(0.0115)	(0.0259)	(0.0218)	(0.0423)
Regional Interaction				
Africa		0.189***	0.338***	0.164*
		(0.0602)	(0.0624)	(0.0853)
Asia		0.0745***	0.0734***	0.0887***
		(0.0152)	(0.0145)	(0.0226)
South		-0.0341***	0.0138*	-0.0421***
		(0.0110)	(0.00740)	(0.0150)
North		-0.0387***	-0.0216***	-0.0339***
		(0.00657)	(0.00745)	(0.00888)
Oceania		-0.0310*	-0.0931***	-0.0166
		(0.0160)	(0.0173)	(0.0233)
Control Variables				
OIGO			-0.00485	
			(0.00851)	
OINGO				-0.0154
				(0.0125)
Value-Added Agriculture			-0.0656	0.171**
			(0.0423)	(0.0666)
Agricultural Openness			-0.0202	0.0163
			(0.0143)	(0.0259)
GDP	0.407***	0.459***	0.344*	0.405**
	(0.132)	(0.122)	(0.197)	(0.185)
Population	0.152	0.273	-0.0460	0.0895
	(0.241)	(0.232)	(0.325)	(0.384)
Domestic investment	0.00155	0.00336	0.00417	0.00116
	(0.00773)	(0.00727)	(0.0108)	(0.0112)
Year	0.0291***	0.0265***	0.0303***	0.0356***
	(0.00296)	(0.00301)	(0.00283)	(0.00542)
Constant	-58.20***	-53.06***	-60.62***	-71.19***
	(5.868)	(5.984)	(5.747)	(10.66)
Observations	3,429	3,429	176	539
R-squared	0.869	0.882	0.947	0.897
Number of id	163	163	76	126
Rho	0.988	0.991	0.993	0.988

Notes: Standardized regression coefficients. Robust standard errors in parentheses.
*** $p<0.01$, ** $p<0.05$, * $p<0.1$

Table 33
Fixed Effects Estimation of Value-Added Agriculture on BMI
Reference: Europe

Variables	1	2	3	4
Value-Added Agriculture	0.0429	-0.119**	-0.477	-0.0691
	(0.0390)	(0.0556)	(0.302)	(0.0764)
Regional Interaction				
Africa		-0.810***	-1.325**	-0.901***
		(0.148)	(0.642)	(0.254)
Asia		-0.234***	-0.267***	-0.228***
		(0.0321)	(0.0692)	(0.0436)
South America		0.0822***	0.0580	0.0904***
		(0.0199)	(0.0397)	(0.0244)
North America		0.0994***	0.111	0.0808***
		(0.0153)	(0.0924)	(0.0231)
Oceania		0.137***	0.324**	0.138*
		(0.0403)	(0.130)	(0.0727)
Control Variables				
OIGO			0.00137	
			(0.00839)	
OINGO				-0.0101
				(0.0119)
FDI			-0.0427**	-0.0585***
			(0.0185)	(0.0152)
Domestic investment			0.0113	0.00369
			(0.0116)	(0.00949)
Agricultural Openness			-0.0194	-0.00765
			(0.0135)	(0.0231)
GDP	0.641***	0.651***	0.395**	0.344*
	(0.109)	(0.105)	(0.163)	(0.196)
Population	0.453*	0.218	-0.318	-0.0322
	(0.258)	(0.253)	(0.308)	(0.337)
Year	0.0215***	0.0234***	0.0330***	0.0357***
	(0.00245)	(0.00264)	(0.00341)	(0.00471)
Constant	-43.08***	-46.82***	-65.90***	-71.22***
	(4.844)	(5.224)	(6.926)	(9.258)
Observations	4,399	4,399	180	541
R-squared	0.828	0.858	0.935	0.910
Number of id	178	178	77	127
Rho	0.992	0.992	0.993	0.987

Notes: Standardized regression coefficients. Robust standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 34
Fixed Effects Estimation of Agricultural Trade
Openness on BMI Reference: Europe

Variables	1	2	3	4
Agricultural Openness	0.0228	0.111**	-0.297***	0.133*
	(0.0150)	(0.0444)	(0.0759)	(0.0779)
Regional Interaction				
Africa		0.302***	-0.339*	0.216
		(0.101)	(0.189)	(0.201)
Asia		0.0980***	-0.0182	0.0892**
		(0.0257)	(0.0314)	(0.0450)
South		0.00524	0.0607***	0.0135
		(0.0180)	(0.0201)	(0.0237)
North		-0.0146	0.0867***	-0.0342**
		(0.0170)	(0.0263)	(0.0171)
Oceania		-0.103***	0.0500	-0.0924
		(0.0257)	(0.0378)	(0.0595)
Control Variables				
OIGO			0.00530	
			(0.00856)	
OINGO				-0.0254**
				(0.0118)
FDI			-0.0330*	-0.0501***
			(0.0172)	(0.0151)
Domestic investment			0.00878	0.00346
			(0.0128)	(0.00969)
Value-Added Agriculture			-0.0666	0.140**
			(0.0527)	(0.0686)
GDP	0.521***	0.517***	0.399**	0.214
	(0.144)	(0.136)	(0.188)	(0.188)
Population	0.349	0.434*	-0.563*	0.221
	(0.241)	(0.226)	(0.317)	(0.324)
Year	0.0236***	0.0225***	0.0348***	0.0367***
	(0.00294)	(0.00274)	(0.00359)	(0.00425)
Constant	-47.35***	-45.15***	-69.46***	-73.34***
	(5.799)	(5.424)	(7.283)	(8.365)
Observations	4,078	4,078	180	543
R-squared	0.838	0.858	0.926	0.898
Number of id	153	153	77	127
Rho	0.989	0.991	0.987	0.990

Notes: Standardized regression coefficients. Robust standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

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