

# Macrosocial Determinants of Population Health

Sandro Galea

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With 31 Illustrations

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Sandro Galea

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# Introduction

# Chapter 1

## The Role of Macrosocial Determinants in Shaping the Health of Populations

Sandro Galea and Sara Putnam

### 1. Introduction

The roots of epidemiology, coincident with the origin of public health, lie in exploring how social conditions may influence health and how these conditions may be manipulated so as to improve the health of populations (Mc Leod, 2000; Halliday, 2000; Hamlin & Sheard, 1998). However, in the last half century, with the advent of antibiotics as treatments for infectious diseases, the shift from infectious disease to chronic disease considerations, and the focus on genetic determination of disease, epidemiologic inquiry has grown increasingly concerned not with the social determination of population health, but rather with the individual exposures or characteristics that influence individual risk of health and disease (March & Susser, 2006). It is the central tenet of this book that social factors that lie beyond the individual and that affect whole populations, factors that we term “macrosocial”, should remain central in our thinking about the production of health and disease, and that public health research and practice would be well served by an improved understanding of how these macrosocial factors shape population health. Setting the stage for the chapters to follow, in this introductory chapter we explore the challenges faced by most current inquiry concerned with the determination of health and argue that epidemiologic inquiry about macrosocial factors can help improve our understanding of population health and potentially guide the development of more effective public health interventions.

We note that this introduction, and this book, adopt very much an “epidemiologic” perspective. We mean this to refer to a central concern with the determination of health and disease and to inquiry aimed at understanding those factors that may influence health. Although the field formally constituted as “epidemiology” today is certainly most concerned with these questions, we do not mean to endorse an exclusive reliance on the methods of epidemiology and certainly do not intend to exclude the role of other disciplinary perspectives. As the chapters in this book amply illustrate, we suggest that disciplines such as economics, sociology, and health policy, among many others, play a central role in our understanding of the determination of health and of how those interested in the health of populations may fruitfully identify areas of intervention that can improve health.

## 2. Understanding the Determination of Health and Disease

The epidemiologic approach typically begins with interest in a particular disease or health indicator (e.g., diabetes or lung cancer). Concurrent with the identification of a disease, we rely on theory and prior research to identify a particular factor that may be associated with the disease. This factor is generally an individual “exposure” (e.g., a gene or mutation) or behavior (e.g., smoking). A study is then designed to determine whether there is an association between the particular factor of interest and the health outcome; once data is collected, statistical methods are employed to measure the association of interest while taking into account other possible alternate explanations.

If a rigorous epidemiologic study demonstrates an association that is biologically plausible and replicable in subsequent studies, we may venture to consider the factor in question a “cause” of disease and recommend an intervention to alter or eliminate this stated cause. Given that most modern epidemiologic research is concerned with individual behaviors or exposures, the recommended interventions are typically behavioral (e.g., smoking cessation) or pharmacologic (e.g., developing a drug to lower high cholesterol levels). This approach has arguably contributed to some of the most compelling public health success stories of the past half-century, including the identification of smoking as a risk factor for lung cancer and cardiovascular disease and low maternal folic acid intake as a risk factor for neonatal neural tube defect.

Nevertheless, there are clear conceptual and practical limitations to this dominant epidemiologic paradigm. A significant limitation is that the principal empiric tools for considering associations within study samples are best for research at the population level. Typical epidemiologic etiologic analysis calculates population rates and risk of disease and then estimates the relative rates and risks of disease in the presence and absence of a particular “exposure” of interest. While these absolute and relative rates and risks that are used to determine association are adequate representations of population-level disease occurrence, they tell us very little about individual risk of disease (Kleinbaum, Kupper, & Morgenstern, 1982; Rockhill, 2005). Statistical associations at the population level may be inconsistent with mechanisms (e.g., biological processes) occurring within individuals. This tension between epidemiologic methods of inference and individual risk is an intractable feature of epidemiologic inference based on population summary estimates and has contributed to three serious challenges facing public health inquiry today.

First, as originally and most forcefully articulated by Geoffrey Rose (1985), there are clear limitations of the epidemiologic approach in informing our understanding of the determination of individual health. Rose noted that many of our attempts to improve health are aimed at improving the health of persons at the tail end of a distribution of risk. For example, all medical screening for risk factors essentially aims to identify and intervene with “high risk” persons. There is no

attempt to reduce risk in the rest of the population, which is considered to be at “low risk” (or at least not at “high risk”). This approach might well be rational if (a) we could identify who is likely to develop disease simply by assessing their disease risk and (b) risk were binary, i.e., either present or absent. However, the first of these requisite conditions is false since our available methods of assessing where an individual sits on a risk distribution tell us little about individual likelihood of a particular disease (Pepe, Janes, Longton, Leisenring, & Newcomb, 2004; Wald, Hackshaw, & Frost, 1999). The second of these conditions is also false since ultimately exposure to risk factors is more likely continuous, and arbitrary cutoffs define and determine “high” vs. “low risks”. Populations characterized by levels of risk that are just below the “high risk” cutoff are likely at much greater risk of an adverse health condition than are populations whose risk is much lower than the cutoff, though both would be identified as “low risk”.

Second, an increasingly worrisome practical limitation is the preponderance of epidemiologic scrutiny focusing on the pursuit of single risk factors for disease in individuals. It is well established that with very few exceptions disease causation is multifactorial. However, our persistent epidemiologic focus on identifying single risk factors for individual disease has contributed to conflicting results from state-of-the-science studies that explore one particular aspect of causation while neglecting others. Unfortunately, the ever-changing catalog of risk and protective factors for disease documented in epidemiologic studies (e.g., the recent very public debate about the role of postmenopausal estrogen therapy) has occasioned substantial public confusion about the methods and conclusions of epidemiology and suggests that the quest for individual risks of individual disease may well be a reductionistic approach that has outlived its usefulness. In addition, as etiologic inquiry has become progressively more concerned with individual disease determination, this inquiry has also increasingly focused on determinants of disease that are, at least for the foreseeable future, immutable. The study of factors that predispose individuals to risk has increasingly involved genetic factors, molecular markers, and exposure to behaviors and environmental toxins that are not readily alterable. Despite several scientists’ brash promises of genetic interventions (Varmus, 2006) and the dedication of enormous financial resources to genetic inquiry, thus far there has been little evidence that genetic manipulation is a realistic near-future goal.

Third, and relatedly, both the above limitations have contributed to a rather poor record of epidemiology and public health in eliciting genuine behavior changes that “address” the burden of individual risk behaviors. The past few decades offer several examples of behavior change interventions that were demonstrably efficacious in small and well-controlled trials but not effective when applied in the general population. For example, although several epidemiologic studies show that sexual behavior contributes to risk of sexually transmitted diseases (Kaestle, Halpern, Miller, & Ford, 2005), and controlled trials have achieved changes in sexual practices (DiClemente & Wingood, 1995), sexual risk behavior remains notoriously difficult to influence at the population level (Lyles et al., 2006; Herbst et al., 2006; Herbst et al., 2005). Comparably, the

recent obesity epidemic has made it all too clear that simply demonstrating associations between greater weight and disease (demonstrated in countless epidemiologic studies during the past twenty years) is not sufficient for improving dietary habits, particularly when individual dietary habits are constrained by lack of healthy food options or safe places to exercise (Fitzgibbon & Stolley, 2004).

Particularly in the instance of enjoyable behaviors, appeals based on epidemiologic observations hold very little sway. This, of course, is not surprising given that epidemiologic studies frequently provide conflicting evidence and focus on factors which are indeed difficult to change. In addition, epidemiologic studies all too often suggest that changing single risk factors may be all important for disease prevention. However, the epidemiologic equating of being in the tail end of the risk distribution with “risk” means that persons with a particular “risk factor” may well not develop disease and others without may well indeed do so, which flies in the face of the notion of multifactorial disease causation that is intuitively and readily understood by the general public. Ultimately, these limitations “stack the deck” against epidemiologically-informed recommendations that put the onus of change only on individuals and promote goals that are, in a practical sense, unattainable. Nothing short of a colossal effort, or a dramatically terrifying disease, is required to change individual behavior. It is worth remembering that only after decades of public health effort in the Western world have population smoking rates decreased, and it took the definitive infectious disease of our time, HIV/AIDS, to change population sexual risk behaviors.

### 3. The Emergence of Social Epidemiology

A growing appreciation of the limitations of the individualization of epidemiologic thinking, coupled with a genuine abiding interest within public health in understanding the role that social factors play in determining health and disease, have contributed to a tremendous surge during the past fifteen years in research that takes a “social epidemiologic” approach (Kaplan, 2004). Social epidemiology emerged first from proponents of social medicine, who argued for greater consideration of social factors in disease determination (Galdston, 1947; Krieger, 2001) and subsequently went on to develop and implement studies on such social factors as gender (Perry, 1998), race/ethnicity (Baltrus, Lynch, Everson-Rose, Raghunathan, Kaplan, 2005) discrimination (Krieger, 2000; Williams, 1999), occupational conditions (Lallukka et al., 2006), socioeconomic status (Kanjilal et al., 2006) and education (Jacobsen & Thelle, 1988). Several books and papers considering social epidemiology as a discrete entity have traced its development (Berkman & Kawachi, 2000; Honjo, 2004; Krieger, 2001; Oakes & Kaufman, 2006), reviewed its methods (Berkman & Kawachi, 2000; Oakes & Kaufman, 2006) and examined the role of social factors as determinants of health (Marmot & Wilkinson, 2006). Formalizing the study of social factors within epidemiology has provided epidemiologists with an opportunity to reintroduce what likely should never have been absent from epidemiology’s domain.

This essay, and this book, clearly and explicitly are informed by a social epidemiologic perspective and a concern with social factors that influence health. However, we propose that social epidemiology as currently understood and implemented falls short of its promise. As social epidemiology has fought for legitimacy within epidemiology and public health, epidemiologists interested in social determination have published studies with increasing methodologic sophistication, including studies that mimic mainstream epidemiologic publications and methods. Therefore, studies have used ever more complex statistical techniques to examine how factors such as gender, race/ethnicity, income, and so forth may come to contribute to individual risk of disease. While this has achieved the goal of establishing social epidemiology's intellectual bona fides within the epidemiologic and public health research and practice community, social epidemiology has not done much better than other risk factor epidemiology in expanding beyond the individual-level risk of disease or in offering practicable insights. This is frequently discussed in the literature as a challenge inherent in the study of immutable social factors, such as race/ethnicity (Berkman, 2004; Bhopal, 1997).

It should be clear from our discussion here that we do not think that this challenge is unique to social epidemiology, but is rather a function of the larger problems that face epidemiology (i.e., the impracticality of evaluating individual risk factors using population based measures, the immutability of individual-level risk factors, and the attempt to isolate single causes of individual disease when the nature of causation is inherently much more complicated). However, we suggest that social epidemiology can do better and consider questions and adopt methods that overcome some of the key challenges facing epidemiologic inquiry today. Indeed, social epidemiology presents an opportunity to address both conceptual and practical limitations of an individual risk perspective and to suggest new and dynamic areas of inquiry. In particular, we argue that this can be achieved by the adoption of a population-level approach to examining the distal social factors and processes that influence health.

#### 4. A Population Health Strategy

Margaret Thatcher famously suggested that “there is no such thing as society. There are [just] individual men and women”. Our central premise is that the health of populations is as much derived from the connections between individuals and the social factors or processes to which a given population is exposed as it is a function of the aggregate persons within that population. We use the term “population health” to refer to the health of whole groups of persons, be they groups within neighborhoods, occupational class, or other levels of aggregation. Therefore, populations are not simply the sum of their individual parts, and subsequently, population health is not simply the sum of individual health. A corollary is that an individual, if she were part of another population, might have a rather different health profile, and a population (e.g., a neighborhood), if

comprised of alternate individuals and characterized by dissimilar local circumstances, might then have rather different population health.

If we accept the notion that population health is worthy of inquiry, we can then imagine solutions to the practical problems facing epidemiology. First, it follows that the epidemiologic methods that are better suited to population-level inference can be applied fruitfully to the study and improvement of population health (Rose, 1985). Second, group-level observations are not informed by the particular multifactorial causation of disease in a given individual and a population strategy avoids the flawed quest to identify single modifiable risk factors that provide (false) promises of improvement in individual risk of disease. Third, and centrally, a population strategy recognizes that population health is our ultimate goal and avoids futile attempts to change the behavior of individuals. Rather, a population strategy aims to improve population health generally, to shift the population disease curve by influencing the overall risk a population faces. From a very pragmatic point of view, this approach sidesteps the challenges discussed earlier that result in limited effectiveness of widespread attempts at individual behavior change. Therefore, a population approach might involve banning the use of escalators, increasing the likelihood that all able population members walk up an extra flight or two of stairs on a regular basis. This would be associated with lower risk of living a sedentary life for the whole population and therefore lower population rates of heart disease. Insofar as it is the aim of public health to improve the health of whole populations, the approach we propose here is congruent with this goal.

Importantly, we note that the improvement of population health is not at odds with the practical desire of improving the health of individuals. Rather, this conception suggests that individual health is so inextricably linked to the populations to which individuals belong that to think of ways only to improve individual health is ultimately a fallacy and a Sisyphean effort, a doomed and impractical attempt at improving health.

Clearly, different moral philosophical perspectives might find this perspective more, or less, appealing. A utilitarian might find the notion of populations as an undifferentiated grouping of individuals (each of whom, implicitly, are equally worthwhile) discomfiting, while this approach may be more congruent with a perspective that is primarily informed by considerations of social justice. Our argument is based strictly on an empiric conceptual and practical rationale; while we do think that there is ample philosophical reason to further buttress this argument, particularly with reference to health equity, a full discussion of the moral implications of a population health approach to epidemiologic thinking is beyond the scope of this brief introduction. We refer the reader to other published works for more on this issue (Bodenheimer, 2005; Brock, 1998; Edney, 2006; Kawachi, Kennedy, & Wilkinson, 1999; Menzel, 2003; Peter, 2001; Popay, 2006).



## 5. Macrosocial Determinants of Population Health

Thus, we suggest that social epidemiology can provide a conceptual lens and empiric methods for evaluating macrosocial determinants of population health. “Macrosocial” here refers to factors, such as culture, political systems, economics, and processes of migration or urbanization (all featured as chapter topics in this book), that are beyond the individual and are explicitly a function of population systems. Taking this perspective, social epidemiology would seek to understand the interconnections between and among the individuals that make up these systems and how these macrosocial factors shape the health of populations. Applying new epidemiologic methods and discipline to the study of macrosocial factors would serve to bring epidemiology back to the core concern that has long motivated public health, that is, discovering how we can improve social structures and circumstances to improve the health of populations.

Identifying macrosocial processes that influence population health can provide opportunities for interventions that influence the population distribution of risk and improve the health of whole populations, avoiding the “high risk” intervention trap into which much of our current individual risk thinking leads us. Improvements in motor vehicle safety, workplace safety, and family planning, as well as introduction of safer and healthier foods, were all recently suggested as among the greatest public health achievements of the twentieth century (Centers for Disease Control and Prevention, 1999), and all result from macrosocial interventions aimed at reducing population-level risk. An explicit focus on the macrosocial factors that underlie population health in the near future may permit us to identify, and effectively intervene on, the key determinants of population health of the twenty first century.

## 6. Conclusion and a Way Forward

A refocus of social epidemiologic methods and approaches to thinking about macrosocial determination of population health will not be easy. There are three likely key limitations to achieving such an end. First, with few exceptions, thinking about macrosocial factors as determinants of population health today is far from the core concern of most health researchers, including epidemiologists. Therefore, such a paradigm shift will require a substantial intellectual investment on our parts and will undoubtedly stretch our imaginations and practical capacities. Second, social epidemiologic methods are still nascent, and there is no question that a systematic consideration of macrosocial determinants of population health will require the refinement of our current methods, the development of new methods, and the judicious and careful interpretation of results from our studies. Researchers who are interested in the macrosocial determination of population health will have to make unimpeachable efforts to draw objective inferences using methods that are as robust as possible. Third, there is little doubt that change in public health, as in all human endeavors, comes slowly. We recognize

that the adoption of research questions such as the role of globalization in influencing population rates of heart disease is a substantial departure from the overwhelming majority of extant modern public health literature that influences and shapes the work we all do. In addition, given the importance of research funding in driving academic and public health inquiry, a conceptual shift predicated on thinking about the macrosocial determinants of population health would need to make substantial inroads into traditionally biomedical-oriented funding institutions to allow for the sustainable grounding of this work.

We have little doubt that with time researchers and public health practitioners will find suitable ingenuity and imagination to develop the field. In meeting all of these challenges, public health stands to benefit greatly from cross-disciplinary communication and collaboration. Insight from multiple disciplines, including economics, sociology, health policy, among many others, play a critical role in advancing understanding of population health and how to improve it. In the following sections, various authors will consider a range of macrosocial determinants that may influence population health, as well as key methodologic challenges this work faces today and in the future. Additionally, they will offer some insights into what the implications of considering macrosocial determinants might be for public health intervention. It is the intent of this book to provide a first step toward the systematic consideration of macrosocial determinants of population health. We hope that this work inspires theoretic and empiric innovation and investigation in this area.

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# Section 1

## Determinants

# Chapter 2

## Macrosocial Determinants of Population Health in the Context of Globalization

Lia S. Florey, Sandro Galea, and Mark L. Wilson

### 1. Introduction

We live in an increasingly interconnected world, as some like to say, a “global village.” As in any village, social, economic and biophysical environments shape individual action and interaction, which, in turn, influence the quality of life and the health of inhabitants. Technology, information, media, food, goods and services, as well as environmental pollution and diseases are shared among villages, cities, countries and continents. Not only are these exchanges great in scope, but the magnitude and speed of interaction among individuals and populations is also increasing. For example, international trade grew 8.6% per year during the decade 1990–1999 (World Trade Organization, 2000a, b), with an estimated US\$1.7 trillion in daily global trading (Lee, 2000). An estimated 760 million people traveled to international destinations in 2004 (World Trade Organization, 2005), and circumnavigation of the globe is now possible in a mere 36 hours (Smolinski, Hamburg, & Lederberg, 2003). Immigration contributes to global exchanges, with an estimated 175 million individuals spending at least one year in another country (United Nations, 2002). Additionally, approximately 17 million refugees and internally displaced persons migrate from their homes every year (United Nations High Commissioner for Refugees, 2004). These trends of growing interactions on the global scale shape the environments in which we live and which influence our well-being and our health.

The term globalization is used to denote these global trends in exchanges and interactions. Historically, globalization has been defined in economic terms as “the removal of tariff and non-tariff barriers to trade” (Weisbrot, Baker, Kraev, & Chen, 2002) or “the process whereby national and international policy-makers promote domestic deregulation and external liberalization” (Cornia, 2001). We argue, as have many before us, that globalization is comprised of much more than fiscal trends and policies. For the purposes of this review we use an expansive definition for which globalization consists of the “processes contributing to intensified human interaction in a wide range of spheres (that is, economic, political, social, environmental) and across three types of boundaries—spatial, temporal and cognitive—that have hitherto separated individuals and societies”

(Bettcher & Lee, 2002). Implicit in this definition is the ubiquity of globalization processes and the pervasiveness with which these processes affect human lives.

Although the existence of global influences on individuals and populations is clear, the effect of globalization on individual well-being and population health is not well established. Empirical evidence suggests both positive and negative effects of globalization on health, but there is no simple equation that can encapsulate how globalization may improve, or harm, population health. Instead it is likely that myriad processes comprise globalization, and each may influence the health of populations through multiple pathways. The challenge lies in elucidating the mechanisms by which globalization affects health. An understanding of these mechanisms will inform the decision-making process and enable implementation of policies that will mitigate the negative consequences of globalization and enhance its potential positive influences.

This chapter addresses an important gap in knowledge on the global context of population health by providing a conceptual framework from an epidemiologic perspective. The aim of this framework is to facilitate understanding of the complex relationships among globalization, macro-level determinants of health, and population health. The relationships between each component of the framework and population health will be briefly discussed followed by a presentation of potential mechanisms that may explain these associations. This chapter integrates current knowledge pertaining to the relationships of interest, generates hypotheses about mechanisms where current knowledge is scarce, and presents a brief discussion of methodological issues pertaining to epidemiologic studies of globalization and population health. We acknowledge that we approach this chapter as epidemiologists, building explicitly on epidemiologic multilevel thinking. We suspect that other disciplinary perspectives may approach the issue of globalization differently. We hope that our approach is illuminating, regardless of the reader's disciplinary orientation, and may engender discussion and debate that can bring about cross-disciplinary synthesis.

### *1.1. Framework*

Globalization is characterized by a plethora of components that may influence health at the population level. We propose a framework that summarizes a few key characteristics of the global, national and community level environments that are associated with population health. This framework builds on previously published conceptual models (Huynen, Martens, & Hilderink, 2005; Labonte et al., 2002; Spiegel, Labonte, & Ostry, 2004; Woodward, Drager, Beaglehole, & Lipson, 2001) and on the broader literature examining the effects of contextual determinants on health (Galea, Freudenberg, & Vlahov, 2005; Galea, Rudenstine, & Vlahov, 2005; Kaplan, 1999; Link & Phelan, 1995).

Our framework (Figure 2.1) builds upon the emerging thinking about multiple "levels" of determination of population health (Kaplan, 1999) and suggests that three levels of variables may be considered central to the role played by globalization in population health. Global-level factors (including global trade, income distribution, population movement, global governance and communications) are

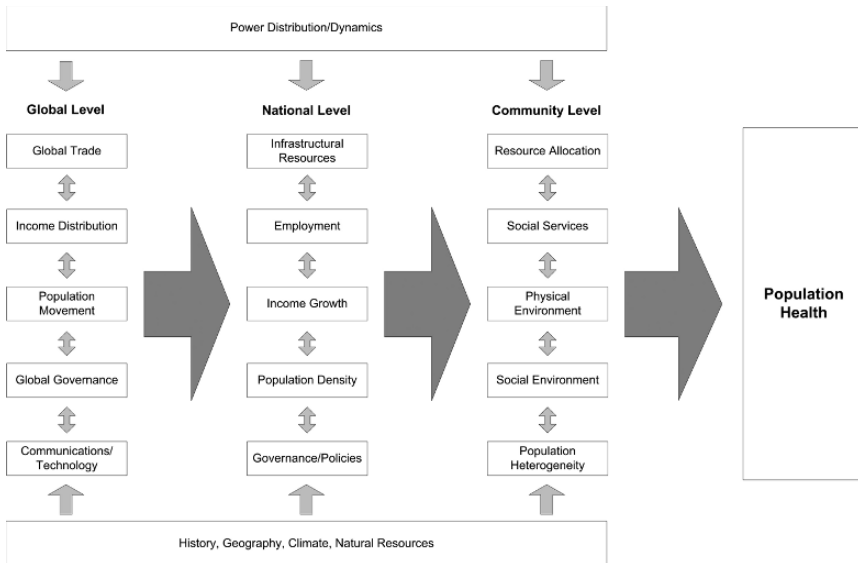


FIGURE 2.1. Conceptual framework summarizing how characteristics of the global, national and community-level environments may influence population health

conceptualized to shape national-level factors (infrastructural resources, employment, income growth, population density, and national governance). The national-level factors influence community-level factors (resource allocations, social services, physical environmental, social environment, and population heterogeneity). In turn, each of these elements are affected by global distributions and dynamics of power, as well as by underlying conditions such as history, climate, and geography that are represented by the horizontal rows at the top and the bottom of the model. Although this framework is designed to be hierarchical, with global-level factors influencing population health through the national and community-level factors, we recognize that there will be some direct effects between elements at any level of organization and health. Similarly, we consider that these associations could be bidirectional (a national-level factor may directly influence a global-level factor) as well as vertical (interrelationships among several components at the global level). The aim of this conceptual model, however, is to present the integral role of global-level processes in influencing population health as part of a multivariate, multilevel framework. This simplification of what is undoubtedly a far more complex web of associations is intended to clarify the current state of knowledge and help guide future research.

Although an unconventional approach, we begin by exploring the proximal relationships between community level factors and population health before approaching the more distal national-level and global-level factors (moving from right to left in Figure 2.1). This strategy allows clearer and more explicit development of the pathways through which globalization exerts influence on health and is compatible with clinical and epidemiologic approaches to health research.



## 2. Community-Level Processes

Despite extensive public health research focusing on the “community,” a definition of this concept remains elusive. Considerable recent investigations have addressed “neighborhoods,” while others have grouped people into census tracts or other administrative units. Such groupings may be convenient, affordable or otherwise useful for spatial analyses; however, they do not always carry social meaning for individuals. For purposes of this review, communities are defined as any sub-national aggregation that is socially meaningful to local residents.

As many scholars have noted, diverse aspects of community life that we characterized in Figure 2.1 play an enormous role in forming the health profiles of populations. These are the most proximal of the macro-level health determinants that are addressed in this chapter and should therefore affect population health indicators through the most direct pathways. Yet we recognize that multiple elements of communities may interact to shape health indicators through more complex, indirect pathways. The brief descriptions and examples presented below are not meant to be exhaustive, but rather to illustrate a few specific mechanisms whereby population health is affected by the environmental, social and political realities of communities.

### *2.1. Resource Allocation*

Community allocation of resources affects population health through direct and indirect pathways. Directly, certain basic resources are necessary for the maintenance of body functions. Access to a sufficient quantity and quality of food is essential for proper nutrition. Malnutrition and undernutrition have severe consequences for growth and development of children (de Onis, Monteiro, Akre, & Glugston, 1993; Stevenson, Latham, & Ottesen, 2000; Weinreb et al., 2002), as well as for the functioning of the immune system and prevention of disease (Cunningham-Rundles, McNeeley, & Moon, 2005). Adequate nutrition requires either enough arable land for local food production or access to markets with imported food and income with which to purchase the food. Not only are the absolute quantities and costs of these resources in a community important, but the distribution of these resources also contributes to shaping health outcomes. For example, the availability and cost of fresh fruits and vegetables and the spatial patterns of supermarkets are strongly linked to the income and racial characteristics of neighborhoods in Detroit (Zenk et al., 2005; Zenk et al., 2006)

Similarly, the quantity and quality of a community’s water supply is an important determinant of people’s health (United Nations, 2005). Insufficient water sources may lead to insufficient food supplies due to lack of irrigation of crops. The economies of developing countries are highly dependent on agriculture, which generates 80% of export earnings; however, this important source of income requires almost 70% of the world’s freshwater use (United Nations, 2005). In communities where potable water sources are far from residences, considerable time and energy is expended supplying households with water (Cosgrove & Rijsberman, 1998).

It has been estimated that 40 billion working hours are lost in Africa each year due to time spent transporting water, with the burden falling most heavily on women and children (United Nations, 2005). Poor quality water in conjunction with poor sanitation and hygiene is responsible for 1.6 million deaths per year due to diseases such as cholera, typhoid fever, trachoma, and schistosomiasis, to name a few (World Health Organization, 2004). Water can also be a source of toxicity. The well water consumed by 28–35 million Bangladeshi (United Nations, 2005) contains high levels of arsenic, causing skin lesions and various cancers (Smith, Lingas, & Rahman, 2000).

Shelter from the elements is another essential resource influenced by community-level factors. Good quality construction (Konradsen et al., 2003; Yé et al., 2006), and screened windows and doors (Lindsay, Emerson, & Charlwood, 2002) prevent spread of vector-borne diseases such as malaria. Quality housing protects inhabitants from climate extremes (heat, cold, wet), which contribute to disease and mortality (Evans, Hyndman, Stewart-Brown, Smith, & Petersen, 2000; Gemmell, 2001; The Eurowinter Group, 1997). Insufficient physical space in a community may lead to overcrowded housing, which facilitates the spread of communicable diseases such as tuberculosis (Antunes & Waldman, 2001) and helminth infections (Carneiro, Cifuentes, Tellez-Rojo, & Romieu, 2002). Indoor air quality is another important characteristic of housing that can influence the risk of respiratory diseases (Bruce, Perez-Padilla, & Albalak, 2000).

In a similar manner, medications also are essential community resources. Insufficient or unreliable supplies of medications have serious health repercussions by contributing directly to morbidity and mortality as well as to drug resistance (Draper, Brubaker, Geser, Kilimali, & Wernsdorfer, 1985).

Aside from essential resources, communities also have differential access to goods that may be deleterious to health, such as tobacco, alcohol and narcotics. For example, neighborhoods in Baltimore, Maryland, comprised predominantly of African Americans have a higher density of alcohol distributing outlets than neighborhoods with different racial demographics (LaVeist & Wallace, 2000). Many other such directly detrimental social impacts exist at the community level.

Indirectly, the distribution, volatility, and cost of these resources in the community are likely to have effects on social interactions and behaviors which may shape population health (Gopalan, 2001). Food insecurity causes psychosocial stresses that are harmful to mental health and can increase susceptibility to other acute and chronic diseases (Weinreb et al., 2002). Residential crowding, resulting from limited access to housing or from prohibitory housing costs, also contributes to psychosocial stress in a community (Krieger & Higgins, 2002). Unequal access to basic resources such as food, water, housing and medical supplies may have repercussions for the entire community by decreasing social cohesion (Wakefield & Poland, 2005). Social cohesion and collective efficacy help to defend a community against crime and vandalism (Kawachi, Kennedy, Lochner, & Prothrow-Stith, 1997) and provide social resources that buffer the negative health effects of being resource deprived (Sampson, Raudenbush, & Earls, 1997) (see Section 2.4).

## 2.2. *Social Services*

Communities with local access to health care, emergency and security services, good educational opportunities and social support systems such as welfare and social security are more likely to have good health (Cheadle et al., 1991). Access to these social services increases opportunities to obtain necessary resources (discussed above) and provides a buffer against volatile economic situations. Important issues include presence of these services, physical proximity to communities, cost and distribution.

Mechanisms by which social services affect community health include the provision of basic human needs (e.g. food, shelter, medications) or the means by which people meet those needs (e.g. employment or supplementary income). For example, communities that lack access to good quality health services may have higher burdens of disease because sick individuals will delay seeking care or will turn to alternative options, such as traditional healers or self-treatment (Chen et al., 2004; Meerman et al., 2005). Delayed treatment can have serious health consequences. For example, Gambian children presenting with severe malaria were significantly more likely to have delayed seeking treatment by more than four days than were those presenting with mild malaria (Meerman et al., 2005). Furthermore, self-medication may encourage drug resistance, as has been observed with malaria (Evans et al., 2005). Because poor or nonexistent welfare services can lead to increased poverty, malnutrition, homelessness, and starvation (Marmot, 2002) as well as reduced access to good education, such services have long been considered a “fundamental” determinant of health (Adler & Newman, 2002; Adler & Ostrove, 1999; Link & Phelan, 1995). Education also may affect health by increasing knowledge of healthy behaviors and by increasing employment opportunities that provide income to meet basic health needs (Ross & Wu, 1995). Individuals with better education live longer and suffer less morbidity than do their more poorly educated counterparts (Bobak, Hertzman, Skodova, & Marmot, 1999; Hemingway, Shipley, Macfarlane, & Marmot, 2000; Lynch, Kaplan, Salonen, Cohen, & Salonen, 1995). Research by Winkleby and others (1992) revealed that even after controlling for the effects of income and occupation, education reduced risk of cardiovascular disease. Emergency services provide urgent care, which can lessen population morbidity and mortality. Security services such as fire fighting and policing help to deter crime and violence, which have serious implications for health. For example, the 1975 fiscal crisis in New York City led to a 20% reduction in the number of city police employees, which likely contributed to the homicide epidemic of the 1980s (Freudenberg, Fahs, Galea, & Greenberg, 2006).

## 2.3. *Physical Environment*

Environments have long been recognized to play an important role in population health, from the ancient Greek's association of malaria with swamps to the miasma theorists purporting that squalid living conditions caused illness. With the advent

of germ theory and the advancement of modern epidemiologic methods and statistical tools, our understanding of relationships between environmental conditions and health outcomes has deepened. We now recognize that forces at many levels shape environments and associated diseases. Global climate patterns influence temperature, precipitation, and extreme weather events; accumulating evidence suggests that human behaviors, such as the expanding use of fossil fuels, are causing rapid changes in climate (Vitousek, Mooney, Lubchenco, & Melillo, 1997). Similarly, air and water pollution are affecting the quality of local, national and global environments and are shaped by human behaviors at each of these spatial/political scales. At the community level, environments are defined in part by physical conditions, which are affected by local, state or federal policies, such as zoning laws (Schilling & Linton, 2005), as well as by human behaviors and actions such as vandalism (Ross & Wu, 1995). The range of health indicators influenced by the built environment is vast and includes mental health (Weich et al., 2002), sexually transmitted infections (Cohen et al., 2000; Cohen, Mason et al., 2003), crime and violence (Newman, 1986; Sampson et al., 1997), substance abuse (Galea, Rudenstine et al., 2005), cardiovascular disease (Diez Roux, 2003) and physical activity (Frumkin, 2002), to name a few. Local environments, including homes and workplaces, may be sources of exposures to toxic substances, allergens or poor air quality (Bruce et al., 2000). Aspects of the outdoor environment are recognized as contributors to increased injury (Moore, Teixeira, & Shiell, 2006) or breeding of disease spreading vectors (McMichael et al., 1999; Moore, Gould, & Keary, 2002).

Community-level environmental characteristics and their potential health effects range in complexity and diversity. Climatic factors recently have drawn the attention of epidemiologists as measurement and analytic tools have improved. Greater climate extremes and global climate changes create or destroy microhabitats for many organisms that affect people's daily lives. Some changes may be beneficial, but most are expected to challenge efforts to improve health, particularly in developing countries. For example, insects that serve as vectors for various infectious diseases may become more abundant or widespread with global warming. One example is the possible increased range of malaria into previously uninfected highland regions (Bouma, Dye, & Van der Kaay, 1996; Loevinsohn, 1994; Zhou, Minakawa, Githeko, & Yan, 2004). Climate change, including extreme events, has been associated with other infectious diseases as well, such as cholera (Pascual, Rodo, Ellner, Colwell, & Bouma, 2000), cryptosporidiosis (Atherholt, LeChevallier, Norton, & Rosen, 1998), and other water-borne diseases (Curriero, Patz, Rose, & Lele, 2001).

Similarly, changes in precipitation and temperature may affect local agricultural yields, thereby affecting food availability with all of the accompanying health implications (Fischer, Shah, Tubiello, & van Velhuizen, 2005). As another example, temperature extremes may have direct effects on mortality, especially for poor, elderly or otherwise disadvantaged individuals. A heatwave in Chicago in July of 1995 resulted in 460 excess deaths that disproportionately affected African Americans and bed-ridden individuals (Semenza et al., 1996). In southern Chile,

exposure to UV-B radiation due to the proximity to the Antarctic ozone hole has been linked to increased sunburn and photosensitivity (Abarca, Casiccia, & Zamorano, 2002), and increases in skin cancer rates have been predicted (Jones, 1987). Limiting outdoor activities might protect against these health risks, yet this may cause other morbidities, such as obesity and diabetes, due to decreased physical activity (Gracey, 2002; McMichael, 2000). Many health effects of the built environment are certainly mediated by forces of the social environment.

#### *2.4. Social Environment*

Environments are not only physical but are also social. Social environments shape our interactions, our beliefs and our behaviors, all of which have health effects. To complicate matters, social environments likely interact with physical environments in their relationships with population health.

Aspects of the social environment that are likely to influence human health include social disorganization, social resources (including support and capital), social contagion, spatial segregation and inequality. The theory of neighborhood social disorganization, arising from sociological research of urban Chicago in the 1940s, posits that social disorder is conducive to deviant behavior and crime (Shaw & McKay, 1942). This theory hypothesizes that social disorder originates from lack of social control, low density friendship networks, and lack of participation in local organizations (Sampson & Groves, 1989). More recent research has shown that communities with high social disorganization are more likely to suffer from violence, victimization and homicide (Sampson et al., 1997), as well as coronary heart disease (Sundquist et al., 2006). Social disorganization may arise from inequalities in levels of deprivation and lead to anomie, defined as strain caused by disparate levels of attainment within a community (Kawachi, Kennedy, & Wilkinson, 1999). Social strain not only encourages deviant behavior and crime (Agnew, 1992), but also has been shown to be associated with increased homicide and cardiovascular mortality (Cohen, Farley, & Mason, 2003). Social strain may also cause physiological stress responses, which have well established links with mental and physical health (Elliott, 2000; Latkin & Curry, 2003; Ross & Mirowsky, 2001).

Social resources, including social support and social capital, are recognized to provide better coping mechanisms for difficult situations and are therefore associated with better health (Kawachi & Berkman, 2001; McLeod & Kessler, 1990). Social capital is also likely to help buffer negative health effects of social disorder by providing economic and social support (Sampson et al., 1997). Negative associations have been found between social capital and mortality (Kawachi et al., 1997; Skrabski, Kopp, & Kawachi, 2004) and violent crime (Kennedy, Kawachi, Prothrow-Stith, Lochner, & Gupta, 1998) and positive associations between social capital and self-reported health (Subramanian, Kim, & Kawachi, 2002).

Social contagion, or social influence, is thought to affect health by the sharing of behaviors and attitudes among members of social networks, which can have both positive and negative health effects. These social norms are important in the

transmission of infectious diseases, such as sexually-transmitted infections (STIs) and HIV (Pick & Obermeyer, 1996; Wellington, Ndowa, & Mbengeranwa, 1997), as well as in the spread of behaviors such as suicide (Phillips & Carstensen, 1986) and criminality (Jones & Jones, 1995).

### *2.5. Population Heterogeneity*

The spatial distribution of racial and ethnic groups or groups of different socio-economic status may contribute to the determination of population health. From an economic viewpoint, segregation leads to homogeneity of resources, where those with low socio-economic position cannot access the resources that benefit more affluent individuals. Segregation by socio-demographic characteristics is known to accompany differential exposure to poor quality environments, including toxins, crime, violence, poverty and infectious diseases (Cohen, Mason et al., 2003). Poor, segregated populations have restricted access to health care services, shortages of health care providers and many under- or un-insured individuals (Mayberry, Mili, & Ofili, 2000). Finally, segregation and income inequality can cause both perceived and actual inequity, which erodes social trust and diminishes social capital with the resulting health effects as discussed above. This process may be enhanced by spatial proximity of the rich and the poor (Kaplan, Pamuk, Lynch, Cohen, & Balfour, 1996; Kawachi et al., 1997; Mayberry et al., 2000).

In contrast, spatial heterogeneity of socio-economic groups encourages diversity and allows an opportunity for resource sharing. Wealthier individuals may be encouraged to use their money and power to improve the access and distribution of resources needed for good health. This heterogeneity may also provide access to broader social networks, including positive role models and salubrious social norms. For example, unequal distribution of education in communities in New York City has been shown to have salutary effects for all residents, suggesting benefits of actions of highly educated individuals (Galea & Ahern, 2005). However, heterogeneous social environments may encourage social strain by providing images of unachievable aspirations to those with poor access to resources and few opportunities for advancement (Kawachi et al., 1999; Sampson & Groves, 1989).

## 3. National-Level Processes

A substantial amount of epidemiologic research has examined the role of globalization in shaping population health at the national level. Cross-national comparisons of health indicators have investigated the effects of national income (Dollar, 2001; Lynch, Smith, Kaplan, & House, 2000; Weisbrot et al., 2002), mode of governance (Navarro & Shi, 2001), and average educational attainment (Williamson & Boehmer, 1997), among others. We add to this body of literature by explicitly hypothesizing mechanisms through which aspects of nations may influence health and the extent to which this is mediated by the community-level determinants of health discussed above.