Modern Environments and Human Health: Revisiting the Second Epidemiologic Transition demonstrates by example how methods, theoretical approaches, and data from a wide range of disciplines can be used to resolve longstanding questions about the second epidemiologic transition - the shift from a high burden of epidemic, infectious illness to greater morbidity and mortality from chronic and degenerative diseases.

Understanding how, why, and when the transition occurred across different regions and communities can provide critical insight into the relationships between economic growth, environmental quality and change, and human health. This comprehensive book brings together skeletal, archaeological, biodemographic, geochemical, environmental, epidemiological, cemetery, social history, and parasitological data to shed light on this important epidemiologic transition, and to inform current debates on the best ways to allocate public health funding in developed and developing countries.

The first book to address the second epidemiologic transition from a multi-regional, comparative, and interdisciplinary perspective, Modern Environments and Human Health will be a valuable resource for students and academics in biological anthropology, economics, history, public health, demography, and epidemiology. Key features include:

- Discusses the second epidemiologic transition from a multi-regional, comparative, and interdisciplinary perspective
- Brings together skeletal, archaeological, biodemographic, geochemical, environmental, epidemiological, cemetery, social history, and parasitological data
- Written in an engaging and jargon-free style by a team of international and interdisciplinary experts
- Provides significant detail on data, methods and findings for potential replication

Molly K. Zuckerman is Assistant Professor in the Department of Anthropology and Middle Eastern Cultures at Mississippi State University. The author of numerous peer-reviewed publications, Dr. Zuckerman also teaches introductory and advanced courses in anthropology and biological anthropology, osteology, and human behavior and disease.
Modern Environments and Human Health
To my mother, Joan Byrne, without whose influence
I would never have become an anthropologist, and to my endlessly
supportive husband and partner, Derek Anderson.
Modern Environments and Human Health

*Revisiting the Second Epidemiologic Transition*

*Edited by*

**Molly K. Zuckerman**

*Department of Anthropology and Middle Eastern Cultures*

*Mississippi State University, Starkville, MS*
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Contributors

Gilda M. Anroman
School of Pharmacy,
Notre Dame of Maryland University,
Baltimore, MD

George J. Armelagos
Department of Anthropology,
Emory University,
Atlanta, GA

Julia Beaumont
Division of Archaeological,
Geographical and
Environmental Sciences,
University of Bradford,
Bradford, UK

Jeffrey K. Beemer
Department of Sociology,
University of Massachusetts Amherst,
Amherst, MA

Alicja Budnik
Instytut Antropologii,
Uniwersytet Im. Adama Mickiewicza,
Poznań, Poland

Simon Chenery
British Geological Survey,
Keyworth, UK

Carolina de la Cova
Department of Anthropology,
University of South Carolina,
Columbia, SC

Sharon N. DeWitte
Department of Anthropology,
University of South Carolina,
Columbia, SC

Jessica Dimka
Department of Anthropology,
University of Missouri,
Columbia, MO

Jane Evans
NERC Isotope Geoscience Laboratory,
Keyworth, UK

Nancy L. Fleischer
Department of Epidemiology and
Biostatistics, Arnold School of Public
Health, University of South Carolina,
Columbia, SC

Timothy B. Gage
Department of Anthropology
and the Department of Epidemiology,
University of Albany,
State University of New York,
Albany, NY

Alain Gagnon
Département de Démographie,
Université de Montréal, Montreal,
Quebec, Canada

Aravinda Meera Guntupalli
Centre for Research on Ageing,
University of Southampton,
Southampton, UK
Contributors

Stacey Hallman
Department of Sociology,
Western University, London,
Ontario, Canada

Peter M. Kitson
Cambridge Group for the History
of Population and Social Structure
and the Faculty of History,
University of Cambridge,
Cambridge, UK

Nikola Koepke
Departament d’Història i
Institucions Econòmiques,
Universitat de Barcelona,
Barcelona, Spain

Robert E. McKeown
Department of Epidemiology
and Biostatistics, Arnold
School of Public Health,
University of South Carolina,
Columbia, SC

Andrew Millard
Department of Archaeology,
Durham University,
Durham, UK

Erin Miller
Department of Anthropology,
University of Missouri,
Columbia, MO

Janet Montgomery
Department of Archaeology,
Durham University,
Durham, UK

Carolyn Orbann
Department of Anthropology,
University of Missouri,
Columbia, MO

Megan A. Perry
Department of Anthropology,
East Carolina University,
Greenville, NC

Elisa Pucu de Araújo
Manter Laboratory of Parasitology,
University of Nebraska at Lincoln,
Lincoln, NE

Karl J. Reinhard
School of Natural Resources,
University of Nebraska at Lincoln,
Lincoln, NE

Lisa Sattenspiel
Department of Anthropology,
University of Missouri,
Columbia, MO

Lawrence M. Schell
Department of Epidemiology and
Biostatistics and Department of
Anthropology, University of Albany,
State University of New York,
Albany, NY

Rebecca S. Lander
Department of Anthropology,
University of Missouri,
Columbia, MO

Richard H. Steckel
Department of Economics,
The Ohio State University,
Columbus, OH

Mark Trickett
Department of Archaeology,
Thomas Jefferson’s Monticello,
Charlottesville, VA

Molly K. Zuckerman
Department of Anthropology
and Middle Eastern Cultures,
Mississippi State University,
Starkville, MS
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Chapter 1

Introduction: Interdisciplinary Approaches to the Second Epidemiologic Transition

Molly K. Zuckerman

Department of Anthropology and Middle Eastern Cultures, Mississippi State University, Starkville, MS

Introduction

The concept of an epidemiologic transition has served as a guiding principle for much of the discussion about economic growth and human health over the past several decades (Barrett et al., 1998). It constitutes a trend wherein a high burden of mortality from infectious disease, largely acute, epidemic childhood diseases, like smallpox or measles, is replaced by one of chronic and non-communicable diseases (NCDs), such as cardiovascular disease, cancer, and diabetes. The classic model was originally formulated to capture the changes in cause-specific mortality that followed the industrial revolution in the United States and Western Europe, but, in a modified form, the transition is ongoing in many developing low- and middle-income countries (LMICs). Recent scholarship has also placed the classic transition within an expanded evolutionary framework, recognizing a first transition coincident with the Neolithic and the intensification of agriculture, and a third of emerging and reemerging infectious diseases in the modern era (Barrett et al., 1998). This positions the classic transition as the second epidemiologic transition, as it is known in this volume.

Epidemiologic transition theory provides a model for understanding the relationships between economic, demographic, ecological, and social factors and the evolution and spread of disease (Armelagos and Barnes, 1999). As such, it has become a paradigmatic theoretical framework in public health (Caldwell, 2001; Sreter, 2002; Girard, 2005; Huicho et al., 2009), demography (McKeown and Record, 1962; Kirk, 1996; Salomon and Murray, 2002), biological and medical anthropology (Armelagos et al., 2005; Munoz-Tuduri et al., 2006), economics (Morand, 2004), and, to a much lesser extent, epidemiology (Harper and Armelagos, 2010; Fleischer and McKeown, this volume). The transition and the economic and social processes that drove it constitute one of the greatest social and environmental transformations in history (Armelagos et al., 2005). Industrialization and urbanization ushered in the chronic conditions and NCDs that not only plague high-income,
developed countries but also increasingly afflict LMICs, which still also carry a high burden of mortality and morbidity from infectious conditions (Marinho et al., 2013).

Because of this, understanding the causes of the transition, particularly the decline in infectious disease, its scope, and how it played out differently across different regions, communities, and time periods is critical to many concerns. These issues are fundamental to planning health services and to public health and economic policy in both high-income nations and LMICs (Caldwell, 2001a). When the health consequences of industrialization specifically are added to the model, they are also significant for modern governmental policy on central control versus free markets (Lindert, 1994). They are important for continuing debates about the allocation of funds for public health and the role of individual responsibility, broad-based efforts to redistribute social, political, and economic resources, or targeted, well-funded interventions in raising living standards and promoting good health, especially in an era of limited funds. Additionally, they are germane to the discussions about the relationship between nutrition, food supply, economic development, and population growth that have engaged the natural and social sciences since Malthus (Colgrove, 2002). Most broadly, they are critical to understanding how humans interact with their environments and the impact of environmental change on human health and adaptability. In the face of climate change, ecological instability, and a world in which more than 50% of humans live in urban environments (United Nations, 2012), it is increasingly important to understand the dynamics between human health and modern environments.

Despite its significance, controversy surrounds the second transition. As contributions in this volume discuss, fundamental questions persist about its causes, especially declining infectious disease mortality (Gage, 2005). Scholars have critiqued the model’s focus on mortality and neglect of morbidity and more holistic components of health (Johansson, 1992; Riley, 1992; Riley and Alter, 1989). They have also highlighted the nearly exclusive use of national-level data from the United States and Western Europe in studies of the transition, which obscures potential variation across regions, populations, and over time as well as by factors such as age, sex, race, or ethnicity (Barrett et al., 1998; McKeown, 2009; Fleishcher and McKeown, this volume).

Most of the controversy arises from the fundamental limitations of the material traditionally used to study total and cause-specific mortality (Gage, 2005). Total mortality is reconstructed from census and vital statistics records. However, vital registration systems only started in the 18th century in Northern Europe and after 1800 in the US and Western Europe; data from other countries, especially LMICs, exists only for much later periods or not at all. Cause-specific mortality is even more poorly documented. Reporting only started in England and Wales in 1830, and little data from other countries predates this. Documentary data on morbidity is even more limited and largely unavailable for any country before 1960 (Vallin, 1991; Gage, 2005).

The contributions to this volume—and the scholarship it is intended to spur—aim to end this stalemate. Both in sum and independently, these studies propose and demonstrate a novel approach to the transition that brings in theoretical approaches, methods, evidence types, and data sets from multiple disciplines. Some are more traditional for this topic, such as demography and economic history. Others, such as archaeoparasitology, bioarchaeology, and geochemistry, are less so. Studies of historical trends in health tend to systematically use and uncritically privilege documentary evidence, producing a one-sided view of history that is mostly reflective of textual sources (Perry, 2007). However, a more balanced, dynamic interplay between sources can be achieved by equally weighting each data type, analyzing them separately, and allowing them to extend and inform each other. Consistencies, non-conformities, and ambiguities between them can be identified and used to generate new research questions (see Buikstra et al., 2000). Ultimately, such a strategy can generate more
information than can separate consideration of the sources (Swedlund and Herring, 2003). This volume applies this approach to studies of the transition, both explicitly and implicitly. Several studies in this volume are explicitly interdisciplinary (e.g., Anroman; de la Cova; Koepke; all this volume). Others studies represent a single disciplinary approach, but grouped, approach a single issue from multiple perspectives (e.g., Orbann et al.; Hallman and Gagnon; both this volume).

**The Volume**

This volume is not comprehensive; it does not represent the full scope of research on the transition or all of the disciplinary approaches that can be brought to bear on it. It is not the final say. Instead, it demonstrates how interdisciplinary approaches can elucidate several specific neglected or poorly understood areas of the transition, such as the experiences of women, children, and ethnic minorities, and regional and temporal variation in the mode and pace of the transition. It also demonstrates how they can be used to investigate unexplored aspects of the transition, such as chronic disease from industrial toxicants during the industrial revolution, which are wholly inaccessible through traditional materials but can yield powerful insights for modern populations.

Each author was encouraged to be explicit about the interdisciplinary aspects of their research, their theoretical approach and methodology, and the rationales for their use. They were also encouraged to discuss the benefits, limitations, and interpretive issues posed by the type of data they employed and, overall, the advantages and disadvantages of their approach. Accordingly, these studies should foster novel, productive approaches to the transition and similar epidemiologic and demographic processes, encourage researchers to explore the unique vantage points of other disciplines for their research, and unite across these divisions.

The volume is divided into sections that reflect not the disciplinary traditions or types of evidence employed by different authors, but the larger issues that each addresses. The studies thus come into play with each other on a series of central uniting themes that are key to research on the second epidemiologic transition. Part 1 addresses standing conflicts and controversies about its causes, focusing on the decline in infectious disease. In Part 2, the authors address the 1918 Spanish influenza pandemic, the last great burst of epidemic crisis mortality, and its role in causing and contributing to the transition. Part 3 discusses largely unanswered questions about how the transition played out across different communities and population subgroups, such as women and ethnic minorities, and the interpretive issues involved for historically underrepresented groups. Part 4 attends to the poorly understood issues of regional and temporal variation in the transition and the implications of this variation for models of the second transition and epidemiologic transition theory. Lastly, in Part 5, the authors address an understudied aspect of the transition: environmental quality and the role of parasites and industrial pollutants in changing patterns of infectious and chronic disease. These sections are not exclusive; nearly all of the studies in Parts 3–5, for instance, shed some light into causes of the transition and, by presenting data from understudied sources, time periods, or geographical regions, into regional and temporal variation. Importantly, many of the studies also defy categorization into a single section by their overarching focus on defining and investigating health as something more than longevity and mortality.

The volume concludes with four epilogues written by senior scholars charged with evaluating where research on the transition currently stands and where it should proceed from here. Armelagos places research in the volume within an evolutionary context and ties it into human adaptability and environmental change. Fleischer and McKeown discuss the place of epidemiologic transition theory in epidemiology and the creation of a space for
dialogue on health transitions between epidemiologists and scholars from other disciplines. Steckel sums the current state of affairs in terms of methodology and highlights important topics for future research and the evidence and data types required for those advances. Lastly, Gage provides an overview of current knowledge on the transition and recommends a framework for future research.

The Demographic and Epidemiologic Transitions

The epidemiologic transition models the causes of death that accompanied the demographic transition, the secular declines in mortality and fertility, and the consequent population growth that accompanied industrialization; several of the contributions to this volume address aspects of both transitions. The theory is a simplified, descriptive model encompassing multiple phases (Thompson, 1929). Stage I is largely preindustrial and is characterized by high mortality and fertility, variable but overall low life expectancy, and slow but stable population growth punctuated by bursts of crisis mortality. Stage II, beginning in the mid-19th century in Europe, featured declining normal mortality and continued high fertility. Stage III continues the mortality trend but features declining fertility, increased life expectancy, and more sustained population growth. Declining normal mortality began in the mid-19th century in Western and Northern Europe and the US and around 1920 in LMICs, like Chile. Crisis mortality continued into the 20th century, perhaps ending only with the 1918–1919 Spanish influenza. Mortality declines completed in the mid-20th century in high-income nations but continue in LMICs. Stage IV features low, still declining mortality, as observed in developed countries after WWII (Vallin, 1991). Mortality declines continue, especially among the elderly, but slowly, likely due to lower mortality among infants, children, and young adults (Gage 2005).

The epidemiologic transition incorporates consistent patterns in cause of death accompanying the demographic transition (Omran, 1977). The original formulation involves several stages. Stage I, the Age of Pestilence and Famine, is preindustrial and features high frequencies of epidemic infectious disease and crisis mortality. Stages II and III, the Age of Receding Pandemics, witness a shift from epidemic to endemic infectious disease and declining crisis mortality. In Stage IV, the Age of Degenerative and Man-Made Diseases [sic], degenerative diseases like diabetes and stroke replace infectious disease, ushered in by age-related degenerative processes and anthropogenic factors, namely the environmental hazards and nutritional and behavioral patterns associated with industrialization and urban living. This neat dichotomy is blurred by the chronic nature of some infectious diseases, such as tuberculosis, as well as increasing recognition of the role of infectious disease and inflammatory processes in many chronic conditions (e.g., HPV and cervical cancer). Rather than precluding use of transition theory, this issue highlights the importance of historical relationships between humans and pathogens for understanding the current patterns of human health (see Zuckerman and Armelagos, this volume).

As contributors discuss, this model also presents a simplification of reality; epidemiologic evidence clearly shows that the timing and pace of the transition varied between nations. As Fleischer and McKeown (this volume) discuss, Omran accounted for variants on the classic model, and several scholars have added additional stages. These variants suggest that socioeconomic and local conditions, such as the degree of urbanization, seem to greatly influence the extent, nature, and timing of the transition (Dobson, 1997; Woods, 2000), both now and historically. For instance, due to structural inequalities, rapid urbanization, and inadequate public health infrastructure in many LMICs, such as India (Guntupalli, this volume), populations seem to be undergoing concurrent second and third transitions, with more affluent segments of the population suffering from chronic and NCDs and poorer
1 Introduction

segments still carrying a high burden of infectious disease. Importantly, the industrial transition also is not yet complete in high-income nations; scholars do not yet know whether postindustrial populations will return to Stage I-type mortality and fertility regimes or not (Gage and DeWitte, 2009).

Some scholars have also proposed the idea of a “health transition” (Riley, 1989), which further elaborates on the demographic and epidemiologic transition models by incorporating the secular trends in morbidity states that accompany the transitions. This model summarizes the finding that the prevalence—but not necessarily the incidence—of morbidity increases as the demographic transition progresses and longevity increases (Riley and Alter, 1989; Roos et al., 1993), with attendant impacts on quality of life (Johansson, 1992; Riley, 1992). Secular changes in morbidity are difficult to document due to complex relationships between the incidence and prevalence of morbid conditions, and because the accuracy of diagnoses of morbidity states has improved over time amongst medical practitioners and examiners (see Gage, 2005). Relationships between morbidity and mortality are also difficult to reconstruct because there is not a straightforward, direct relationship between them (Gage, 1989; Crimmins et al., 1994; Usher, 2000); as Gage and DeWitte (2009: 650) note, this is because mortality is an “absorbing state”: it removes individuals from a population. If individuals in poor health are more likely to die than those in better health, there will often be an inverse relationship between morbidity and mortality in a given population (Riley, 1992). Reconstructions of this dynamic also depend on the exact definition of “health” employed in a given study. These range from a focus on mortality, such as the definition of life expectancy or age-standardized death rates employed by Gage (2005), to a biomedical definition premised on the absence of disease, to the more holistic one currently favored in epidemiology and anthropology: a “complete physical, mental, and social well-being” (WHO, 1948). Studies of the health transition typically define health as the ability to perform “activities of daily life” and occasionally the capacity for more vigorous physical measures, but as Gage and DeWitte (2009) note, these capacities are culturally relative. Furthermore, documentary data on such aspects of “health,” as well as morbidity, is limited to the 20th century, meaning that traditional studies of health and the health transition are largely limited to the current era and western, highly documented populations. However, as several of the studies in this volume demonstrate, skeletal data, particularly frequencies of various pathological lesions and skeletal stress indicators, can be used to reconstruct morbidity in past populations, therefore providing a venue for evaluating health transitions. While interpretive biases must be taken into account (e.g., Wood et al., 1992), skeletal data can give direct insights into patterns of morbidity surrounding the second transition. For instance, Perry (this volume) supplements her analysis of patterns of infant mortality preceding the transition with skeletal data on multiple indicators of quality of life and health, such as frequencies of metabolic deficiencies and general infection. DeWitte (this volume) employ frequencies of various skeletal lesions to reconstruct levels of morbidity prior to the epidemiologic transition in London, and assess relationships between morbidity and mortality.

Part 1: Causes of the Second Epidemiologic Transition

Despite decades of research, the cause of the transition, specifically the decline in infectious disease mortality, remains contentious. The most influential proposal came from McKeown (1976), who proposed that a rising standard of living, namely, improved per capita nutritional consumption from improved economic conditions, bolstered resistance to infectious disease. In other words, rather than medical advances or public health interventions, the invisible hand of economic forces precipitated the transformation of human demographic
and epidemiologic patterns (Colgrove, 2002; see Kitson, this volume). The McKeown thesis has ongoing resonance due to its public health and economic policy implications (Bynum, 2008) but it has ultimately been discredited; scholars now understand that public measures played a major role in reducing infectious disease mortality (Szreter, 1988), with medical advances such as vaccination, the germ theory of disease, general education, and improved hygiene exerting a delayed, supplemental effect (Cutler and Miller, 2005; see Reinhard and Pucu de Araújo, this volume). However, these insights have not resolved the controversy; a convincing and more comprehensive explanation of the decline remains unavailable (Gage, 2005).

This volume does not attempt such an explanation, but studies within demonstrate that an interdisciplinary approach can reduce ambiguity and isolate causal factors in the decline. They also reveal that causes of the decline were in many cases condition-specific rather than universal, with clear implications for public health and economic policy. For instance, due to the temporal limitations of documentary evidence, why and when the transition began in a particular context is often unclear. As DeWitte (this volume) emphasizes, this is a substantial issue. The reasons underlying the transition—why some portions of the population begin to succumb to some causes but not others, while others are able to survive to older ages—are ultimately of the greatest interest to researchers. With an eye on specifics rather than universalities, this can be accomplished by looking beyond traditional historical methodologies and available documentary materials. For instance, Anroman (this volume) brings together methods, paradigms, and evidence types from multiple disciplines to knit together an understanding of how individual’s relationships and interactions with (rather than within) their total environment— their physical, biological, psychological, cultural, political, and socioeconomic universe—precipitated patterns of declining crisis mortality in 17th- to early 19th-century Philadelphia. Other contributors employ skeletal evidence, particularly taking advantage of the insights that large, aggregated databases of skeletal data can grant into population-level processes. While skeletal material is subject to a raft of theoretical and material interpretive issues (e.g., Wood et al., 1992), it provides direct evidence of the biological experiences of past populations and exists for most regions and time periods (see Steckel, this volume). Perplexingly though, it has scarcely been applied to studies of the demographic or second epidemiologic transition. In this volume, DeWitte employs skeletal data from the Wellcome Osteological Research Database,¹ which includes thousands of skeletons from London from the Roman era into the mid-19th century, to model relationships between age, sex, experiences of physiological stress (e.g., malnutrition), and patterns of frailty and mortality from the 16th century into the mid-19th and assess how these patterns may have caused the transition in London. Importantly, by analyzing skeletal data from such a well-documented urban center, DeWitte also generates paleodemographic signatures that can be used as baseline expectations for studies of the transitions in less well-documented samples. Koepke applies econometric history methods to data on stature, a sensitive measure of environmental quality and net nutrition, from nearly 20,000 skeletons from across Europe, dating from the 8th century BC to the 18th century AD, to reconstruct the biological standard of living and therefore the conditions that preceded and precipitated the transition in Europe. This data set allows detection of key regional differences that highlight why the transition began first in Western Europe.

Lastly, interdisciplinary approaches can also highlight and engage with issues that are fundamental to understanding the causes of the decline yet are inaccessible to or neglected

¹http://www.museumoflondon.org.uk/Collections-Research/LAARC/Centre-for-Human-Bioarchaeology/Database/
by traditional methods and national-level data. Beemer (this volume) engages with an issue highlighted by Gage (2005) and others: that much of the controversy is attributable to the cause-of-death data and the ways in which diseases and causes of death are identified, grouped, disaggregated, and interpreted. Rather than exploring this issue on the national level, Beemer investigates on-the-ground difficulties experienced by medical and public health officials in implementing vital registration systems, changes in the structure of cause-of-death reporting, and evolving conceptions of disease, in two industrializing communities in Massachusetts. This community-level approach demonstrates that researchers must consider not only the quality of cause-of-death data but also the complex, contingent social, historical, and political interplay between lay communities, the state, and the medical community that created these sources.

**Part 2: Epidemic Infectious Disease, Chronic Disease, and the Epidemiologic Transition**

Interdisciplinary approaches can also shed light on declines in chronic and NCD mortality. Many aspects of modern, westernized environments are hazardous to human health: industrial pollution, sedentism, poor nutrition, obesity, and cigarette smoking, among others. However, since the risk of chronic and NCD mortality has declined over the 19th and 20th centuries (Preston, 1976; Gage, 1993, 1994, 2005), one or more yet unconfirmed aspects of modern environments must buffer humans from these risks. These include 20th-century lifestyle changes and medical advances, both too delayed to contribute much; the infectious origin of many degenerative conditions; direct interactions with infectious diseases; and indirect interactions with infectious disease mortality (Gage, 2005). Studies in this volume address the latter two.

In the 20th century, pandemics, particularly of respiratory diseases, such as the 1890 and 1918 influenza pandemics, were associated with excess chronic disease mortality (Lancaster, 1990; Azambuja and Duncan, 2002). The stress of these events may have contributed to chronic disease mortality, which then declined as infectious mortality declined. If so, the mechanisms are unknown (Gage, 2005). Indirect interactions may take several forms, including conditions wherein negative early life health experiences increase susceptibility to chronic and NCDs later in life (e.g., the Developmental Origins of Health and Disease Hypothesis (DOHaD)). Hallman and Gagnon (this volume) investigate the role of both direct and indirect effects on differential, age-dependent mortality in the 1918 pandemic using a biodemographic framework that draws from immunology, epidemiology, and human development. Their finding that 1918 mortality was a direct product of exposure to previous influenza pandemics not only potentially explains the dynamics of the 1918 pandemic, the last burst of crisis mortality, but also highlights the need to bring current scholarship on the DOHaD and fetal origins models into scholarship on the transition. Such models may be critical for understanding the segments of the population spared by epidemic infectious disease and therefore those who went on to experience continued declines in chronic and NCD mortality. Lastly, Orbann and colleagues (this volume) propose the use of agent-based modeling for studying epidemics in the context of the second transition and present a model designed for studying the dynamics of the 1918 pandemic in a small community. Agent-based modeling is widely employed in many different disciplines and is particularly useful for research questions that make use of incomplete and imperfect data but had yet to be applied to the second transition. The model enables analysis of how behavior patterns, particularly those related to social identity, affected disease spread. Future versions will be useful for addressing the interplay between overall health status (e.g., chronic disease presence) and modeling and potentially resolving many aspects of the transition: the effects of medical
interventions and community-level health improvements on population structure and the relative importance of different factors in precipitating the transition.

**Part 3: Regional and Temporal Variation in the Second Epidemiologic Transition**

Researchers have roundly critiqued studies of the second transition and epidemiologic transition theory for their bias towards national-level data, Northern and Western European countries, and of large cities within these nations. This has led to questions about how well these models capture and explain global health trends and patterns of mortality and morbidity within different demographic units. It also leaves regional and temporal variation in the early phases of the second transition as a major source of debate (McKeown, 2009).

Several of the studies in this volume are the first to explore the epidemiologic transition—or aspects of the transition—within their respective areas or time periods. For instance, very little is known about the transition in Eastern Europe largely because of material issues. Therefore, Budnik (this volume) casts a wide net over a variety of evidence types to test the effects of local ecologies on mortality regimes in Poland, a late industrializer, and whether different regions experienced a transition similar to other parts of Europe or followed a unique model. Importantly, Budnik also uses various indices to test how environmental change associated with urbanization and industrialization affected forces of natural selection in Polish populations, demonstrating an avenue for assessing the successfulness of different adaptive strategies humans employed during the second transition.

Studies here also demonstrate routes for circumventing the scarcity of subnational-level data and exploring regional- and community-level heterogeneity in the transition. Kitson (this volume), for instance, employs family reconstitution, which is infrequently applied to urban, industrializing communities, to address a relatively neglected question: how, why, and when the transition played out outside of large urban centers. Kitson highlights the fact that the models’ core assumptions about preindustrial society—that communities were small and wholly agrarian, or rooted in the manufacture and sale of agricultural products—are misleading in the face of evidence that preindustrial England instead featured numerous organizational paradigms, distributed differently across communities in time and space, with attendant heterogeneous mortality and fertility regimes. This accounts for one of the less commonly mentioned critiques of Omran’s model: despite being created, in large part, to model the mortality patterns of 18th- to 19th-century England, it fails to map onto and thus explain many of these patterns in England. Kitson’s unpacking of mortality patterns in relation to changing socioeconomic structures in a small English community draws attention to the fallacy of looking for a universal transition in epidemiologic regimes during the 18th and 19th centuries. Sattenspiel and Shattuck (this volume) also tackle problems involved in studying the demographic and epidemiologic transitions outside of large urban centers; smaller communities experienced major lags not only in sanitary improvements and therefore the transition but also in the initiation of vital registration systems. They propose a novel method for circumventing this evidentiary issue: headstone data, which are reliably available for many communities and time periods. Importantly, as this represents a highly accessible and valuable resource, the authors also discuss larger issues of whether and how local cemeteries can be used to reconstruct the transition in other regions and time periods.

These and other studies enrich our knowledge of variation in the transition and the relative importance of different factors in causing it in particular times and places. They also suggest a way forward for refining and expanding current models to more accurately reflect epidemiologic and demographic change both regionally and globally.
Part 4: Underrepresented Communities in the Second Epidemiologic Transition

Studies of the transition that rely on national-level data also mask variation in relation to sex, gender, race, class, and other aspects of social identity (Gaylin and Kates, 1997). However, even the original formulation posited that different experiences of mortality and longevity within populations, such as among children and reproductive-age women, would be key drivers of the transition and that different epidemiologic regimes would be found among whites and blacks (Omran, 1971; see Fleischer and McKeown, this volume). As women, children, and nonwhite ethnic groups are consistently underrepresented in the historical record, their biological experiences of the epidemiologic and demographic transitions remain poorly understood. Several studies in this volume demonstrate ways to circumvent these limitations, largely by embedding direct, biological evidence of health into textual evidence.

Several studies use skeletal evidence to do so. Importantly, skeletal remains can provide empirical evidence for the biological experiences of women and children that is largely free of the sex and age biases that affect documentary data (Grauer, 2003; Lewis, 2007). There is also a growing body of scholarship on reconstructing gender- and age-related identity in relation to health and disease (Hollimon, 2011; Lewis, 2007). However, few studies have used skeletal evidence to examine age-, sex-, and gender-based variation in experiences of the second transition. Here, DeWitte (this volume) explores sex-based patterns in mortality rates and frailty in preindustrial London. Koepke (this volume) uses stature data to test for evidence of gender discrimination in the net nutrition and biological standard of living in Europe. Guntupalli (this volume) also employs stature data, but from modern populations, specifically that of women experiencing the second transition in postcolonial India. Little is known about how the biological standard of living changed in India during the rapid population growth, shifting epidemiologic and demographic regimes, and slow economic growth of the postcolonial period. Guntupalli uses women’s stature data to examine this process, but does not assume that gender is the most important variable in women’s biological experiences. Instead, she takes an intersectional, multivariate approach, examining gender in relation to class, caste, religion, and other aspects of identity, and, by doing so, derives findings with substantial implications for economic policy.

Perry addresses children’s experiences of the transition. As Lewis (2007) and others have emphasized, children come with a raft of interpretive issues that are very different from those of adults—and which are infrequently taken into consideration. Perry discusses these with an eye towards studies of the epidemiologic and demographic transition and an emphasis on how they can be accommodated and resolved. For instance, social definitions like “infant” carry cultural expectations, imply behavioral shifts, and therefore produce different mortality risks. Perry utilizes the Wellcome database to interrogate the validity of Omran’s proposition that infant mortality and morbidity declined as part of the demographic and epidemiologic transitions. This study embodies the interdisciplinary approach favored in this volume; these skeletons represent the living populations upon whose vital statistics Omran’s model is based, allowing an explicit, dynamic interrogation of these two lines of evidence.

While there are many ways that social inequality expressed during the transitions (see Schell, this volume), de la Cova (this volume) addresses the influence of race and class on populations undergoing the transition, specifically lower-class African-American and Euro-American communities. Using historical and well-documented skeletal evidence from anatomical collections, de la Cova investigates whether urbanization, industrialization, the Civil War, emancipation, reconstruction, and in-migration differentially affected the health of African-American and Euro-American males and whether traces of the second transition can indeed
be found within these communities. This study demonstrates how scanty historical evidence can be integrated and interrogated against skeletal evidence to reconstruct the health experiences of otherwise invisible, highly underrepresented groups and how this evidence can be used to identify the intertwined effects of racial prejudice and tensions and poverty on experiences of the transition.

**Part 5: The Environment and the Second Epidemiologic Transition**

Studies of the historical relationship between environmental quality, industrial pollution, and the rise of chronic and NCDs can also greatly benefit from interdisciplinary approaches. Omran’s model specified an increase in ‘man-made’ [sic] diseases, which anticipated the roles that pollution and other by-products of the industrial age currently play in the disease process (Caldwell, 2001a). As Schell (this volume) discusses, studies of modern populations suggest that exposure to industrial by-products, such as lead and mercury, have insidious effects on human health, particularly for lower-class, minority, and other marginalized communities, who tend to have higher rates of exposure. For instance, industrial water and air pollution has been linked to reduced life expectancy (Pope et al., 2009), allergies (Saxon and Diaz-Sanchez, 2005), and other conditions. Both Schell and Millard and colleagues (this volume) state that this dynamic likely existed during industrialization in the 19th and 20th centuries as well and that anthropogenic toxicant exposure should be evaluated as a defining characteristic of the second transition. However, few studies have assessed this issue.

One approach to this issue involves integrating geochemical evidence of toxicant exposure derived from human tissue with documentary evidence and data from environmental toxicology and human biology. Levels and sources of exposure as well as the biosocial consequences of exposure, such as impairment, can be reconstructed through skeletal and documentary data. This approach could, for instance, elucidate whether contemporary relationships between exposure, morbidity, mortality, and social inequality have time depth, linked to long-term processes of modernization, urbanization, and industrialization, or instead reflect purely current conditions. Here, Schell provides a historical framework for thinking about industrial toxicant exposure, highlighting morbidity issues among modern communities. Schell reminds us that social inequality—more so than direct proximity to polluted industrial areas—is one of the primary determinants of exposure in the present and likely in the past. Millard and colleagues present a case study demonstrating this interdisciplinary approach. They employ documentary data, trace element analysis, and lead isotopes to track the influence of rural versus urban environments, socioeconomic status, and gender on anthropogenic lead exposure in skeletons from 17th- to 18th-century London and investigate the source of lead exposure and evidence for chronic disease morbidity from toxicity. In addition to demonstrating the value—and limitations—of such an approach, this study and its findings complicate our understanding of which segments of urban, industrial populations suffered morbidity and mortality from exposure to industrial pollution.

In addition to chronic and NCDs like cardiovascular disease, diabetes, and stroke typically recognized in the second epidemiologic transition, some studies have also shown an increase in chronic inflammatory conditions such as allergic and autoimmune diseases in high-income nations since the mid-20th century (Aberg et al., 2005). As Zuckerman and Armelagos (this volume) highlight, many researchers have interpreted this through the hygiene hypothesis, which posits that sanitary improvements and the consequent lack of childhood exposure to parasitic and symbiotic microorganisms have deprived our immune systems of key modulatory stimuli and increased our inflammatory responses.
This hypothesis, embedded in evolutionary medicine, grants a historical trajectory to current diseases and therefore opens up the issue to investigation using archaeological and historical evidence. To encourage this, Zuckerman and Armelagos provide a set of best practice guidelines for conducting interdisciplinary research on levels of hygiene, sanitation, and exposure to symbiotic microorganisms and helminthic parasites in past and recent populations. The goal is to open dialogue between archaeologists, archaeoparasitologists, immunologists, and epidemiologists on this subject.

Reinhard and Pucu de Araújo (this volume) take up part of this challenge with an archaeoparasitological analysis of the first and second transitions in Europe and the Americas. This study fuses data and methods from parasitology with documentary and archaeological data to reconstruct household hygiene, sanitation levels, and changes in—and the effectiveness of—sanitation systems in various environments before and during the transitions. Archaeoparasitological evidence had yet to be applied to studies of the second epidemiologic transition but has the potential to not only provide direct evidence for our evolutionary relationships with parasites but also indirect insights into sanitation systems, water quality, and hygiene in areas and time periods backed by incomplete or absent documentary evidence.

**Conclusion**

This volume demonstrates how theoretical approaches, methods, evidence types, and data sets from diverse disciplines can be critically integrated to generate novel insights into the dynamics of the second epidemiologic and demographic transitions, particularly into issues that remain inscrutable in vital records, cause-of-death data, and census records. Health is most insightfully approached as a holistic phenomenon, with diverse psychological, social, cultural, biological, and ecological components, cross-cultural variation, and multiple levels of causation, and this can be best captured through diverse perspectives. Hopefully, other researchers will build upon and ultimately move past these studies to not only resolve lingering questions about the transitions but also generate new ones and devote this information to better understanding and predicting patterns of health and disease in contemporary populations.

**References**


Part 1

Causes of the Second Epidemiologic Transition
Chapter 2

Infectious Disease in Philadelphia, 1690–1807: An Ecological Perspective

Gilda M. Anroman
School of Pharmacy, Notre Dame of Maryland University, Baltimore, MD

Introduction

The epidemiologic transition model broadly describes the changing relationships between humans and their diseases and provides a means for understanding the evolution and spread of emerging diseases (Harper and Armelagos, 2010). The dramatic decline in mortality from infectious diseases (often referred to as the second epidemiologic transition) that became evident in the later years of the 19th century is often attributed to advancements in medical science and public health. Although these advancements likely contributed to this decline, they were by no means the only factors. The fundamental role of cultural, social, economic, and political conditions in modulating the ecological opportunities for infectious diseases is a dominant theme running through the ancient narrative of the interplay between human ecology and the microbial world. As early as 1940, Burnet (1940) argued that interactions between human beings and infectious agents are so complex that they can only be understood in the context of their mutual relationship to the global ecosystem. It was Dubos (1959), however, who conceived of the entire process as taking place within a total environment. He explained that “the process of living involves the interplay and integration of two ecological systems” (1959: 10). These systems include the community of interdependent cells, body fluids, and tissue structures that make up an organism’s internal environment and all the living and inanimate things with which it comes into contact. Although he did not define it explicitly, the total environment evidently encompasses one’s physical, biological, psychological, cultural, political, socioeconomic, and historical universe. As living things ordinarily achieve an unsteady and temporary ecological equilibrium sufficient for survival, any change in the constellation of circumstances under which the equilibrium evolved can upset the balance. Under these circumstances, it is possible for disease to swamp the host defenses if the change is too sudden for adaptive mechanisms (Dubos, 1959).

This case study uses an ecological perspective to understand the patterns of infectious disease in Philadelphia between the years 1690 and 1807. This study interprets the historical record with conceptual guidance from the health sciences to set Philadelphia’s health crises
in a historical framework that shows people interacting *with*, rather than acting *within*, their total environment (Dubos, 1959; De Bevoise, 1995). Using an interdisciplinary model to incorporate thinking from the biological sciences, anthropology, and history, an ecological approach was fashioned here to understand the Philadelphia experience. Typically, three general forces can affect the burden of infectious diseases in a given population: change in abundance, virulence, or transmissibility of microbes; an increase in probability of exposure of individuals to microorganisms; and an increase in the vulnerability of hosts to infection and to the consequences of infection. A wide range of biological, behavioral, cultural, and social factors can influence one or more of these forces. Many are interrelated, and multiple synergies exist (Wilson, 1995). Consequently, an ecological approach allows us to see human health as an outcome of multiple, reciprocal, and continuing interactions between pathogens, hosts, and their pervasive environment.

The period between 1690 and 1807 was selected for this study for two reasons. First, it was a time during which the population of Philadelphia underwent a significant transition with respect to its disease environment. This period was characterized by events that considerably changed the risk factors for disease through alterations in population levels, pathogens, and human behavior. It was also characterized by intense change in the physical environment of the city. Short-term transitions are often distinguished by the introduction of new pathogens, new therapies, environmental modifications, or demographic changes brought about by contact with outside groups (Swedlund and Armelagos, 1990). Philadelphia experienced not one but many of these transition factors, and the extraordinarily high levels of morbidity and mortality in the city reflected this. As a result, this period in Philadelphia’s history provides an excellent opportunity for scholars to study disease *emergence* as a dynamic feature in the interrelationships between people and their sociocultural and ecological environments. Second, the data for this type of study was readily available, thanks to the meticulous compilation and reconstruction of Philadelphia’s vital rates by Klepp (1991), supplemented by church registers and other records.

**Background**

Historical inquiry can bring a valuable perspective to the understanding of disease emergence by focusing on “the consequences of human actions and the conditions that permit certain developments” (Morse, 1992: 38). The emergence and spread of microbial threats in 18th-century Philadelphia were driven by a complex set of factors, the convergence of which led to outcomes of disease much greater than any single factor might have suggested. Genetic and biological factors, for example, allow microbes to change and can make people more or less susceptible to infections. In addition, changes to the physical environment can impact the ecology of vectors and animal reservoirs, the transmissibility of microbes, and the activities of humans that expose them to certain threats (Smolinski et al., 2003). Human behavior, however, both individual and collective, is (and was) perhaps the most significant of all. The 18th century was a time of dynamic growth and change for Philadelphia. The size and mobility of the city’s population increased the potential for pathogens to escape their prior geographic boundaries. High levels of immigration, coupled with high population densities, increased both interpersonal contact and contact between people and animals. Domesticated animals such as goats, sheep, cattle, pigs, and fowl provided novel reservoirs (Last, 2001) for zoonoses. Endemic diseases such as dysentery, malaria, and tuberculosis severely weakened their victims and increased their susceptibility to other infections. And industries such as tanning, sugar refining, and milling altered the physical environment and caused ecological disruptions. Infectious disease is a moving