Beyond One Health

From Recognition to Results

Edited by John A. Herrmann · Yvette J. Johnson-Walker

WILEY Blackwell

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This edition first published 2018

Registered Office(s)
John Wiley & Sons, Inc., 111 River Street, Hoboken, NJ 07030, USA

Editorial Office
111 River Street, Hoboken, NJ 07030, USA

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Wiley also publishes its books in a variety of electronic formats and by print-on-demand. Some content that appears in standard print versions of this book may not be available in other formats.

Library of Congress Cataloging-in-Publication Data

9781119194491 [paperback]

Cover design: Wiley
Cover image: Courtesy of John Herrmann

Set in 10/12pt Warnock by SPi Global, Pondicherry, India

Printed in the United States of America

10 9 8 7 6 5 4 3 2 1

For Wanda, who always knew the way; and for Anne and Kate, who are my guideposts.

J.A. Herrmann

For Mom, Lauren, and Lamar – my shelter from the storm; and for Ikenna, Ndidi, and Amaya – my windows of hope.

Y.J. Johnson-Walker

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Foreword

We encourage you to set aside time to read *Beyond One Health: From Recognition to Results*. We hope that you will be as inspired by its contents as we are.

One Health is one of the great innovations of our time. It is an idea, a concept, a way of thinking and working, and a means to organize action. One Health starts from a recognition that 75% of the new infections affecting humans come from animals. The risks of animal diseases can be decreased through proper attention to livestock health in livestock production: the One Health approach guides efforts to intensify production. It recognizes the benefits of food systems that are sensitive to nutrition and the threats posed by infections that are resistant to antimicrobial therapies.

The One Health idea came to life in 2004 as scientists considered how best to tackle diseases that move between human, domestic animal, and wildlife populations. It reflected experiences with the Ebola virus disease, avian influenza, and chronic wasting disease. It is set out as the Manhattan principles (https://www.cdc.gov/onehealth/pdfs/manhattan/twelve_manhattan_principles.pdf) for *One World, One Health.*[1] It is an international, interdisciplinary approach for tackling threats to the health of life on Earth. It has practical application for reducing risks of unsafe foods and diseases that move from animals to humans.

1 Organized by the Wildlife Conservation Society and hosted by The Rockefeller University (http://www.oneworldonehealth.org).

One Health connects science and systems to the needs of society. It has matured into a new way of thinking and working and contributes to the health of both humans and animals. It links several disciplines that focus on health. It helps professionals to see their work differently and to do it with new purpose. It stimulates integration when remaining separate is less effective. One Health frames how we speak and act: it encourages us to focus on the interfaces between human, animal, and environmental systems. It helps us make sense of multiple interacting determinants of illness. It helps us to better reduce risk and prepare for threats.

Many of us with coordination responsibilities have found that One Health makes our joint working more effective and efficient. It makes sense on the farm, in the factory, and at home, encouraging us to prevent costly outbreaks.

More recently, One Health has helped with restructuring institutions and transforming education. It helped drive collaboration between the World Health Organization (WHO), the World Organisation for Animal Health (OIE), and the Food and Agriculture Organization of the United Nations (FAO). It stimulated new academic departments and degree programmes. It provided a basis for local and national governments to combine animal, human, and environmental health programs, and to reap economic benefits.

Beyond One Health: from Recognition to Results offers us an update on One Health topics from the perspectives of different professional and academic disciplines. It includes

an analysis of different threats to people and planet (including zoonoses and climate change), the epidemiology that underlies One Health, as well as the evidence base for different One Health policies and their benefits. It shows how One Health is best approached from a systems perspective and explains the importance of good leadership in making One Health a reality.

If we want to learn how One Health can best be applied in practice, we should study its use in different situations. In this book, we can see how One Health approaches help when analyzing risk and devising prevention, preparedness, and response strategies; when monitoring the evolution of threats and establishing early warning systems; or when prioritizing actions and coordinating actors during implementation. We can understand how One Health has been used in responses to avian influenza, yellow fever, Zika, Middle East respiratory syndrome (MERS), and Ebola.

When combining animal and environmental health practice, we must be sensitive to variations in motivations, responsibilities, and accountability of practitioners in these disciplines. In our experience, the One Health approach is especially useful when coalitions of actors are being established and a consensus is being built. It should be applied in ways that are sensitive to context, adapted to capabilities of systems (for public, veterinary, and environmental health), and adjusted to ecosystem, economic, and societal realities of interfaces between humans, animals, and nature.

We are starting to see One Health approaches being used to frame analyses of costs, benefits, acceptability, and scalability of different interventions. Academic groups are often asked to provide the evidence base for One Health policies and interventions. Their inputs are most helpful when interdisciplinary research methods are used. This is especially necessary when exploring links among environmental dynamics, disease vectors, pathogens, and human susceptibility.

Enlightened approaches like One Health – which focus on prevention and response from the perspectives of multiple disciplines – are vital to success in achieving the 2030 Agenda for Sustainable Development and in building a common future for all. This book will help you move along that path.

Chadia Wannous
David Nabarro

Chadia Wannous, PhD, is a Public Health professional and expert in prevention, emergency preparedness, and risk reduction for health threats. She previously served in several senior policy advisory positions with the UN.

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Foreword

The naturalist and conservationist, John Muir, once stated, "When one tugs at a single thing in nature, he finds that it is attached to the rest of the world." The interconnectedness that Muir described in the early twentieth century is much more profound today, and much more consequential, regarding our health. The globalization of trade, travel, information, and investments, integrated and consolidated global food systems, urbanization, and a group of anthropogenic drivers that negatively impact our ecosystems, have created a new dynamic and an unprecedented interdependence among the health and well-being of people, animals, and our environment. The complex construct that describes these three domains of health is termed "One Health" and, indeed, tugging on any one of these domains demonstrates their significant attachments to one another. As a corollary to this axiom, we can no longer focus on health through a single lens or discipline.

Our new twenty-first century interdependence, including social, economic, political, and biological factors, has created new threats and risks to our health and has produced ecological changes that have fractured our planet. Several decades ago, the concept of One Health re-emerged from past medical thinking and gained important traction and acceptance. Recently, there have been many articles and books published focusing on One Health but, fortunately and very timely, this book has added special insights and brought together diverse disciplines and thinking to give us a better understanding of One Health in our contemporary lives, with an important and unique emphasis on operationalizing the concept. The book's authors have substantially improved our understanding of the key themes of One Health, added to our knowledge base, and stressed that new skills and competencies need to be acquired to successfully address the threats to human, animal, and ecosystem health.

The factors and drivers of our interdependent world, and increasingly risky lives, show no signs of abating; rather, they are accelerating. These drivers are leading to the intensification of the human-animal-ecosystem interface and causing further ecological damage. One consequence of this reality has been the dramatic increase in zoonotic diseases worldwide over the last few decades, which is thoroughly detailed in several chapters. This book also discusses the serious consequences of the degradation of our water resources and ecosystems, as well as threats to biodiversity and food security, all underpinned by climate change. The authors present evidence that our complex and interconnected world has generated a group of "wicked problems" that demand our attention and resources to resolve. A key feature of "wicked problems" is the recognition that past solutions and practices are not likely to be relevant or effective when applied to today's unparalleled challenges. A One Health mindset and an ability to work holistically across disciplines need to become the new norm to address complex problems and to take appropriate actions. In addition, we must champion new partnerships and innovations, and learn to effectively lead and manage change.

However, our medical fields continue to become progressively more specialized and, at the same time, progressively more isolated and siloed. While we appreciate the impressive advances in medicine, our health systems are increasingly disease-oriented and reactive. One Health, on the other hand, stresses disease prevention, shifting interventions closer to the origins of the problem, often in our animals and environment. *Beyond One Health:From Recognition to Results* argues that improving animal and environmental health can be a very effective and cost-beneficial public health strategy. As this text points out so well, maintaining and improving health must go beyond a strictly disease-oriented approach to consider the impact of the environment, social-economic status, genetics and human behavior, and other social determinants of health, which is truly a One Health perspective. This timely book makes the case that we need to normalize good health through this larger and more comprehensive context.

In differentiating *Beyond One Health: From Recognition to Results* from past One Health books, this book emphasizes the need to translate new knowledge into practice. We know that this transformation is a difficult and dynamic process that involves synthesis, dissemination, exchange, and finally application of One Health knowledge to the maintenance and improvement of health in all of its domains and dimensions. The book's authors acknowledge and present compelling evidence that critical gaps exist today between the promise of good health and actual results. The book reiterates that developing and implementing new strategies and polices represent the tactics necessary to support a One Health framework and plan of action. In addition, the authors argue in favor of the growing evidence that One Health thinking can offer a favorable value proposition, demonstrating that maintaining the status quo for our current healthcare delivery and disease response system is no longer acceptable, cost-effective, or scientifically valid.

While we remember John Muir as an outstanding ecological thinker, we also recognize that he was a very effective political spokesperson who understood the importance of translating science and knowledge into policies in support of conservation. Likewise, we need to move One Health from an abstract concept to a catalyst for new policies and interventions that can change the existing dynamic and improve health outcomes across all the domains of health. We understand that there are three stages of translating knowledge into practice, and this book discusses all three throughout its chapters. Awareness, acceptance, and adoption comprise the sectors of translation and all are integrated throughout the text. The authors also stress an important lesson: as we develop and adopt new strategies and policies, we also must design and carry out processes for outcome measurement and evaluation and continuous improvement for them to remain relevant and effective.

We are indebted to the editors and authors who have successfully built momentum toward a more universal acceptance of One Health and, perhaps even more importantly, have been especially instructive in helping us appreciate the need to enact new policies and shift One Health from theory to effective field implementation. They have reminded us, throughout this text, that One Health is likely just to be relegated to an academic exercise if it is not accompanied by a new value proposition, new policies, and more efficient interventions in the rapidly changing human, animal, and environmental health dynamic. Finally, we are grateful for both the intellectual and practical contributions of the book's editors and authors and are well advised to use their ideas and examples to better address the threats to our health, in all its dimensions.

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Preface

"One Health" has caught on, some 140 years after Virchow coined the term "zoonoses" and said, "between animal and human medicine there are no dividing lines – nor should there be" (Schultz, 2008). The principles of One Health are often assigned singular ownership of that conceptual triad. However, other models, such as the Ecological Model in public health, eco-social theory, EcoHealth, conservation medicine, ecological medicine, and others, also take the holistic view that individual or population health outcomes are the result of many interrelated exposures, determinants, and contributing factors, and that an understanding of them, and their relatedness to each other, is required to formulate effective public policy designed to improve health.

Much has been written about One Health, its history and importance, especially in the context of emerging infectious diseases. One cannot minimize previous essays and textbooks focused on the need for viewing modern challenges to population health through a One Health lens, or the many peer reviewed journal articles that framed their research findings as examples of the demand for One Health thinking. We also must appreciate the excellent efforts of various national and international groups devoted to promoting One Health concepts and spreading awareness of their importance. However, we are at an inflection point in world events at which it has never been more critical that policy-makers set aside their ideologies and prejudices and promote science and technology policies that affect health, broadly defined. Those policies must be based on scientific consensus drawn from independent, well-constructed, repeatable research that is published for all to read and analyzed in well-respected, peer reviewed journals. We need to get beyond the abstract and actually do. Centuries ago, the German writer and statesman, Johann Wolfgang von Goethe, counseled that knowing and willing to do something is all well and good but eventually we must actually do it.

When we received a request from our publisher to edit a textbook about One Health, we initially declined. There were already four or five excellent books that describe One Health thinking and the challenges associated with it. It was only after we discussed our interest in public policy, and our experiences in the policy formation process, that we came up with the idea to edit a One Health book that is directed at policy solutions. The title of this textbook should be instructive. Our book is intended to serve as a reference for students and professionals in many disciplines, from architecture through urban planning, and not just for those working in traditional healthcare and health-related fields. The concept of One Health, that human, animal, and ecosystem health are inextricably linked, is an idea that is, at its core, about prevention. One Health may be easy to describe but it is a challenge to operationalize as policy. One Health thinking recognizes the interrelatedness of determinants of health and uses the scientific method to discover how strongly exposures are related to outcomes. Data are tested until they are accepted as fact; those facts

can, gradually, after the iterative process of the scientific method, be translated into policy that should be designed to prevent the adverse effects of natural and human-derived phenomena on an ecosystem and to improve health.

Population growth, climate change, environmental degradation, inconsistent food production and distribution, water resource management, nonparticipatory governance, lack of civil society – all of the many determinants of global health – indicate that we are at a critical point in world history. To make significant improvements in global health, to improve the lives of global societies, we must engage thinkers from virtually all academic and professional fields and develop solutions, in public policy and in individual behaviors, that are effective, efficient, and sustainable. This is true One Health.

So, it is in this context that we offer this collection of critical population health topics, written by an international group of experts, that addresses not only the technical aspects of their topics but also offers potential policy solutions to help mitigate current threats and to prevent additional threats from occurring. Too often, public policy is based on the short-term benefit for the few at the long-term cost to the many. Too often, short-sighted policies defer current costs to future generations.

Reference

Schultz, M. (2008). Rudolf Virchow. *Emerging Infectious Diseases* 14(9), 1480–1481.

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Section 1

The Science of One Health

1

Epidemiology: Science as a Tool to Inform One Health Policy

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1.1 Introduction

Epidemiology is the study of disease dynamics in populations. It seeks to understand patterns of disease as a means of identifying potential prevention and control measures. It has been described as "an interesting and unique example of cross-fertilization between social and natural sciences" (Vineis, 2003). The basic principle of epidemiology is that disease is not a random event. Each individual in a population has a unique set of characteristics and exposures (risk factors) that determine his or her probability of disease. Clinical medicine is focused on the health of the individual while epidemiology and public health seek to apply assessment of risk factors at the community level. Understanding how those risk factors impact a community provides public health officials with the tools to develop policies and interventions for disease control and prevention in the community as a whole.

The One Health concept is coherent with the principles of epidemiology because risk factors for many diseases occur at the interface between humans, animals, and the environment. Failure to consider the interactions between them may result in public health policies that fail to effectively control disease and protect the environment. The One Health triad (Figure 1.1) of humans, animals, and the environment is analogous with the other triads that epidemiologists use to describe disease dynamics within a population:

- The host, agent, environment triad (Figure 1.2) is used to describe the interplay between these three key components of infectious disease transmission. Changes in any of these components alters the probability of disease.
- The three states of infectious disease status are illustrated by the susceptible, infected, removed (SIR) triad (Figure 1.3).
- Outbreaks of disease are characterized in terms of person or animal, place, and time as the first step of identifying the population at risk.
- Risk factors for disease causation are categorized as: necessary, sufficient, and component causes (Figure 1.4).

The goal of public health policy is to prevent transmission of disease agents to the susceptible segment of the population by controlling and treating disease among the infected and increasing the segment of the population that is removed (recovered or resistant). Identification and isolation of cases, quarantine of the exposed, and vaccination of the

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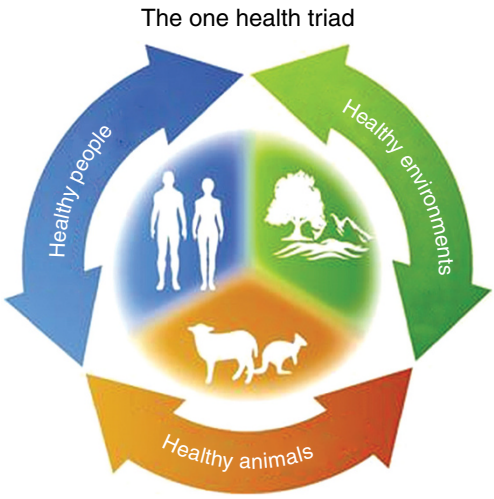

Figure 1.1 The One Health triad. *Source:* Thompson, 2013. Reproduced with permission of Elsevier.

susceptible are the primary tools employed by public health practitioners for infectious disease control. Development of effective programs to accomplish these goals requires an understanding of the:

1) Causes of disease (etiologic agent, pathophysiology, and risk factors.
2) Impact of the disease on the population (number of cases, ease of transmission, economic and social impact).
3) Natural course of the disease (reservoirs for the agents of disease, means of introduction of the agent into the population, period of infectivity, severity of disability, length of immunity, and potential for long-term sequelae) (Figure 1.5).

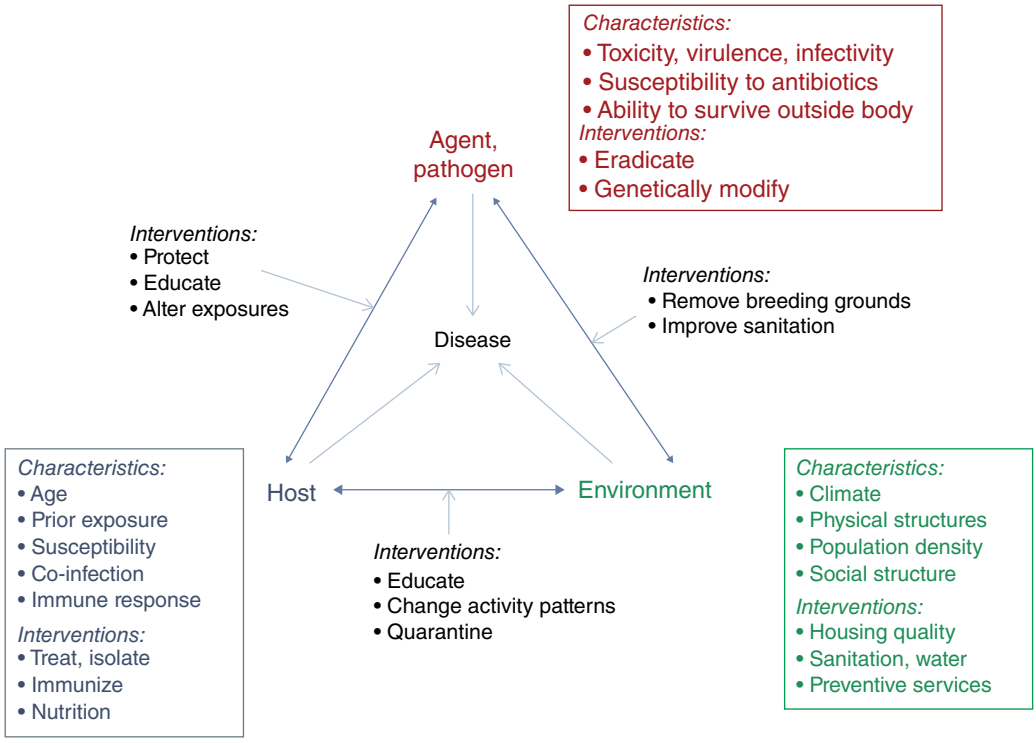

Figure 1.2 The "epidemiologic triad" of infectious disease summarizes the factors that influence an infection, and the measures you might take to combat the infection. *Source:* Used with permission from Ian McDowell (http://www.med.uottawa.ca/SIM/data/Pub_Infectious_e.htm#epi_triad).

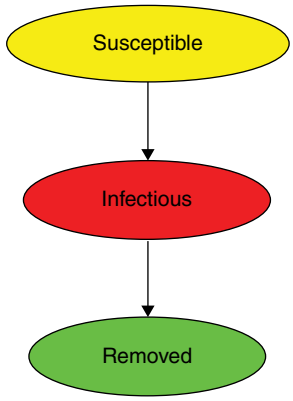

Figure 1.3 Infection modeling: the SIR model. **Susceptible** nodes – have not been infected yet and are therefore available for infection. They do not infect other nodes. **Infectious** nodes – have been infected and infect other nodes with a certain probability. **Removed** (recovered) nodes – have gone through an infectious period and cannot take part in further infection (neither actively nor passively). *Source:* Used with permission from Michael Jaros (http://mj1.at/articles/infection-modelling-the-sir-model/).

The goals of this chapter are to elucidate how epidemiology can 1) provide a tool for understanding the causes, impacts, and course of disease in human and animal populations within various ecosystems, and 2) form the basis for evidence-based health and environmental policy development.

1.2 Enhancing Our Understanding of Health and Disease

1.2.1 Causes of Disease

Epidemiology is unique among biomedical investigative approaches because of the observational nature of many of the study designs. Unlike laboratory studies, the epidemiologist often studies a naturally

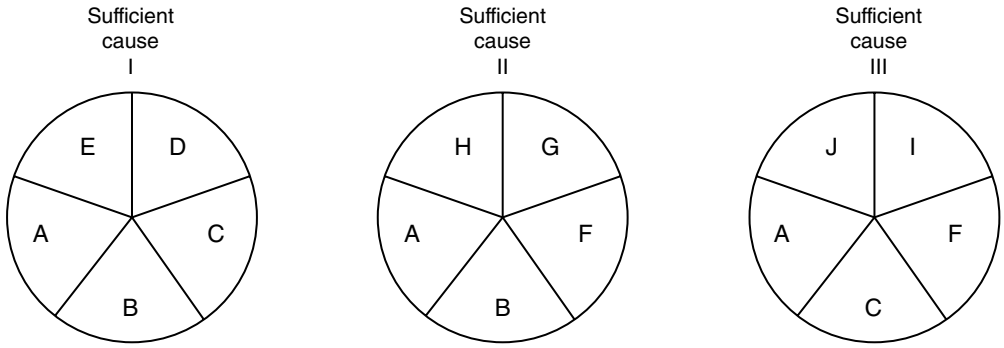

Figure 1.4 Necessary, sufficient, and component causes. The individual factors are called component causes. The complete pie (or causal pathway) is called a sufficient cause. A disease may have more than one sufficient cause. A component that appears in every pie or pathway is called a necessary cause, because without it, disease does not occur. *Source:* Rothman, 1976. Reproduced with permission of Oxford University Press.

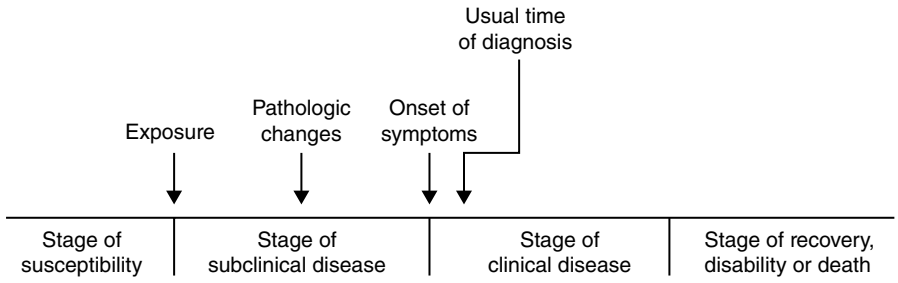

Figure 1.5 Natural history of disease timeline. *Source:* CDC, 1992.

occurring disease within a free-living population in which study subjects are not assigned to intervention groups (except in the case of clinical trials). Individuals may have a variety of independent exposures during the study period. Whether studying human or animal populations, the epidemiologist seeks to identify exposures that are associated with the probability of disease using statistical analysis of data from carefully documented exposures and outcomes. However, even if a statistically significant association between an exposure and disease outcome has been identified, that does not necessarily mean that a cause and effect relationship has been established. Much more rigorous standards have been set for establishing a causal relationship between a risk factor and the probability of disease.

1.2.1.1 Deterministic Models of Disease

Criteria for establishing causation for infectious disease have been described since the nineteenth century. Research by Robert Koch, Friedrich Loeffler, and Jakob Henle resulted in the Koch–Henle postulates published in 1882 (Sakula, 1983; Gradmann, 2014) (Figure 1.6). While this approach is useful when seeking to identify the etiologic agent responsible for an infectious disease, it has many limitations. The simplistic approach of a deterministic model for establishing disease causation is insufficient for identifying risk factors for chronic noninfectious diseases (such as type II diabetes) or even infectious diseases with a multifactorial etiology (such as new variant Creutzfeldt–Jakob disease, or CJD). In more recent years more complex

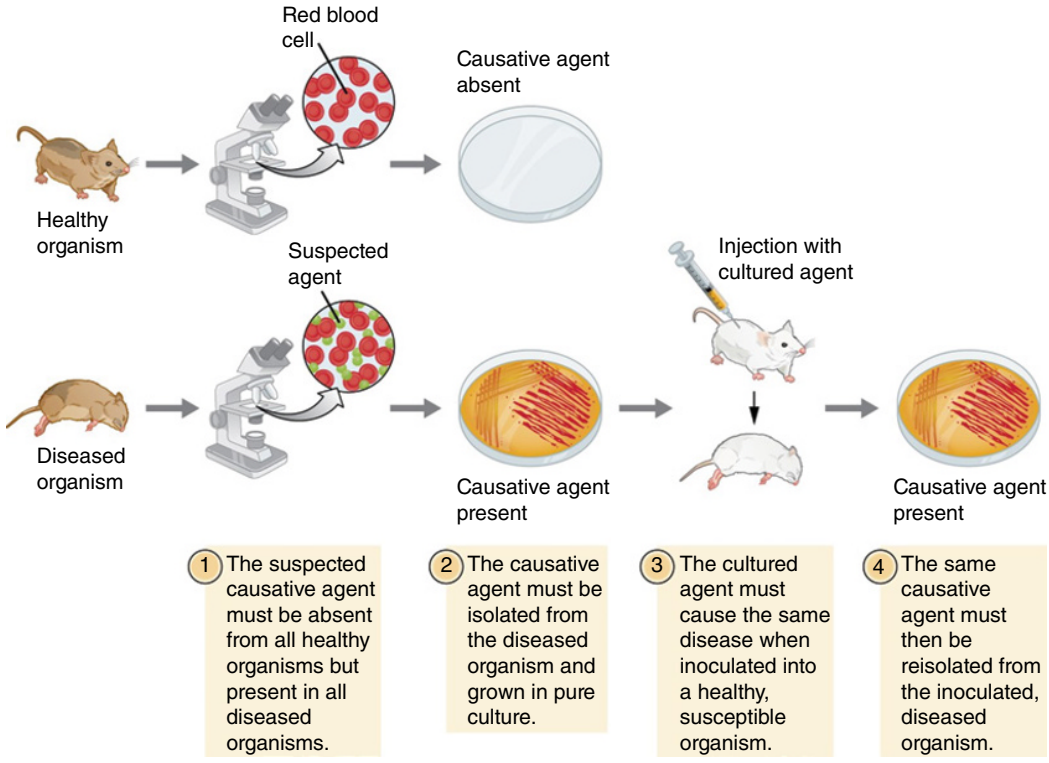

Figure 1.6 The steps for confirming that a pathogen is the cause of a particular disease using Koch's postulates.

models have been used to establish a causal relationship between a putative risk factor and disease.

1.2.1.2 Hill's Causal Criteria

Austin Bradford Hill published "The environment and disease: association or causation?" in 1965 (Hill, 1965). The manuscript describes nine criteria necessary for establishing a causal relationship between a risk factor and a disease:

1) *Strength of association:* the greater the magnitude of the association between the risk factor and the outcome, the more likely the relationship is to be causal.
2) *Temporality:* the risk factor must precede the onset of the disease.
3) *Consistency:* the same association should be observed in multiple studies with different populations.
4) *Theoretical plausibility:* the association should be biologically plausible and consistent with the pathophysiology of the disease.
5) *Coherence:* the association should be consistent with what is known about the disease.
6) *Specificity in the causes:* a risk factor should be associated with a single disease or outcome.
7) *Dose-response relationship:* as the dose of the risk factor is increased the probability and severity of the disease should increase in a linear fashion.
8) *Experimental evidence:* data from *in vitro* studies and animal models should support the causal association between the risk factor and the disease.
9) *Analogy:* similar causal relationships should be known.

The nature of these criteria makes it impossible for a single observational study to establish a causal relationship between an exposure and a disease outcome. The criterion of consistency requires that multiple studies, in different populations, show the same association. The criterion of temporality also requires that the association be demonstrated in prospective studies. Prospective study designs monitor the study population prior to the onset of disease and follow their exposures over time until the disease of interest occurs. However, as we learn more about the complexity of the interactions between hosts and their exposures, limitations of the Bradford Hill Causal Criteria have also been described (Rothman, 2012). Some of Hill's Causal Criteria have been challenged by known causal associations that are contradictory. Specificity of effect, dose-response gradient, and coherence are all criteria whose validity has been challenged.

The criterion of specificity fails to acknowledge the potential for a single exposure to cause a multiplicity of pathologic effects. One well-known example of this is seen with exposure to tobacco smoke, which is associated with lung cancer, chronic obstructive pulmonary disease, heart disease, stroke, asthma, impaired fertility, diabetes, premature/low birthweight babies, blindness, cataracts, age-related macular degeneration, and cancers of the colon, cervix, liver, stomach, and pancreas (American Lung Association, 2017).

Many disease-causing exposures fail to produce a linear dose-response gradient. Goldsmith and Kordysh (1993) reviewed the literature for examples of nonlinear dose-response relationships and concluded that nonlinear causal relationships are equally as common as linear associations. Their analysis of the literature concluded that dose-response relationships are often nonlinear when countervailing outcomes are likely. They cautioned against linear extrapolation of dose-response data to develop policies and regulations for the protection of human populations. Exposures such as ionizing radiation and vitamin toxicity have been reported to produce U- or J-shaped dose-response curves (May and Bigelow, 2005). Inadequate sample size in the research study, insufficient range in the exposure dosages, and variability in individual susceptibility are all factors that

impede the identification of these nonlinear dose-response causal relationships.

The criterion of coherence doesn't allow for paradigm shifts in models of disease causation. Identification of new mechanisms of disease pathogenesis may require elucidation of relationships that are not coherent with the current body of knowledge about the disease process. This is illustrated by the work of Marshall and Warren (1984) and their discovery of the role of *Helicobacter pylori* in the etiology of gastritis and peptic ulcers. Prior to their research, acid production was believed to be the key risk factor for the development of gastritis and peptic ulcers. Gastritis was thought to be a chronic inflammatory disease; the concept that it was actually due to a bacterial infection, was not coherent with the theory of the disease at the time of the findings by Marshall and Warren.

1.2.1.3 Multifactorial Models of Disease Causation

Krieger (1994) describes the transition in epidemiology from a focus on acute and infectious diseases to research focused on chronic disease. These more complex disease etiologies were first described as a "web of causation" in 1960. Multifactorial causes of disease have been framed as host-agent-environment models and social determinants of health. The public health application of these models is manifested as identification of the necessary component causes of disease and directing policies and interventions at those causes that are most amenable to alteration (see Figure 1.4).

In summary, epidemiology has evolved from a monocausal (deterministic) model to the multicausal concept of the "web of causation" (Vineis, 2003). The models that seek to describe disease causation continue to evolve. More recently, an "ecosocial framework" has been proposed as a more holistic, comprehensive approach to describing the how and why of disease occurrence (Krieger, 1994) (Figure 1.7). Unlike the web of causation, this model takes a One Health approach to understanding disease in human populations. Krieger concludes that "encouraging a social and ecologic point of view, this image also serves as a reminder that people are but one of the species that populates our planet; thus implies that the health of all organisms is interconnected."

1.2.1.4 Breaking the Chain of Transmission

The goal of epidemiology is to enhance the health of populations. The rationale for researching risk factors for disease is to identify policies and interventions that can be employed to prevent disease. One of the most important lessons of epidemiology is that disease can be controlled even when there is incomplete knowledge of the etiologic agent responsible for the disease. Louis Pasteur conducted research that led to the germ theory of disease between 1860 and 1864. Prior to this discovery, John Snow's classic work on the epidemiology of the 1854 cholera epidemic in London demonstrated that an infectious disease outbreak can be controlled by understanding risk factors for disease, even if the etiologic agent is unknown. In the 1854 outbreak, new cases of cholera were prevented by removing the handle from the Broad Street pump once the water source was identified as being the important exposure associated with cholera deaths in that part of London.

More recently, the first case of acquired immune deficiency syndrome (AIDS) was reported in 1981 and it wasn't until 1984 that the etiologic agent, human immunodeficiency virus (HIV), was discovered. However, in 1982, it was known that the disease was caused by a blood-borne or sexually transmitted virus and high-risk segments of the population had been identified. Even before the etiologic agent had been discovered, measures to prevent disease transmission were identified, including condom use and avoidance of needle-sharing among IV drug users (https://history.nih.gov/nihinownwords/docs/page_02.html) (Poundstone et al., 2004).

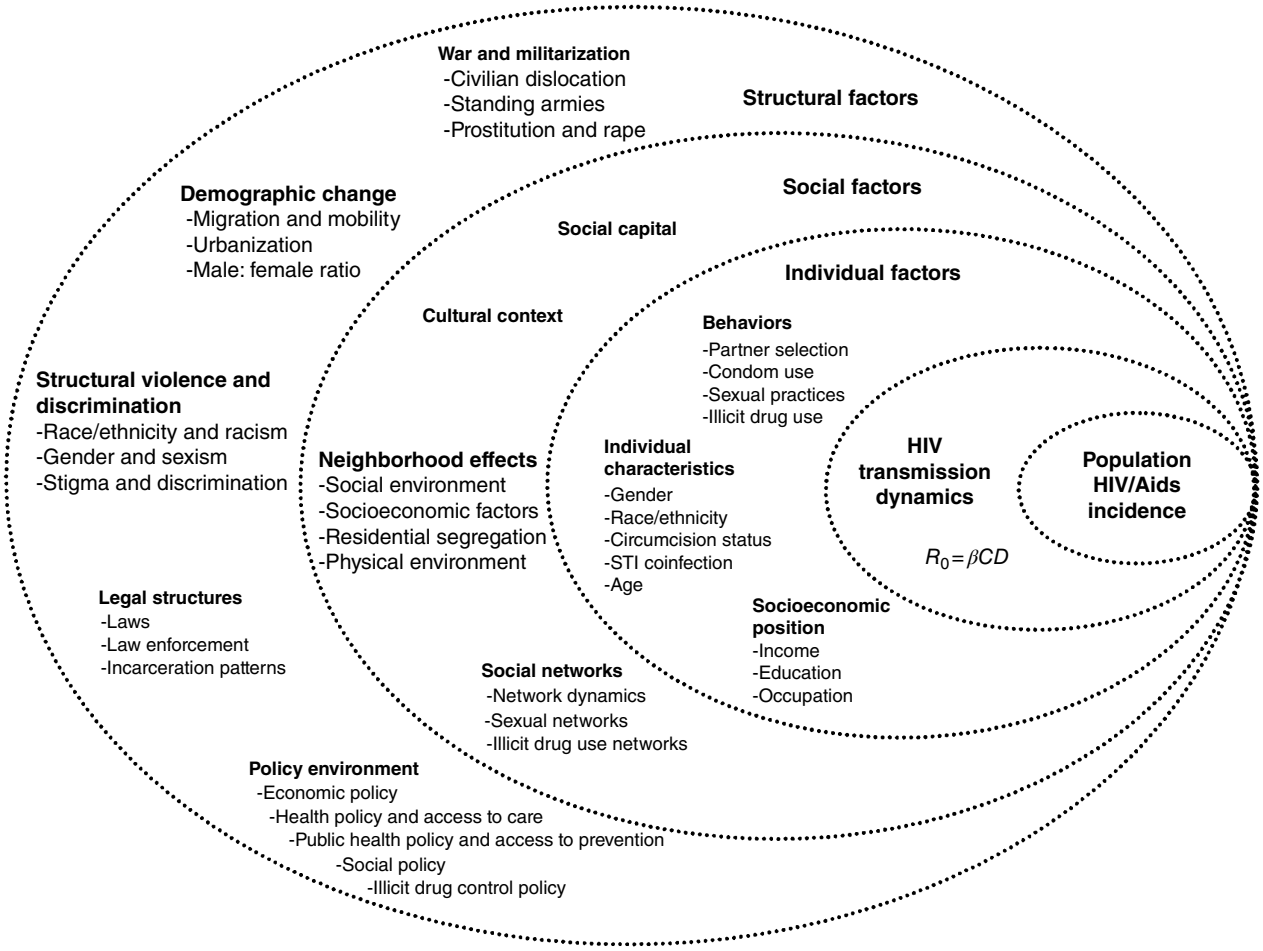

Figure 1.7 Ecosocial framework. An heuristic framework for the social epidemiology of human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome (AIDS). The dotted lines separating the levels illustrate the porous nature of the distinctions made between levels of analysis. In reality, there are extensive linkages between factors at all levels that give rise to observed epidemic patterns. STI, sexually transmitted infection. *Source:* Poundstone et al., 2004. Reproduced with permission of Oxford University Press.

1.2.2 Assessing the Impact of Disease

The foundation of assessing a population health problem is determining the impact of the disease on the population. How big is the problem? Answering this question requires establishing the:

- number of individuals in the population with the disease (prevalence);
- number of new cases of disease that will occur in a given period of time (incidence);
- ease with which the disease spreads within the population (infectiousness);
- severity of the illness (agent virulence); and
- cost of the disease to society.

The *prevalence* of disease is a measure of the number of cases of disease at a given point in time. Prevalence of disease includes both recently diagnosed cases and chronic cases that have lived with the disease for some time. Knowing the prevalence of a disease in a community allows public health personnel to determine the resources necessary to manage the disease in the community. The *incidence* of disease is focused only on those new cases of disease identified within a given time period. Incidence tells how frequently new cases of the disease are occurring.

Infectiousness is a description of how easily an agent is transmitted from one host to another. Some agents are inherently very infectious and can spread quickly and easily to multiple susceptible hosts. The basic reproduction number, or R_0, is a measure of infectiousness of an agent in a totally susceptible population. The R_0 is the number of new cases of disease a single case will generate during its infectious period. Examples of highly infectious pathogens include measles virus in humans and foot-and-mouth disease (FMD) virus in livestock. Measles has an R_0 of 12–18 (CDC and WHO, 2014) meaning that in an unvaccinated population, each case of measles can be expected to infect an additional 12 to 18 people. A recent study of FMD transmission in dairy cattle reported an R_0 of infinity for nonvaccinated dairy cattle in the same pen. In contrast, other agents are inherently less infectious. Estimates of infectiousness of the seasonal influenza virus report an R_0 of approximately 1.3 (Biggerstaff et al., 2014). R_0 is an inherent characteristic of an infectious agent. However, it is the interaction between the population, the environment, and the agent that best describes the spread of disease within a population. This is expressed by the effective reproduction number (R). "R" is the average number of new cases generated by a single case in a population that consists of both immune and nonimmune individuals. If R is less than 1.0, sustained transmission within a population cannot occur. As long as the R is greater than 1.0, meaning each case spreads the disease to more than one new case, the disease will continue to spread in the population. Without intervention the entire population will eventually get the disease.

The basic reproduction number (R_0) not only provides information about how likely an agent is to cause an epidemic, it also indicates the percentage of the susceptible population that must be vaccinated or be immune through natural infection to prevent disease transmission. This is referred to as *herd immunity* – a state in which enough members of the population are immune to the disease to prevent spread, thus protecting those who are not immune. So, for measles, 83–94% of the population must be vaccinated to achieve herd immunity (CDC and WHO, 2014), while for influenza it has been reported that only 13–40% (depending on the influenza strain) of the population needs to be vaccinated to establish herd immunity (Plans-Rubio, 2012).

The practical application of this information is that it can be used to direct public health interventions that have the potential to stop transmission. Vaccination programs reduce the number of individuals in the population who are susceptible to the disease, and the population can achieve a state of herd immunity if a sufficient percentage is vaccinated. Case finding efforts, combined with treatment and isolation of infectious

individuals, and education programs, such as hand washing and social distancing campaigns, can reduce the number of individuals in the population who are exposed to the agent, thereby preventing spread of the agent to new susceptible hosts.

The *virulence* of an agent is an indication of the severity of the illness it causes. Some pathogens cause mild, self-limited illnesses with few clinical signs, while more virulent agents result in debilitating disease or even death. Agent virulence is assessed using the *case-fatality rate* (CFR). The CFR is simply the rate of death due to a disease among all cases of the disease. The CFR for chickenpox in children (varicella) is 0.001% or 1 in 100 000 (Heymann, 2008). In comparison the CFR for rabies is 100% (WHO, 2017). Thus, the virus causing rabies in humans is much more virulent than that causing chickenpox.

In addition to considering the number of sick individuals, the rate of disease spread, and the severity of the illness, assessing the impact of a disease must also take into consideration the burden of the disease on society (Figure 1.8). Direct economic costs of disease include the cost to diagnose, treat, or prevent the disease. Indirect economic costs may include lost productivity due to absenteeism from work or losses due to declines in trade and tourism caused by fear of the disease, and so forth. Lastly the social disruption caused by the disease, or fear of the disease, can be more costly than the actual cases of disease. Remnants of this disruption may last years beyond the disease event.

It is easy to see how an outbreak of a high-incidence, rapidly spreading disease, caused by a very virulent agent, can have a huge economic and social impact on a community. This was apparent during the 2014–2016 West African Ebola virus disease outbreak in which there were an estimated 28 652 human cases and 11 325 deaths in 10 countries (CDC, 2016). The 2015 United Nations Development Group (UNDG) report on the socioeconomic impact of Ebola virus disease in West African countries indicates that the impact of the 2014–2015 Ebola outbreak was pervasive in the affected countries: labor markets shrank; access to food and the quality and quantity of food consumed was decreased; access to education declined for children, due both to mortalities among educators and school staff and to school closures; access to health services declined substantially; and there was an erosion of

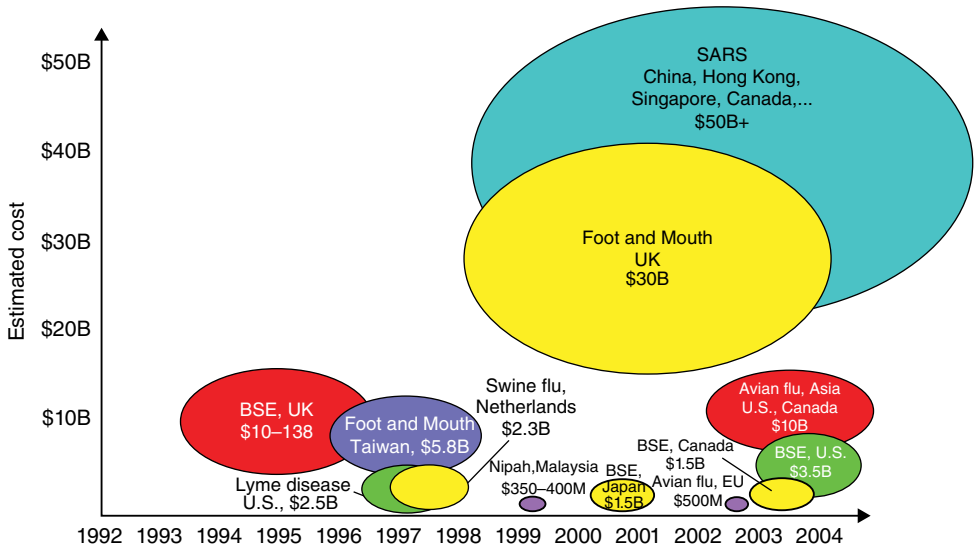

Figure 1.8 Economic impact of disease. *Source:* Karesh, 2007. Reprinted with permission from Bio-era.

communal cooperative behaviors and relationships (UNDG, 2015). Declines in gross domestic product (GDP) for the most severely impacted countries were estimated to range from 3.4% for Guinea to 13.7% for Liberia (UNDG, 2015). As a result, prior trends in poverty reduction are expected to slow or reverse in Guinea, Liberia, and Sierra Leone. In fact, the economic impact will be felt throughout the entire region. West Africa is expected to incur losses of approximately 3.6 billion US dollars per year for the period 2014–2017 (UNDG, 2015).

In some cases, the devastating socioeconomic impact of a disease outbreak is caused by a combination of illness and death in both human and domestic animal populations. Rift Valley fever (RVF) is a mosquito-borne emerging viral disease that causes severe disease in human and animal populations. First reported in Kenya in 1930, outbreaks have been documented in several countries in sub-Saharan Africa. However, in the year 2000 a large outbreak of human cases was reported in Saudi Arabia and Yemen. A total of 516 human cases of severe RVF, with 87 deaths, were documented between August and November of 2000 (CDC, 2000). A 2007 outbreak of RVF in the Sudan was reported to have caused an estimated 75 000 human cases (Anyamba et al., 2010). Symptoms in human cases may range from mild febrile illness to vision loss, encephalitis, and hemorrhagic disease in 8–10% of cases (CDC, 2013). In cattle, sheep, goats, and camels, RVF causes abortion and perinatal mortality rates in excess of 95% (Hassan et al., 2011). Outbreaks of RVF in livestock result in reduced access to food, loss of income from livestock production, and loss of export markets due to trade bans, in addition to costs to the government for disease control, surveillance, and assistance to producers (Hassan et al., 2011). A 2007 RVF outbreak in Kenya was reported to cause US$32 million in losses (Rich and Wanyoike, 2010).

However, a relatively rare disease with few cases in any community can still place a huge burden on society in terms of the direct and indirect economic and social costs of the disease. The economic and social impact of diseases like severe acute respiratory syndrome (SARS) and bovine spongiform encephalopathy (BSE, or mad cow disease) illustrate that a limited number of human cases of disease can have huge social and economic consequences on the affected community and beyond (Figure 1.8). The SARS pandemic occurred from November 2002 through July 2003. During that period there were 8098 total cases of SARS with 774 deaths across 29 countries with an estimated economic impact of US$30–50 billion. Human exposure to the prion that causes BSE in cattle is a cause of variant Creutzfeldt–Jakob disease (vCJD) in humans. There were 178 human cases of vCJD in the UK between 1995 and 2016 (CDC, 2017), while concern that BSE may pose a human health risk resulted in losses of 740–980 million GBP in 1992 in the UK (Atkinson, 2014).

Even a disease outbreak in which only domestic animal health is at risk can have a substantial economic impact. From December of 2014 through June of 2015, the USA experienced its largest foreign animal disease outbreak in history. Only avian species were affected in this outbreak of highly pathogenic avian influenza, which spread across three migratory bird flyways and resulted in the death or euthanasia of more than 50 million birds on 232 premises (https://www.aphis.usda.gov/aphis/ourfocus/animalhealth/animal-disease-information/avian-influenza-disease/sa_detections_by_states/hpai-2014-2015-confirmed-detections). Laying hens and turkeys were the predominant agricultural species impacted by the outbreak. Total economic losses associated with the outbreak were estimated to be US$3.3 billion (https://www.aphis.usda.gov/aphis/ourfocus/animalhealth/animal-disease-information/avian-influenza-disease/sa_detections_by_states/hpai-2014-2015-confirmed-detections). In the UK, the 2001 outbreak of FMD was estimated to causes losses of 3.1 billion GBP to the food and agricultural segment alone, with additional losses to tourism that were similar in magnitude (Thompson et al., 2002). Over 10 million cows and sheep

were euthanized to get the outbreak under control. Although human health was not directly impacted by FMD, the outbreak response activities, which included the mass depopulation of livestock, restrictions in human movement, social isolation, and resultant job losses, took a heavy psychological toll on affected communities. As a result, increased rates of psychological morbidity were reported in affected areas, with morbidity rates in farmers correlated with the level of livestock culling and movement restrictions (Peck, 2005). These events highlight the inextricable connections between human, animal, and ecosystem health – demonstrating that events effecting one segment of the triad inevitably impact the others even if indirectly.

1.2.3 Natural Course of Disease

Each case of a disease in a population follows a progression from susceptible to recovery or death (see Figure 1.5). Interactions between the host, the agent, and the environment influence the rate of progression and the end result. *Susceptible* individuals are those at risk for becoming a case of the disease. Exposure to risk factors for the disease or to the infectious agent increases the probability of becoming a case only for those members of the population who are susceptible. Once a susceptible population member is exposed, the disease process may begin. This early phase of the disease often poses the greatest risk to the rest of the susceptible population because clinical signs of illness have not developed and the disease is difficult, if not impossible, to detect. For infectious diseases, this means that infected humans or animals may infect others in the population without showing clinical signs. The length of time from exposure to a disease-causing agent to the onset of clinical signs is referred to as the *incubation period*. Agents have different incubation periods, with some as short as a few minutes, while others may take decades before clinical signs develop (Table 1.1).

Once clinical signs appear, there is the possibility that the disease can be detected and steps taken to intervene and prevent transmission to other susceptible population members. Even if control measures or treatments are not implemented, the simple onset of signs can reduce contacts with noninfected susceptible population members. Animals that are clinically ill often distance themselves from the rest of the herd or flock (Lopes et al., 2016). In human populations, public health policies focused on social distancing have been shown to effectively reduce transmission of infectious disease (Glass et al., 2006).

The final stage of disease is also influenced by host and agent factors. As discussed earlier in this chapter, virulence of the agent influences severity of the illness, degree of disability, and the rate of death among cases. Agent *immunogenicity* reflects the host's ability to develop immunity to the disease upon recovery and the duration of this immunity. These agent characteristics also impact the ease with which effective vaccines can be developed to reduce the number of susceptible individuals in the population. The duration of the period of time from onset of clinical signs to the resolution of any secondary sequelae or long-term disability has a large potential impact on the economic and social costs of the disease.

1.2.3.1 Reservoirs of Disease

So far in this chapter, host factors and agent factors have been the focus of discussion. Where does the environment fit into this triad? Where does the infectious disease agent "live" when it is not infecting a host? In addition to understanding the agent and the susceptible hosts that it infects, breaking the chain of transmission requires understanding where that agent can be found in nature and how the host becomes exposed to it. The *reservoir* of a disease is the habitat in which the agent normally lives, grows, and multiplies (http://www.cdc.gov/ophss/csels/dsepd/ss1978/lesson1/section10.html). Humans, animals, and the environment are potential reservoirs for infectious disease agents and, in some cases, insects serve as vectors transmitting infectious disease agents to new hosts (Table 1.2). Identifying the reservoir and finding measures to control or eradicate