

Network Paper

In brief

- Never before has it been clearer what interventions must be implemented to mitigate the adverse health consequences of wars and natural disasters, and what standards those interventions must strive to achieve; similarly, the range of interventions at our disposal has never been greater.
- Despite these advances, reviews of the global relief system suggest an ongoing failure to deliver. They also highlight the dire lack of credible data to help us understand just how much populations in crisis suffer, and to what extent relief operations are able to relieve that suffering.
- Political considerations often obstruct the delivery of appropriate relief. The premise of this paper, however, is that lack of knowledge is also an important limiting factor. This paper argues the need for advocacy for timely and appropriate relief, grounded in clearly outlined, scientifically sound reasoning, focusing discussion on substantive matters and reducing the scope for political manipulation. As such, the paper aims to enable readers to better face up to the political and bureaucratic aspects of the global relief system.

About HPN

The Humanitarian Practice Network at the Overseas Development Institute is an independent forum where field workers, managers and policymakers in the humanitarian sector share information, analysis and experience. *The views and opinions expressed in HPN's publications do not necessarily state or reflect those of the Humanitarian Policy Group or the Overseas Development Institute.*

Public health in crisis-affected populations

A practical guide for decision-makers

Commissioned and published by the Humanitarian Practice Network at ODI

**Francesco Checchi, Michelle Gayer,
Rebecca Freeman Grais and
Edward J. Mills**



Britain's leading independent
think-tank on international development
and humanitarian issues

Overseas Development Institute
111 Westminster Bridge Road
London SE1 7JD
United Kingdom

Tel. +44 (0) 20 7922 0300
Fax. +44 (0) 20 7922 0399

HPN e-mail: hpn@odi.org.uk
HPN website: www.odihpn.org

Humanitarian Practice Network (HPN)

Overseas Development Institute
111 Westminster Bridge Road
London, SE1 7JD
United Kingdom

Tel: +44 (0)20 7922 0331/74

Fax: +44 (0)20 7922 0399

Email: hpn@odi.org.uk

Website: www.odihpn.org

Printed and bound in the UK

About the authors

Francesco Checchi is an epidemiologist at the London School of Hygiene and Tropical Medicine, where he is an honorary lecturer and part-time PhD student. He has worked for Médecins Sans Frontières (MSF), Epicentre and the World Health Organisation (WHO) in various conflict-affected countries. Most of his work has been on African sleeping sickness, malaria, health survey methods and epidemiology in crisis-affected populations.

Michelle Gayer is a medical officer at the WHO in Geneva, where she develops standards, guidelines and tools, conducts training and provides field assistance on communicable disease surveillance and control in humanitarian emergencies. She has provided technical support to national authorities, UN agencies, NGOs and international organisations in numerous conflict and post-conflict settings and after natural disasters. She also serves on WHO's Ethics Review Committee. Previously she worked in sub-Saharan Africa as a clinician and on various public health projects with MSF.

Rebecca Freeman Grais is an epidemiologist at Epicentre in Paris. Her background is in mathematical epidemiology and its use in informing optimal strategies for disease control. Another of her main research interests is adapting and improving existing methodologies used to conduct field epidemiological studies. She has done research on various topics including measles, avian influenza, malnutrition and rapid population estimation.

Edward Mills is a clinical epidemiologist with additional training in international human rights law. He has broad interests in HIV/AIDS, the systematic review of evidence and health and human rights. He currently directs the Centre for International Health and Human Rights Studies at Simon Fraser University, and is a scientist at the British Columbia Centre for Excellence in HIV/AIDS.

Acknowledgements

The authors would like to thank the Department of Foreign Affairs and International Trade (DFAIT), government of Canada, and particularly Valerie Percival, for having generously supported the writing of this paper. We are also grateful to the Humanitarian Practice Network, particularly Rachel Atkinson, for advice and help with this publication, and Matthew Foley, for editorial work.

The following people contributed to the development of the paper during a meeting in Ottawa, Canada, on 8 March 2007: Aranka Anema (British Columbia Centre for Excellence in HIV/AIDS), Chris Beyrer (Johns Hopkins University Bloomberg School of Public Health), Michael Bonser (DFAIT), Rob Chase (University of Manitoba), Stephanie Chong (University of Toronto), Curtis Cooper (Ottawa Hospital), Richard Garfield (Columbia University), P. Gregg Greenough (Harvard Humanitarian Initiative), Colleen Hardy (International Rescue Committee), Andrew Mack (Human Security Centre, University of British Columbia), David Meddings (World Health Organization), Alexandra Mihailovic (University of Toronto), James Orbinski (University of Toronto), Stephen Salewicz (Canadian International Development Agency), Michael Schull (University of Toronto), Sonal Singh (Wake Forest University), Sumeet Sodhi (Dignitas International), Annie Sparrow (Catholic Relief Services), Paul Spiegel (United Nations High Commissioner for Refugees), Christine Tapp (Simon Fraser University), Brad Woodruff (US Centers for Disease Control and Prevention). Many thanks also to Jacqui Tong for her helpful suggestions.

ISBN: 978 0 85003 855 2

Price per copy: £4.00 (excluding postage and packing).

© Overseas Development Institute, London, 2007.

Photocopies of all or part of this publication may be made providing that the source is acknowledged. Requests for the commercial reproduction of HPN material should be directed to the ODI as copyright holders. The Network Coordinator would appreciate receiving details of the use of any of this material in training, research or programme design, implementation or evaluation.

Contents

Chapter 1 Introduction	1
The importance of knowledge in humanitarianism	1
Scope of this paper	1
Chapter 2 A crash course in epidemiology	3
A definition of epidemiology	3
Epidemiological properties of infectious diseases	3
Incubation period, duration of infection, and serial interval	5
Epidemiological properties of non-infectious diseases	13
Notions of risk	14
Causality and confounding	15
Chapter 3 The effects of crises on health	17
A simplified general model of disease in the population	17
Excess morbidity and mortality	18
Notes on specific risk factors	20
Notes on some important direct causes of death	26
HIV, measles and malnutrition: killers by proxy	27
Epidemics versus endemic diseases	29
Mental illness: overlooked but omnipresent	33
Chapter 4 Determining priority health problems and interventions	35
Which health problems?	35
Which interventions?	37
Chapter 5 Maximising impact	41
A framework for interventions	41
Coverage versus effectiveness	44
Monitoring and evaluation	45
Chapter 6 Conclusion	49
Review of key principles	49
Ten common fallacies	49
Glossary	51

Chapter 1

Introduction

'The endeavour to understand is the first and only basis of virtue.'

Baruch de Spinoza, *Ethics*

The importance of knowledge in humanitarianism

A considerable proportion of humanity is currently living in crisis conditions. As of end-2006, 8 million were refugees and 25m internally displaced; about one billion lived in 40 'fragile states'. Between 2000 and 2005, a yearly average of 398 country-level natural disasters was reported, affecting almost 300m people annually.

Never before has it been clearer what interventions must be implemented to mitigate the adverse health consequences of wars and natural disasters, and what standards those interventions must strive to achieve; similarly, the range of interventions at our disposal has never been greater. Over the last decade, collective knowledge on the public health aspects of emergencies has resulted in seminal publications, including guidelines such as *Médecins Sans Frontières' Refugee Health*, the World Health Organisation's *Communicable Disease Control in Emergencies* manual, and the Sphere Guidelines.¹ Despite these advances, recent reviews of the global relief system suggest an ongoing failure to deliver.² They also highlight the dire lack of credible data to help us understand just how much populations in crisis suffer, and to what extent relief operations are able to relieve that suffering.

Political considerations often obstruct the delivery of appropriate relief. Our premise for this paper, however, is that lack of knowledge is also an important limiting factor. Field practice may often be unsatisfactory because of 'muddling through', taking the safest decision given the knowledge available, but failing to consider hidden aspects of the problem from an overarching perspective, using a combination of background knowledge, quantitative analysis, logic and common sense.

We believe in advocacy for timely and appropriate relief, grounded in clearly outlined, scientifically sound reasoning, thus focusing discussion on substantive matters and reducing the scope for political manipulation. In this respect, we hope that this paper will enable readers to better face up to the political and bureaucratic aspects of the global relief system.

Scope of this paper

This paper attempts to present a bird's eye view of the risks to health inherent in crises, and the potential for impact of health interventions, using the language of epidemiology.

Which crises?

We restrict ourselves to the following five crisis conditions, brought about by war and/or natural disasters:

- **Condition 1:** Progressive loss of livelihoods and deterioration of essential services, with entrapment in one's community due to the ever-present risk of violence. Examples of this condition could be the eastern Democratic Republic of Congo (DRC), eastern Chad, regions of Nepal affected by the Maoist insurgency, western Côte d'Ivoire and Iraq.
- **Condition 2:** Mass displacement into regimented or camp-like settlements of large population size. This is the classic relief scenario (think of IDP camps in Darfur and northern Uganda, or refugee camps on the Thai–Burma border).
- **Condition 3:** Displacement into neighbouring host communities. Examples include Lebanese IDPs during the 2006 Israel–Hezbollah war, and Sri Lankans displaced by recent fighting. This displacement may be directly due to violence, but could also occur indirectly due to loss of livelihoods and social pressures: Burma is a prime example of this.
- **Condition 4:** Sudden loss of livelihoods and rapid environmental change (including flooding) due to a natural disaster. Major examples are the Indian Ocean tsunami of 2004 and the Pakistan earthquake of 2005.
- **Condition 5:** Food crises. Examples include Niger in 2005–2006 and the famine in the Somali region of Ethiopia in 2000.

The above conditions are not exclusive. Combinations are common: the term complex emergency indicates precisely this. For example, during the 1990s, Bahr el Ghazal, southern Sudan, simultaneously experienced militia attacks, camp displacement, flooding and famine. The acute, post-acute and post-emergency or recovery phases of a crisis are difficult to delineate, especially in protracted cases. Here we consider simply that, as long as people are exposed to an increased risk of disease and death because of any of the above conditions, a crisis is occurring, and fits within our scope.

The paper does not cover the following topics:

- Mass-casualty incidents and their management, e.g. following earthquakes, landslides and terrorist attacks.
- Biological, chemical and nuclear attacks or accidents.
- Consequences of armed conflict on combatants themselves (i.e. military medicine).

Which health issues?

Figure 1 presents a simple framework of the possible health effects of crises. Deaths and injuries sustained in battle or during a natural disaster are conventionally

referred to as direct health effects. Deaths and disease resulting from an increase in the risk of infectious and non-infectious diseases (including unintentional injuries) attributable to the presence of a crisis are referred to as indirect effects. With the exception of natural disasters, the indirect effects of crises on population health usually far exceed direct ones.³

Although we will discuss injury and non-infectious diseases, we focus on infectious diseases, for two reasons. First, the vast majority of preventable indirect deaths are related to infectious rather than non-infectious diseases. Second, crises increase the risk of infectious diseases by directly interfering with their transmission, progression and lethality. In short, infectious diseases present more challenges and opportunities for control than non-infectious ones. One extremely important exception to this is mental illness, a much-overlooked aspect of health relief operations, which we will briefly discuss. Sexual violence is another extremely important health issue, and one which clearly interacts with both physical and mental illness. However, we feel that we cannot do justice to this subject here.⁴

This paper discusses leading causes of morbidity (disease) and mortality (death) in crises, but stops short of charting every possible pathway by which crises worsen health. Furthermore, we focus on ‘proximate’ and ‘intermediate’ risk factors (defined later), rather than general underlying issues such as insecurity, ethnic strife, poverty and gender relations, which are usually beyond the scope of relief.

Intended audience

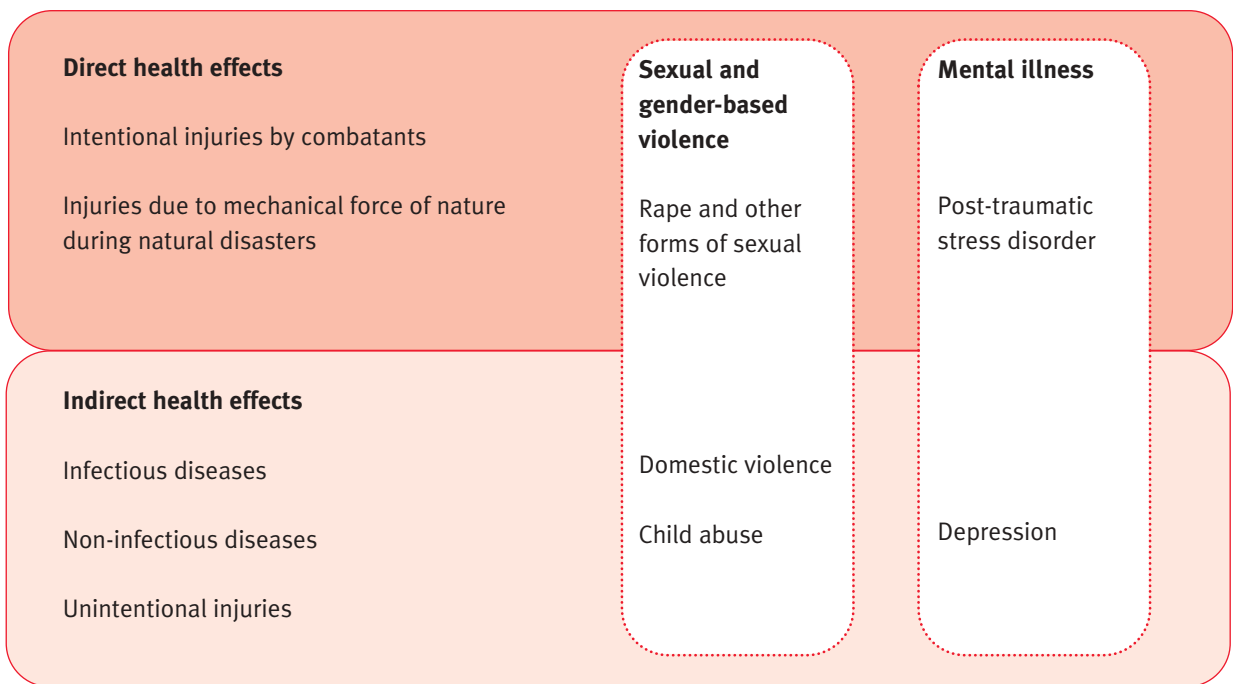
This paper is meant for a non-technical audience without prior training in health or field experience. While recognising the complex interplay of factors that, on a global level, results in greater or lesser interest in and allocation of resources for specific crises, in this paper we situate ourselves at the level of field interventions. Accordingly, the primary target audience consists of professionals from non-governmental organisations, United Nations agencies, donor agencies and host governments involved either in policy-setting, coordination and financing relief operations at the crisis-wide level, or in field implementation of specific projects. Senior-level programme officers at international level, advocacy groups, students and journalists might also find the paper a useful resource.

Structure

After presenting key epidemiological concepts used throughout the paper (Chapter 2), we explore a framework linking types of crisis conditions to certain risk factors, and to a consequent increase in risk of transmission of or exposure to disease, disease progression and death (Chapter 3). In Chapter 4 we discuss the prioritisation of health problems and interventions. Chapter 5 illustrates the main determinants of an intervention’s impact, and discusses monitoring and evaluation. We conclude (Chapter 6) with a summary of key points and a reminder of typical epidemiological fallacies in relief operations.

Figure 1

Schematic of direct and indirect health consequences of crises



Chapter 2

A crash course in epidemiology

A definition of epidemiology

Epidemiology is the study of the distribution of diseases in the community, and of the factors affecting their frequency. Consider the following statements:

- 1) 'In February 2007, 46% of children living in camp A were infected with intestinal worms.'
- 2) 'Among displaced villagers living on the hillsides of district B, clinic-based surveillance of a malaria epidemic occurring between August and November 2006 showed that malaria accounted for 55%, 46% and 18% of all outpatient consultations occurring at altitude <1,500m, 1,500–2,000m and >2,000m, respectively; the corresponding proportion of severe cases was 8%, 14% and 32%; the average age of cases was 8 years, 13 years and 17 years, respectively.'
- 3) 'In December 2006, a mental health assessment in country C found that the proportion of people 15 years or older with signs of post-traumatic stress disorder was 36% in a randomly selected sample drawn from villages affected by fighting between rebel and government forces in the previous 12 months; the same proportion in a random sample of similar age and sex make-up, but drawn from villages not affected by conflict, was 11%.'
- 4) 'An intervention study implemented between June and December 2006 in camp D found that the rate of occurrence of burn-related trauma among children under 5 years of age living in refugee households provided with adequate cooking stoves at the beginning of the study was 3.7 times lower than the corresponding rate among children living in households that had not received a stove.'

Statement 1 merely describes the frequency of disease in a given population group, at a specified time. Statement 2 breaks down the distribution, and two of the characteristics of cases (age and severity), by a given factor (in this case, altitude). Statement 3 compares the frequency in comparable groups exposed to different circumstances, highlighting a factor (in this case, conflict) potentially associated with this frequency. Statement 4 is a typical result from a randomised trial, in which an intervention (household stoves) is tested, and an outcome (rate of childhood burns) is compared between those that received stoves and those that did not.

Epidemiology can tackle any cause of ill-health, from infections to chronic diseases, injury and mental illness. Whereas doctors examine and administer cures to individual patients, epidemiologists evaluate the health situation of entire communities or populations, and develop mass treatments in the form of public health interventions.

Epidemiological concepts are based on logic and common sense, and are thus accessible to all. Epidemiology is

somewhat like a language, with a few syntax rules. The most important rule when making any epidemiological statement is to always refer implicitly or explicitly to a specific time period, group of persons, and place or context. Such time-person-place reference is indispensable. Statements 1, 2, 3 and 4 above all contain these unequivocal references.

Different epidemiological quantities or indicators are expressed as ratios, proportions or rates. A ratio is simply quantity A over quantity B, where A is not part of B, and vice versa (e.g. male to female ratio; hospital bed per inhabitant ratio). A proportion is quantity A over quantity N, where A is a portion of N (e.g. proportion of all pregnant women who are HIV-positive); a percentage is also a proportion, expressed as per hundred (e.g. percent who have a food registration card). Finally, a rate expresses the speed with which new events occur, per unit population and per unit time. Unfortunately, some key epidemiological indicators have been mistakenly called rates or ratios when in fact they are proportions. This incorrect terminology is too widely accepted for us to adopt a different one here.

Epidemiological properties of infectious diseases

Route of transmission

Epidemiologists use the terms *communicable* and *infectious* diseases interchangeably. Here we adopt the latter, since we believe it is more specific: for example, certain bacteria, such as tetanus, are undoubtedly infectious to man, but exist in nature and can be acquired from accidental wounds without being communicated to and from other humans. Also, some genetic diseases can be communicated from parent to child, but are not infectious.

Infection can be due to pathogens, which include (from smallest to largest) prions, viruses, bacteria, fungi and various parasites ranging from one-cell protozoa such as the malaria parasite *Plasmodium* spp. to multi-cellular, macroscopic organisms such as intestinal tapeworms. Infectious diseases have different routes of transmission, and indeed this is a good characteristic by which to classify them (Table 1), since it also determines what interventions can prevent them.

Endemic versus epidemic diseases

Some infectious diseases occur year-round in a community, whether sporadically or frequently. Their transmission may feature seasonal peaks, but, over a timescale of years, it nonetheless remains within an expected range. These diseases are said to be *endemic* to a given community.

Other diseases are usually absent, but can be introduced suddenly. Alternatively, diseases may be present year-

Table 1: Main routes of transmission of infectious diseases, and main diseases of relevance to crises transmitted through each

Transmission route	Main diseases	How transmission occurs
Air droplet (i.e. pathogens are breathed, sneezed or coughed out of the respiratory system of the infected person, and enter the respiratory system of another)	Tuberculosis Measles Whooping cough Most other respiratory diseases, including those caused by: Common flu/cold viruses <i>Streptococcus pneumoniae</i> <i>Haemophilus influenzae B</i> Pandemic influenza Meningitis Trachoma ^W	Inhalation of or eye contact with droplets containing pathogens as a result of close interaction with infectious person Especially likely if infectious person sneezes or coughs
Faecal-oral (i.e. pathogens are excreted from the gut of an infected person, and enter the gut of another person through his/her mouth)	Diarrhoeal diseases, including: Cholera ^W Shigella ^W (bacterial dysentery) Salmonella ^W <i>Escherichia coli</i> ^W Rotavirus ^W Amoebiasis ^W Giardiasis ^W Typhoid ^W Most intestinal worms ^W Hepatitis A ^W Hepatitis E ^W Polio ^W	Ingestion of faecal matter (see Chapter 3: Poor water, sanitation and hygiene conditions)
Sexual (i.e. pathogens are transferred from the blood and fluids of an infected person to his/her sexual partner during intercourse)	HIV Syphilis Chlamydia Gonorrhoea Hepatitis B	Unprotected sex (anal sex particularly hazardous)
Vector-borne (i.e. pathogens undergo a life cycle inside humans as well as inside another 'vector' species, usually insects: they need both life cycles to sustain themselves, and are most commonly transmitted from the vector to the human and back to the vector via insect bites)	Malaria ^W Dengue fever ^W Japanese encephalitis African sleeping sickness Leishmaniasis/kala azar River blindness Schistosomiasis ^W Typhus ^W Relapsing fever	Mosquito bite (night-biting) Mosquito bite (day-biting) Mosquito bite (day-biting) Tsetse fly bite Sand fly bite Black fly bite Fresh-water snail Bites of lice, fleas, mites Bites of lice and ticks
Blood (i.e. pathogens are directly transferred from the infected person's blood to another person's)	HIV Hepatitis C Hepatitis B	Unsafe injections Transfusions with unsafe blood
Unclean wound (i.e. pathogens exist in nature and enter the body through a wound)	Tetanus	Deep cuts Infection of umbilical cord after birth
Mother to child (vertical) (i.e. pathogens are transmitted by the mother to her newborn baby)	HIV Hepatitis B Syphilis	During childbirth Breast milk

W indicates diseases heavily dependent on water, sanitation and hygiene conditions.

Table 2: Examples of outbreak/epidemic thresholds for selected infectious diseases

Disease	Context	Outbreak/epidemic threshold
Measles	Camp	1 case
Cholera	Overcrowded community	1 case
	Rural community	Significant increase from expected
Meningococcal meningitis	Community of <30,000 people	5 cases in 1 week <i>or</i> doubling of cases in 3-week period <i>or</i> decision on a case-by-case basis
	Community of $\geq 30,000$ people	10 cases per 100,000 people per week if no epidemic in last 3 years and vaccination coverage <80% <i>or</i> alert threshold crossed early in dry season; otherwise, 15 cases per 100,000 people per week

round, but suddenly reach levels far above the expected. Such situations are known as *outbreaks or epidemics* (the term outbreak generally indicates a smaller-size event), and diseases that can bring about such phenomena (not all can) are considered *epidemic-prone*. The definition of what constitutes an epidemic is often based on an arbitrary and context-specific threshold (i.e. a level of disease occurrence which, if exceeded, triggers the declaration of an epidemic; see Table 2 for some examples) defined based on research or policy considerations.

The distinction between endemic and epidemic is not always clear-cut, and some diseases, like malaria, can be both depending on the context. However, the two terms are mostly useful for control purposes rather than classification: for epidemic-prone diseases, the emphasis must

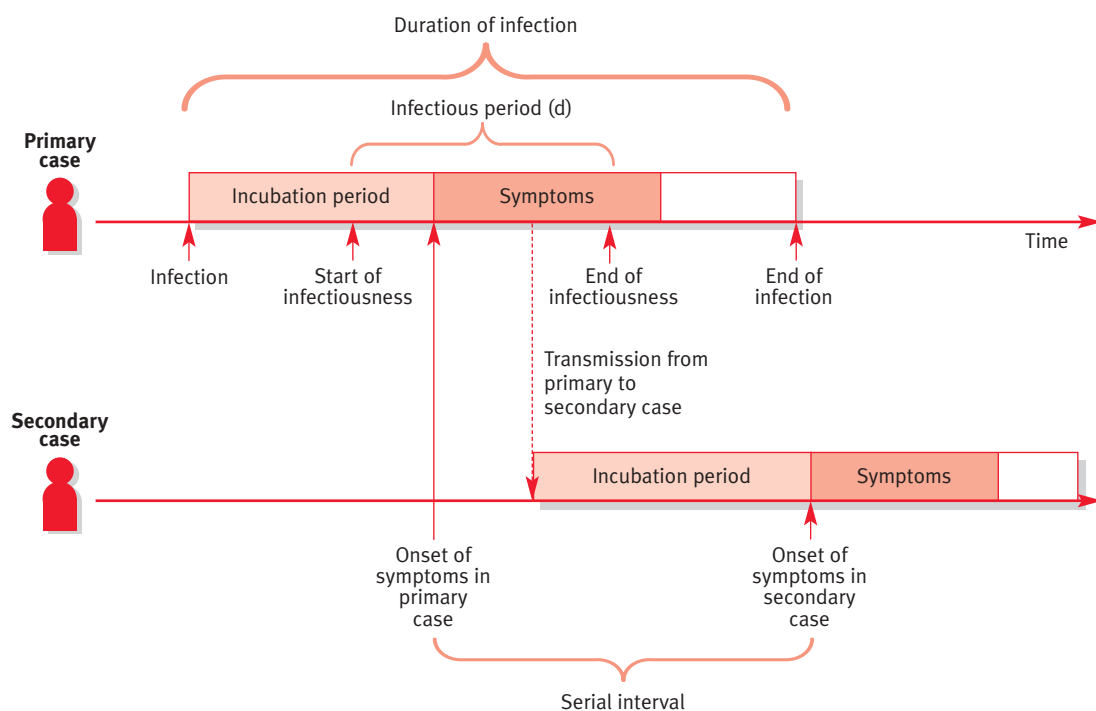
be on preparedness, early warning and surveillance, and outbreak prevention. For endemic diseases, control activities must be maintained on an ongoing basis.

Incubation period, duration of infection, and serial interval

The time elapsing between infection (i.e. when the pathogen establishes itself in the body) and the appearance of and symptoms of the disease is known as the *incubation period*, which can be hours for diarrhoeal diseases, two weeks for malaria, and years for tuberculosis and HIV/AIDS. There is always individual variability, and for each disease we can speak of an average incubation period, and of a typical range. Epidemiologists are also interested in the average *duration of infection*, i.e. from time of infection to its final outcome,

Figure 2

Illustration of incubation period, duration of infection and serial interval



be it spontaneous cure, successful treatment or death of the individual. Some infectious diseases, like herpes, can lead to chronic infections that may last a lifetime.

The *infectious period* is usually less than the duration of infection: an infected person may be infectious (i.e. able to transmit the infection) during part of the incubation period and part of the symptomatic period.

A related quantity is the serial *interval*, namely the average amount of time between a 'primary' case and the 'secondary' cases it causes. For example, a child with measles transmits the virus to a second child, and the second to a third. The time elapsed between the first child's onset of symptoms and the second child's onset of symptoms, or that between the second and the third's, is the serial interval. Many pathogens are not very infectious during the incubation period: thus, the incubation period heavily influences the serial interval. Epidemic diseases with a short serial interval (e.g. measles, diarrhoeal diseases, meningitis) will exhibit a much more explosive growth in the community (since it takes less time for transmissions to occur), thus leaving little time for reaction. Figure 2 represents these quantities graphically. Table 3 provides examples for several important epidemic-prone diseases in emergencies.

Disease transmission in quantitative terms: the reproductive ratio

While a person remains infectious, (s)he is able to transmit the pathogen and thus cause additional infections. But just how quickly and extensively will a disease spread in the community? Predicting this is possible if one knows, on average, how many additional infections will result from any given case. This crucial quantity, known as the *reproductive ratio or reproductive number* (R), has wide-ranging implications for disease control. Consider the following:

- 1) In an IDP camp previously cholera-free, three cholera cases appear. In this first phase of the outbreak, each 'primary' case of cholera results in ten further 'secondary' cases ($R=10$). This situation will lead to an explosive, exponentially growing epidemic (the first three cases will result in 30 more, which will result in 300 more, and so on).
- 2) In a shantytown, tuberculosis is endemic and frequent. Each case leads to about one additional case ($R=1$). The disease will remain at roughly the same levels unless something is done: this is a classic endemic scenario.
- 3) In a village there are 20 cases of river blindness (onchocerciasis), but a control programme starts treating all villagers once a year with ivermectin (a drug that clears the worms responsible for the disease); thanks to this, on average each case only gives rise to 0.5 new cases ($R=0.5$); thus, out of the 20 cases only ten new cases will arise; if control is sustained, the third generation will only consist of five, and so forth until the disease dies out.

Briefly, if R is around 1, the disease will be endemic and stable. If $R > 1$, caseload will increase, as at the beginning

Equation 1

Components of the reproductive ratio of an infectious disease

$$R = c \times p \times d$$

where:

c = average number of susceptible people (i.e. who are not infected and can contract the infection) an infectious person comes into contact with, per unit time (e.g. per day)

p = average probability that transmission will take place, per unit contact

d = average duration of infectiousness, or infectious period (e.g. in days)

Note that these symbols do not reflect any standard notation.

Any increase in **c**, **p** or **d** will result in an increase in **R**.

of an epidemic; if $R < 1$, the disease will usually go extinct: interventions to eliminate or eradicate a disease work by reducing R to below 1.

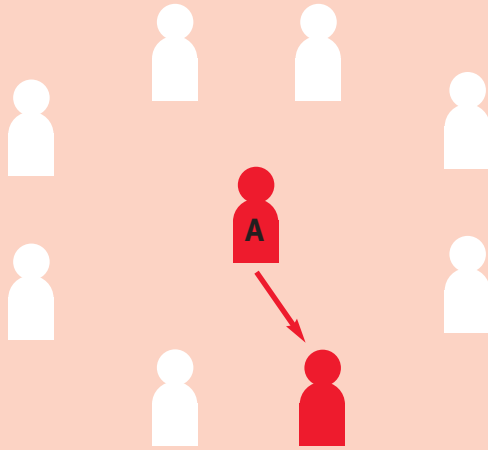
R for the same disease varies widely across communities and/or over time. This variation results from the components that make up R , namely (i) the degree to which infectious people come into contact with those who are susceptible; (ii) the transmissibility of the pathogen, i.e. how easily it passes from one person to the next; and (iii) the amount of time an infectious person has to spread the infection, before (s)he is treated, dies or has cleared the infection spontaneously (see Equation 1 and example in Figure 3).

Logically, 'c' depends on both context and transmission route. In an overcrowded camp where people are living in close quarters, a flu patient will contact many healthy, susceptible people. In a dispersed rural village, opportunities for contact will be considerably fewer. Similarly, 'c' for malaria, a vector-borne disease, depends on the intensity of mosquito bites (e.g. more near a swamp, less in a grassland); and 'c' for sexually transmitted diseases depends on behaviour (e.g. frequency of unprotected intercourse). The presence of susceptible people drives 'c'. As discussed later, interventions that reduce susceptibility, such as vaccination or bed nets, dramatically reduce 'c', and thus R . In short, 'c' depends greatly on what people do, how they live and how susceptible they are.

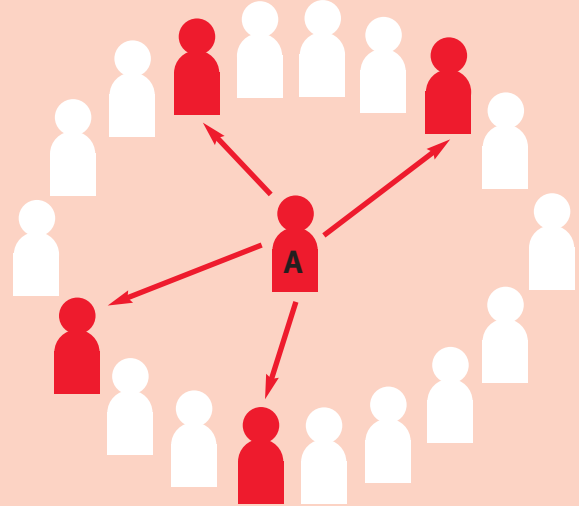
By contrast, 'p' depends mostly on the biology of the pathogen: how contagious it is. Sometimes, it can be behaviour-related: for example, the 'p' of HIV is about five out of 1,000 episodes of unprotected intercourse if sex is anal, and one in 1,000 if it is vaginal.

Figure 3**Illustration of the reproductive ratio (R) and its components (c, p and d)***Example: A patient (A) with meningitis who remains infectious for 4 days*

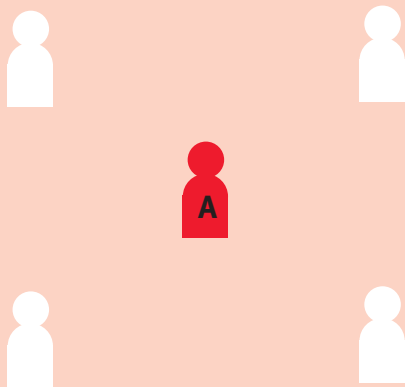
Day 1 (first day of infectiousness): A still feels well; he comes into close contact with 8 susceptible people, of whom he infects 1.



Day 2: A is still well and attends a gathering, where he comes into contact with 18 susceptibles, of whom he infects 4.



Day 3: A feels sick and restricts his movement. He comes into contact with 4 susceptible people, and infects 0.



Day 4 (last day of infectiousness): A is bed-ridden, only coming into contact with 2 susceptible relatives, of whom he infects 1. He is then hospitalised and treated, following which he is no longer infectious.



Overall, A has come into close contact with 32 people, or an average of 8 per day ($c = 8$). Out of these, he has infected 6 ($p = 6/32 = 0.1875$). He has been infectious for 4 days ($d = 4$). Thus, $R = c \times p \times d = 8 \times 0.1875 \times 4 = 6$. We know R must be 6, because A caused 6 secondary infections.

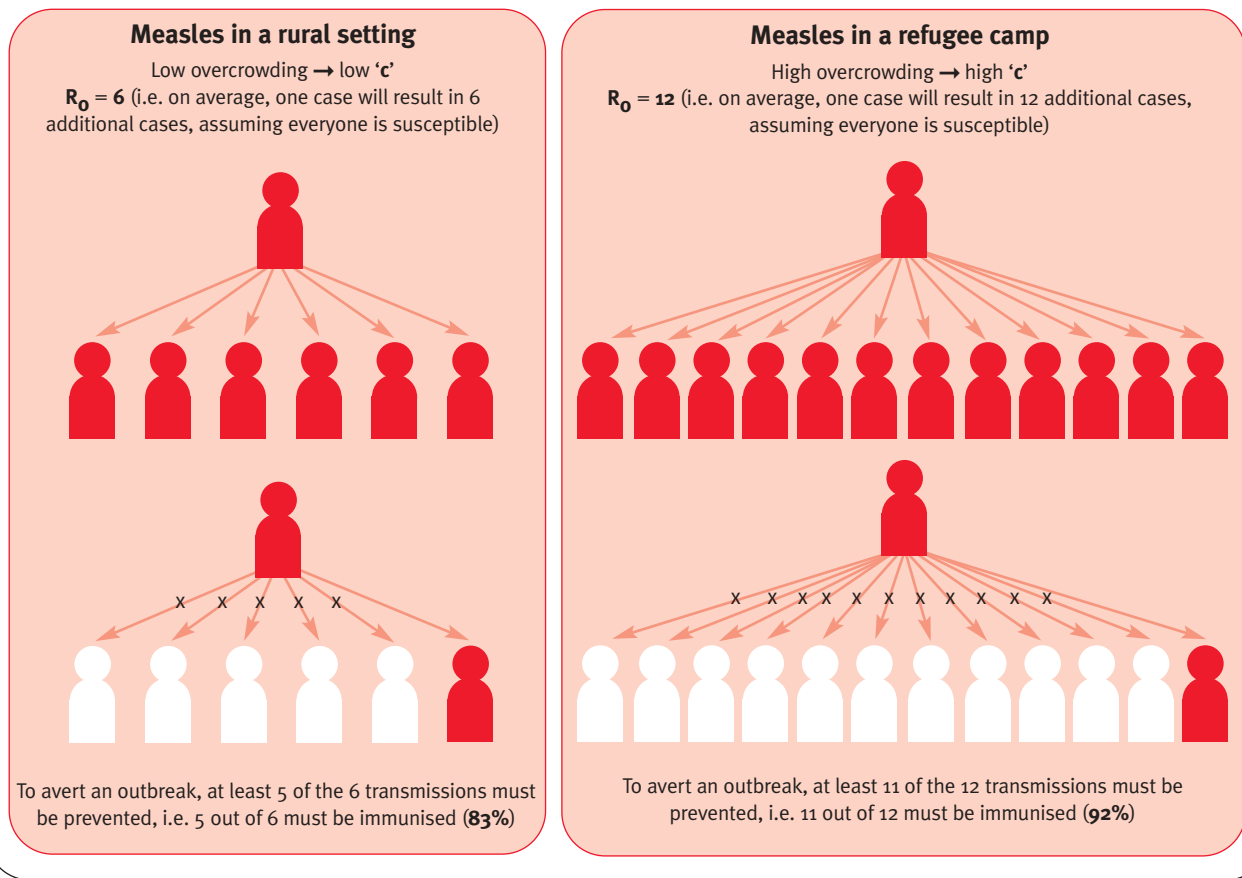
Finally, 'd' determines, along with 'c', how many opportunities for transmission an infectious case has. For example, although the 'p' of HIV sexual transmission is low, asymptomatic HIV infection lasts for years, during which someone may have hundreds of unprotected sexual contacts. Treatment can reduce the natural 'd'. Furthermore, a person's immune response can also change 'd' (e.g. by spontaneously overcoming the infection).

R is greatest when everyone is susceptible and there is no treatment or control. The R under such conditions, which

usually occur only at the very outset of an epidemic, is known as the *basic reproductive ratio* or R_0 . We can now explain the classic shape of epidemic curves: at the outset, almost everyone is susceptible, R approaches R_0 , and the epidemic expands exponentially. As the epidemic progresses, an increasing proportion of the population is infected, may develop immunity, and thus is no longer susceptible: eventually, there are so few susceptibles left that R first plateaus and then declines below 1, thus resulting in the epidemic's demise, even without an intervention. Due to this decline, some (or many) will

Figure 4

The basic reproductive ratio: an example of vaccination to prevent measles outbreaks



escape infection. Interventions are crucial to minimise or prevent epidemics; however, when implemented late they may have limited impact: an intervention implemented mid-way through an epidemic may in fact not be responsible for its decline, although agencies eager to demonstrate their programme's impact may claim this.

Crucially, R_0 determines requirements for disease control. To prevent an outbreak, we must reduce R below 1: this requires protecting a given proportion of the population, for example through vaccination (see Figure 4). However, in a refugee camp, where 'c' is higher due to overcrowding, the measles R_0 is about twice that in a rural setting. Thus, a measles case introduced in a camp will cause about 12 more, virtually ensuring that an outbreak will take off. Preventing an outbreak requires ensuring that more than 11 out of 12 children are immunised (through vaccination or previous exposure to the virus), so that any imported case will not be able to reproduce ($R < 1$). This explains why near-100% measles vaccination is necessary in camps, and why the proportion that needs to be vaccinated changes according to the setting.

R_0 also determines why some diseases are very difficult to completely eliminate, which would require maintaining $R < 1$ indefinitely. For example, in mosquito-infested Sierra Leone, the R_0 of malaria is in the range 100 to 1,000. Any intervention would thus have to reduce R by 99 to 999

units, i.e. protect more than 99.0% to 99.9% of the population, an extremely arduous task.

Incidence and attack rates

Incidence is the occurrence of new events: new infections, cases of disease or deaths (mortality is simply the 'incidence of death'). However, in this paper incidence will only refer to the occurrence of new cases of disease.

Imagine a camp where suddenly a child infected with the whooping cough bacterium arrives, and that the whooping cough $R=5$. If the serial interval is ten days on average, assuming every infected child becomes sick, the incidence of whooping cough over the following 30-day period (i.e. three serial intervals) will be $5 \times 5 \times 5 = 125$ new cases. If $R=3$, incidence = $3 \times 3 \times 3 = 27$. If the serial interval were 15 days, only $3 \times 3 = 9$ cases would occur (since 30 days would encompass only two serial intervals). If only $2/3$ of infected children developed symptoms, incidence would be $9 \times (2/3) = 6$. Thus, incidence is simply the mathematical consequence of reproductive ratio, serial interval and the infection's ability to cause disease (sometimes called *virulence*). This is a key relationship.

In absolute terms, 125 new cases per month is much more than six – but is it a lot or a little? Is it so unusual we could call it an epidemic? If a neighbouring camp experienced

Table 3: Epidemiologic characteristics of the main epidemic-prone diseases in emergencies: basic reproductive ratio, incubation period, serial interval, case-fatality ratio

Disease	R ₀	Incubation period (days)	Serial interval (days)	CFR if untreated
Cholera	4–15	2–3	7–10	up to 50%
Shigella (bacterial dysentery)	unknown	1–3	unknown (a few days)	up to 10%
Malaria	Low-transmission areas: ~1–10 High-transmission areas: ~100–1,000	9–13	~60–120	30–50% of severe episodes ~1% of all episodes in non-immunes
Measles	Rural: 5–6 Urban or crowded: >12	10–12	~15	3–5% (developing countries) 10–30% (displaced populations)
Meningococcal meningitis	unknown	3–4	unknown (a few days)	up to 50%
Pandemic influenza (based on 1918 Spanish flu)	3	2	unknown (a few days)	2% (depends on age and previous exposure to related strain)

Box 1**How to compute incidence rates**

Step 1. Decide on a **time unit**: should one monitor incidence on a daily, weekly, or monthly basis? In a fast-evolving epidemic, daily or weekly calculations are needed; for endemic diseases, monthly incidence is sufficient.

Step 2. Decide **who is at risk** for the disease in question: is it the entire population or only a sub-group (e.g. children)?

Step 3. Find the best **population estimates** available for the group at risk. Consider whether they could be over- or under-estimates.

Step 4. Find the most comprehensive **data on number of new cases** among the group at risk, broken up by the chosen time unit. Usually, these data will only be available from health facilities. Consider the limitations of these data: health facilities will usually reflect only a fraction of total cases occurring in the community. However, the main function of incidence rates is to monitor trends: health facility data are usually sufficient for this. Also be aware of data quality issues, and how cases were diagnosed: if different facilities use different diagnoses, it is best to analyse them separately. If a data source seems very unreliable, exclude it.

Step 5. **Divide** the number of new cases by the population estimate, for each time unit.

Step 6. Decide on a **multiplier** (ex. per 100/1000/100 000 people), based on the data themselves (avoid unwieldy decimals: see example below).

Step 7. **Multiply** the result of Step 5 by the chosen multiplier.

Example. The rainy season began one month ago. To detect a possible malaria outbreak as early as possible, any rising trends in the malaria incidence rate need to be observed. Malaria epidemics evolve rapidly: weekly incidence calculations are needed. The community consists of IDPs from a non-malarious region, so everyone can be assumed to be non-immune and thus at risk. The best estimate of the population is 23 000. There is one hospital, where all malaria cases are confirmed via rapid blood test: this seems a good data source. This week, 112 new malaria cases were recorded at the hospital outpatient department. $112/23\,000 = 0.0049$. Choosing a multiplier of 1000 (i.e. ‘per 1000 people’), incidence rate = **4.9 cases per 1000 people per week**.

250 cases over the same period, could we say the epidemic is spreading less quickly in ours? To answer these questions, we must relate incidence to the size of the population, i.e. calculate an *incidence rate*. This rate expresses the number of new cases of disease per unit number of people (e.g. per person; per 1,000 persons) per unit period of time (e.g. per week; per year). Box 1 shows how to calculate incidence rates.

Incidence rates are ubiquitous in infectious disease epidemiology, and the ‘currency’ of disease surveillance. Whenever possible, they are calculated not among the general population, but among the *population at risk*, which, depending on the disease, may or may not be everyone (e.g. we might express the incidence rate of neonatal tetanus as the number of cases per 1,000 children under 30 days old per year).

Box 2

Example of incidence rate calculation, and its common pitfalls

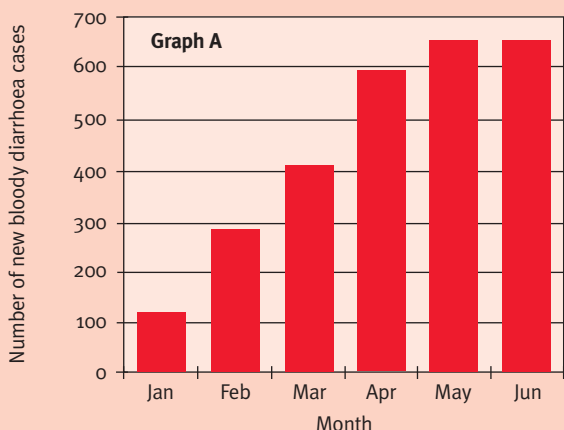
Imagine a group of nomadic settlements experiencing a food crisis. Many families have congregated in the settlements recently, attracted by food distributions. Three government primary health care centres are operational: they charge user fees and, starting in May, began to experience drug shortages. In June, a health team from the

capital, responding to a late epidemic alert, wishes to use these clinics' data to calculate the incidence rate of bloody diarrhoea. The team also has population estimates from food registrations, as well as population figures provided by community chiefs.

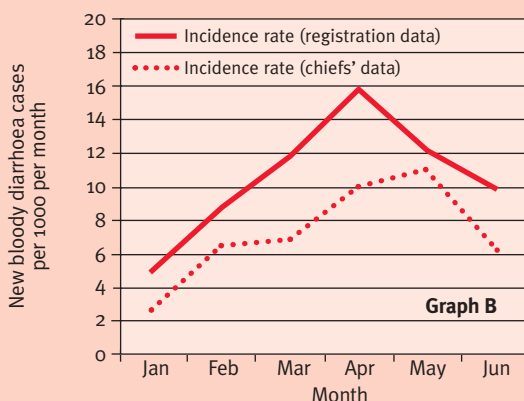
The question: has a bloody diarrhoea epidemic been occurring? Available data is below.

	Jan	Feb	Mar	Apr	May	Jun
Bloody diarrhoea cases	121	288	413	598	655	661
Clinic consultations for any cause	4672	5412	5389	5211	2417	1914
Population (food registrations)	25 000	33 000	35 000	38 000	54 000	67 000
Population (community chiefs)	45 000		60 000			105 000

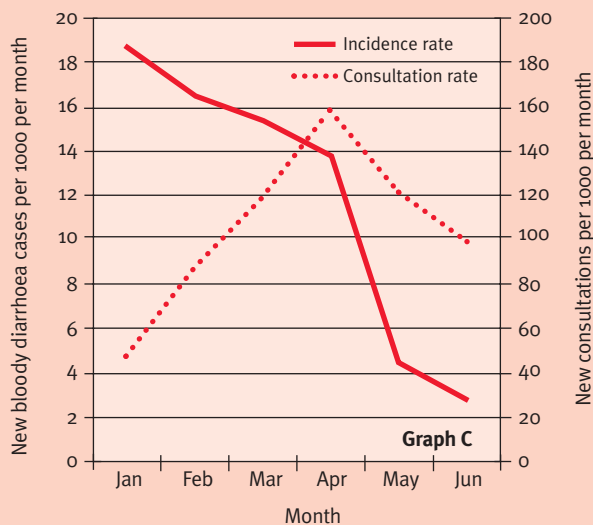
Simply looking at the raw number of cases per month (Graph A), we might conclude there is an epidemic, peaking about now.



If we compute the incidence rate using the population data from registrations (Graph B), the resulting curve suggests the epidemic peak was in April. Note the difference with Graph A, due to changing population size.



If we instead use the chiefs' population data, we get a rather different picture.



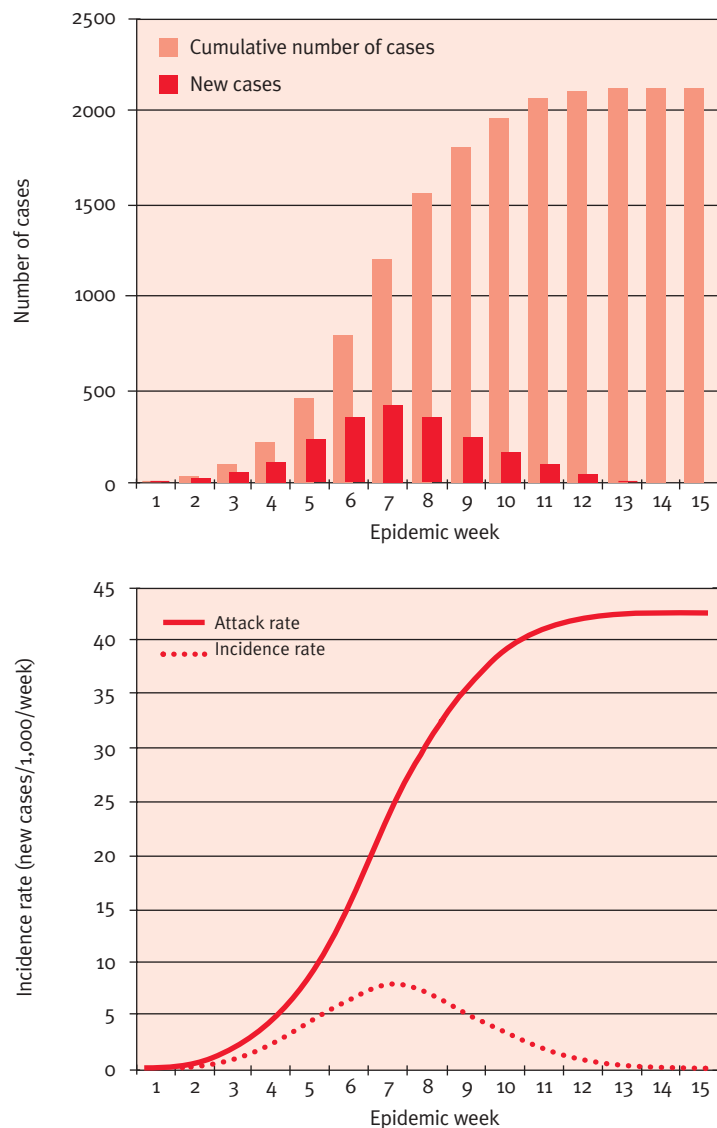
But is the epidemic really declining? If we overlay the incidence rate and the clinic consultation rate (calculated in the same way as incidence; Graph C), we see that clinic attendance slowly declined until April (perhaps due to increasing inability to afford the fees), and plummeted in May (probably due to drug shortages).

This observation casts doubt on whether one can safely assume that the epidemic is declining: if less people are attending the clinic, fewer cases will be reported!

Conclusion. Different analysis of the same data leads to different conclusions about disease trends, and potentially wrong operational decisions.

Figure 5

Graphs of new and cumulative cases by week (top) and incidence and attack rate by week (bottom) for the same hypothetical epidemic



Cumulative cases per week are obtained by summing all incident cases up to and including the given week.

The epidemic peaks on week 7. Accordingly, cases eventually plateau as the epidemic comes to an end.

This graph however only shows raw figures, and does not reveal the extent to which the population was affected by the epidemic.

This graph shows the weekly incidence rate (as new cases per 1,000 people per week), obtained by dividing the number of new cases in the given week by 50,000 and multiplying the result by 1,000, and corresponding attack rate.

The pattern is identical to above, but now we can say that, by the end of the epidemic (week 15), about 43 people out of 1,000 (or 4.3%) had fallen ill (=attack rate).

Pitfalls to calculating and interpreting incidence rates (Box 2) are due to uncertainty about the population denominator and data source: data from health facilities is heavily affected by the degree to which people are utilising health services.

Changes in incidence rate over time may indicate the beginning, or the beginning of the end, of an epidemic, and help to monitor the impact of certain control interventions. By contrast, a good way to quantify the degree to which the community has been affected by the disease overall is to calculate the proportion (or percentage) of people who have newly developed the disease over a given period: this proportion is known as the attack rate (another misuse of the term rate). An *attack rate* is simply a cumulated

incidence rate (i.e. the attack rate over a three-month period is the sum of the incidence rates in months 1, 2 and 3).

Case-fatality ratio

Certain diseases are more lethal than others. The proportion of cases that dies of the disease is known as the *case-fatality ratio* (CFR; sometimes case-fatality rate is used instead – both are misnomers). CFR could also be calculated for all patients hospitalised irrespective of cause, for a specific ward (e.g. surgery). Similarly, Sphere guidelines state that the CFR of severely malnourished children in rehabilitation centres should be under 10%. One must specify among which group of patients the CFR is being calculated: for example, the CFR of untreated

Table 4: Main indicators of mortality

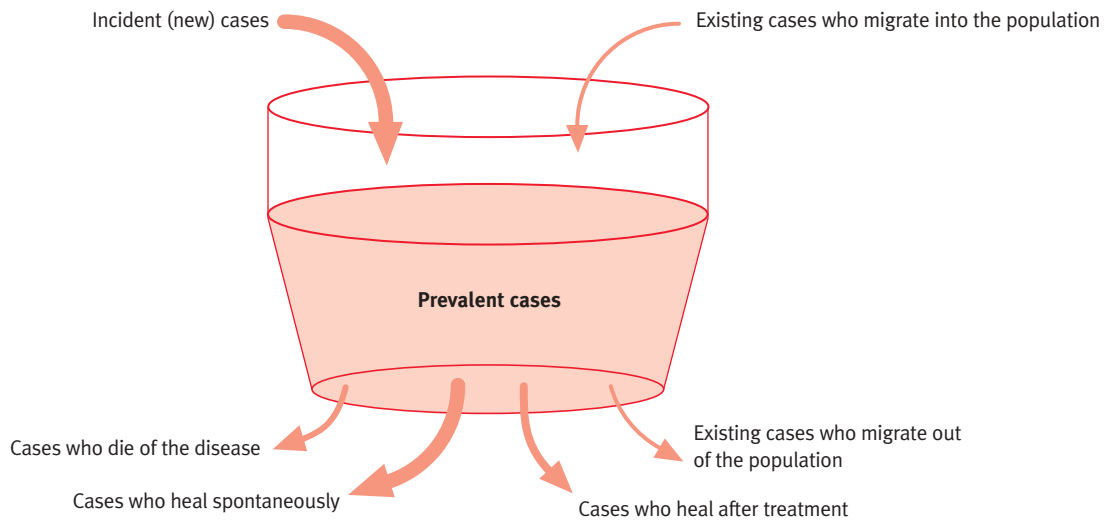
Indicator	Basic formula	What it quantifies	Common applications
Indicators commonly used in emergencies			
Crude mortality rate (CMR, or death rate)	Deaths due to any cause, in any age group/(population at risk x period of time)	Rate of occurrence (incidence) of death in the general population	Usually expressed as deaths per 10,000 people per day; always presented
Age-specific mortality rate (or death rate)	Deaths in age group/(population in age group at risk x period of time for those within the age range)	Rate of occurrence of death in a given age group	Most common is under-5 mortality rate (U5MR): deaths among children <5 years per 10,000 children <5 years per day
Group-specific mortality rate	Deaths among members of a given sub-group/(population belonging to the group at risk x period of time)	Rate of occurrence of death in a given group	Usually calculated for especially vulnerable groups, such as IDPs, orphans, etc.
Period-specific mortality rate	Deaths during sub-period/(population at risk during sub-period x duration of sub-period)	Rate of occurrence of death during a specific sub-period within the crisis	Monthly MR, MR during epidemic period, MR before/after displacement
Cause-specific mortality rate	Deaths due to a given cause/(population at risk x period of time)	Rate of occurrence of death due to a given cause in the general population	MR due to intentional injury; MR due to disease causing epidemic
Proportionate mortality	Deaths due to a given cause/total deaths	Proportion of all deaths that are attributable to a given cause	Usually expressed as a percentage; can be calculated in the general population or among people dying in a health facility
Case-fatality ratio (or rate) or CFR	Deaths due to a given cause (disease)/total cases of given disease	Probability of dying as a result of a given disease/cause of ill health (lethality of a given disease)	Can be calculated for a given disease/cause, or when evaluating the situation in a whole hospitalisation ward
Excess mortality rate (and total number of excess deaths)	Observed MR – expected non-crisis MR (x population at risk x period of time)	Rate of occurrence of death attributable to crisis conditions (total death toll attributable to the crisis)	Fundamental and objective indicator of crisis severity
Indicators less commonly used in emergencies, but prominent in long-term development settings			
Neonatal mortality ratio (or rate)	Deaths among neonates <28 days old/Live births	Probability of dying before age 28 days	Usually calculated for a given year (i.e. on an annual basis), and out of 1,000 live births
Infant mortality ratio (or rate)	Deaths among children < 1 year old over one year/Live births	Probability of dying before age 1 year	
Under 5 mortality ratio (or rate); also known as Child mortality ratio (or rate)	Deaths among children <5 years/Live births	Probability of dying before age 5 years	
Maternal mortality ratio	Deaths while pregnant or within 42 days of pregnancy termination, due to pregnancy-related causes/Live births	Probability of dying as a result of one's pregnancy	Usually calculated for a given year (i.e. on an annual basis), and out of 100,000 live births

falciparum malaria is roughly 1–2% among all children who contract it, but 30–50% among those with cerebral forms.

CFR is one of many indicators of mortality. The terminology for mortality indicators is often misused, resulting in confusion (see Table 4).⁵

Prevalence

Whereas the incidence rate expresses the number of new cases arising in the population per unit time, *prevalence* quantifies the number of cases (or infections) present at a given time. *Point prevalence* indicates cases present at a very specific point in time (a snapshot photograph), while

Figure 6**Relationship between incidence, prevalence and case-fatality ratio**

period prevalence indicates cases present over a given period, for example a month or a year (as in a photo taken with a very slow shutter speed). Prevalence is expressed as a proportion or percentage of the total population at risk (e.g. 40 prevalent cases in a population of 800 = $40/800 = 5\%$). Prevalence deals with all existing cases, new and old (but not, of course, with people who are no longer cases, i.e. who either recovered or died). The group of people need not be the entire population: one could calculate the prevalence of post-traumatic stress disorder in ex-child soldiers in Liberia in May 2007, or tuberculosis in adult males 45 years and above in a Somali refugee camp during 2006. This time-person-place reference resembles a sink, yielding an ever-changing water level (Figure 6). The greater the duration of disease or infection, the greater the prevalence (the longer water stays in the sink, the higher the water level).

When should one use incidence, attack rate and prevalence?

Prevalence, attack rate and incidence are often confused. One can calculate the prevalence of any disease. However, prevalence is greatly dependent on the duration of the disease or infection. For example, cholera is an extremely acute disease, which is healed or kills within days. Calculating the prevalence of cholera or other short-duration diseases is not meaningful.

Presenting incidence rates is generally useful, even if the diseases have a long duration, but is sometimes inappropriate (e.g. for conditions with uncertain onset, or that began at childbirth). When monitoring trends, incidence rate is generally the best indicator (HIV and malnutrition are important exceptions: because it is difficult to measure their incidence, we instead follow the evolution in their prevalence).

When wishing to quantify the importance (or *burden*) of the disease in a community, both the attack rate over a given time and the point or period prevalence can be presented. Attack rates are most useful to describe the cumulative impact of an epidemic. Consider the following:

- A meningitis epidemic: incidence rates are useful to monitor trends. The attack rate will show the proportion of the population that has fallen sick. Prevalence is not useful because the disease's duration is very short.
- Tuberculosis: incidence rates help to monitor transmission trends. Prevalence gives a measure of community burden. The disease evolves very slowly, so it would not make sense to calculate an attack rate, as one would in an epidemic.
- The onset of PTSD is difficult to establish; furthermore, incidence seems less relevant than prevalence, which quantifies burden and needs for mental care services.

'Prevalence rate' is a misnomer; 'morbidity rate' or 'disease rate' are equivocal; they should all be avoided.

Epidemiological properties of non-infectious diseases

The epidemiological quantities presented above also apply to non-infectious diseases. Exceptions are the serial interval, duration of infection/infectiousness and reproductive ratio, since these pertain to infectious pathogens.

With non-infectious diseases, we speak of *exposure* to factors (e.g. toxins, cancer-causing compounds, genetic traits, diet, traumatic experiences): this exposure is analogous to infection, since it initiates the disease

process. The interval between exposure and disease onset is not strictly speaking an ‘incubation’, but the same concept holds. In non-infectious disease, the dose (intensity) of exposure is usually paramount: it makes disease more likely, and accelerates its onset. Because non-infectious diseases are mostly chronic, prevalence is the most useful indicator of their burden.

Notions of risk

Risk and risk factors

Many epidemiological quantities above quantify, on an individual or population basis, the ‘risk’ of disease, i.e. the amount of disease in the population now (prevalence), its rate of occurrence (incidence rate) or the probability of dying from it (CFR); here we mean risk in the broadest possible sense.

Epidemiology not only describes this disease risk, but also explores *causal associations* between certain *risk factors* and disease. Risk factors (discussed in Chapter 3) can range from basic underlying issues, such as poverty, to environmental conditions, such as poor sanitation, to individual characteristics such as age, behaviour or genetic traits, situations such as living in a camp or being orphaned, or time periods, such as ‘after the start of conflict’, or ‘March 2007’. Protective factors reduce risk (e.g. exclusive breastfeeding protects against infant infections). Statements about exposure to a risk factor must be unequivocal: thus, ‘low food intake’ or ‘young age’ are not acceptable definitions of exposure; ‘average caloric intake <2,100 Kcal/day over the last three months’, or ‘age 59 months or less’, is.

The relative risk

By how much does a risk factor increase risk, or a protective factor decrease it? Compare the risk A in people who are exposed (or more exposed) to the factor to the risk B in those who are not (or less) exposed. A/B is the *relative risk* (RR or *risk ratio*), namely the answer we seek: it tells us by how much the risk is multiplied in the exposed versus the non-exposed. If $A > B$, $RR > 1$, i.e. exposure to the factor increases risk. If $A < B$, $RR < 1$, i.e. the factor is protective. If $A = B$, $RR = 1$, i.e. there is no effect.

The indicator of risk depends on what we are interested in observing. Thus, incidence rate ratios, prevalence ratios, mortality ratios and so on are all valid expressions of relative risk (Box 3). The *odds ratio* is less intuitive, but often measured, especially during outbreak investigations, essentially when measuring more straightforward RR expressions is unfeasible. It compares exposure in cases to exposure in non-cases. If cases have been exposed to a factor more than non-cases, there is probably an association between this factor and disease.

Disease itself can be a risk factor for further outcomes (e.g. malaria causes poor pregnancy outcomes), and relative risks are often bi-directional (e.g. see the vicious cycle of malnutrition and disease, discussed in Chapter 2).

Why think in terms of relative risk?

There are two main applications of relative risk:

- Identifying risk or protective factors, so as to identify interventions that are most likely to help. Various types of studies (e.g. *case-control*, *clinical trials*, *cross-sectional*, *cohort*) can compare groups of people exposed to a hypothesized risk factor, or to a given intervention, to groups that were not exposed, to measure the RR of disease in the exposed group, and thus investigate the causal link between risk factors and disease, or the effectiveness of interventions.
- Comparing the risk in different population groups, locations or periods, to better target interventions. Here, a baseline group (or category) is delineated to serve as the non-exposed denominator of the RR (e.g. people not displaced; the period before the conflict; the month of April 2007; persons > 15 years of age).

Relief health programmes are informed heavily by existing evidence about risk or protective factors. Sometimes, the evidence is weak, highlighting the need for more research. For many factors, however, the evidence is extremely strong: for example, it is well-known that indoor smoke increases the risk of childhood pneumonia; and that people who sleep in insecticide-treated shelters have a lower risk of malaria. Knowledge of relative risks from previous studies is summarised through *systematic reviews*, and used to formulate guidelines and standards (like Sphere).

Sometimes, RR are calculated during the crisis itself, usually through disease outbreak investigations, and can have immediate applications. For example:

- During a 1999–2000 epidemic of bloody dysentery in Sierra Leone, the RR of dying of the disease (CFR) among children under 5 years was 2.9 compared to older persons.⁶
- In West Darfur, investigation of a 2004 outbreak of hepatitis E in a camp showed that, surprisingly, the odds of having consumed chlorinated surface water were 2.2 times higher among cases than among healthy people ($OR = 2.2$).⁷
- A 2002 survey of mortality among Angolan IDPs found that the risk of death was 1.4 times higher in camps than before arrival to camps.⁸

The population-level effect of risk factors

On a population level, we need to consider how high the RR of a given risk factor is, but also what proportion of the population is exposed to the factor. Together, these two elements yield the *population attributable risk*. Compare factor A which increases risk 20-fold, but to which only 1% of the population is exposed, to factor B, which increases risk only two-fold, but to which 50% of the population is exposed: A is more dangerous at the individual level, but clearly B is a greater priority for population control.

Box 3**Examples of different expressions of relative risk****Relative risk as a prevalence ratio**

Records of a mental health clinic show that the prevalence of Post-Traumatic Stress Disorder (PTSD) was 103 out of 151 (68.2%) bomb survivors screened, and 21 out of 126 (16.7%) people who had not been in a bombing.

The relative risk (RR) of PTSD was thus 4.1 (68.2% /16.7%) in bomb survivors, compared to others.

Thus, bomb survivors were about 4 times more likely than others to have PTSD.

Relative risk as an incidence rate ratio

An observational study shows that the incidence rate of diarrhoea among children < 5 years was 11 per 1,000 per month before the instalment of new boreholes, and 6 per 1,000 per month afterwards.

The RR of diarrhoea was thus 0.54 (6/11) after the intervention, compared to before.

After borehole installation, diarrhoea occurred at about half the rate in children as before.

Relative risk as an attack rate ratio

During an earthquake, about 450 severe injuries occurred among approximately 7,000 schoolchildren (attack rate 6.7%), whereas among adults the severe injury attack rate was 540 out of 35,000 (1.5%).

The RR of severe injury in schoolchildren versus adults was thus 6.7/1.5, or 4.5.

The risk of experiencing a severe injury during the earthquake was 4 to 5 times higher in children than in adults.

Relative risk as an odds ratio

Epidemiologists investigating an outbreak of an unknown fever-causing disease discover that, among 45 cases, 20 had drunk cow's milk in the past two weeks, while 25 had not. Among 85 healthy people, 6 had drunk milk, while 79 had not. The odds of drinking milk were 20:25 (0.80) among the cases, and 6:79 (0.08) among the non-cases. The odds ratio (OR) of having drunk milk was thus 10.0 (0.80/0.08) among cases, compared to non-cases, suggesting milk could be a risk factor for infection.

Cases were 10 times more likely than non-cases to have drunk milk. Note the upside-down logic behind the odds ratio.

Similarly, a powerful protective factor will not have a considerable effect unless a considerable proportion of the population benefits from it (e.g. exclusive breastfeeding will only improve child health on a population level if many mothers adopt it; see Chapter 5).

Causality and confounding

A common mistake in epidemiology is to observe correlations between any factor and the outcome, and thereby mistake a spurious relationship for true causality. Consider the following:

- Maternal mortality is three times higher in ethnic group 1 than in ethnic group 2. The superficial conclusion might be that ethnicity, i.e. genetics, affects the risk of dying in pregnancy. However, a much more likely explanation is that ethnic group 1 is poorer or has less

access to treatment than group 2. Ethnicity is thus here a *confounder* of the causal relationship between poverty or health access and maternal mortality.

- Ex-child soldiers who spent time in rehabilitation centre A were evaluated on exit, and 37% were found to have signs of mental illness. By contrast, children exiting centre B had a mental illness prevalence of 18%. Would it be right to conclude that centre B provided more effective rehabilitation? Perhaps not: the observed difference might actually reflect the proportion of children admitted to each centre who had been directly exposed to violence during the war (perhaps centre A children had been more directly involved in combat). Centre of residence is a possible confounder here.
- Two months into a malaria epidemic, bed nets are distributed: entomologists find that the mosquito population has fallen two-fold since the start of the bed net programme, and the epidemic starts declining right

afterwards. The agency in charge believes it has had an impact, but is this justified? While decreasing mosquito population is correlated with increasing bed

net ownership, in reality climate (e.g. end of the rains and drying of stagnant pools) may be the true reason for the falling mosquito numbers.

Chapter 3

The effects of crises on health

A simplified general model of disease in the population

From healthy state to infection or exposure, disease and death

Understanding how different crisis conditions result in morbidity and mortality requires some grasp of the population dynamics of disease, in particular of infections. We can now tie the epidemiological quantities defined above together into an overall model (or representation) of disease (see Figure 7). This simplified model does not accommodate various nuances specific to individual diseases, but probably suffices to extract key quantitative relationships that illustrate the effects of risk factors and protective factors (interventions).

At any time, a proportion of the population is susceptible, i.e. can contract the infection or be exposed to the disease-causing agent (for non-infectious diseases). For infectious diseases, susceptibility will usually be highest if the disease has long been absent from the community, and will decrease the more people have been infected previously, and have developed some immunity. It will also decrease the more people are vaccinated (assuming a vaccine exists). However, many diseases do not confer any immunity. Furthermore, some diseases and vaccines do confer immunity, but, with age, the body's immunological memory wanes and antibody production decreases, so that people once again become susceptible. For most diseases there is a back and forth flow between 'fully susceptible' and 'fully non-susceptible', with

many shades in between (e.g. malaria infection never yields complete immunity, but in endemic areas leaves adults with a far lower risk of life-threatening episodes). Thus, susceptible and non-susceptible are grouped into one box: the non-infected/non-exposed.

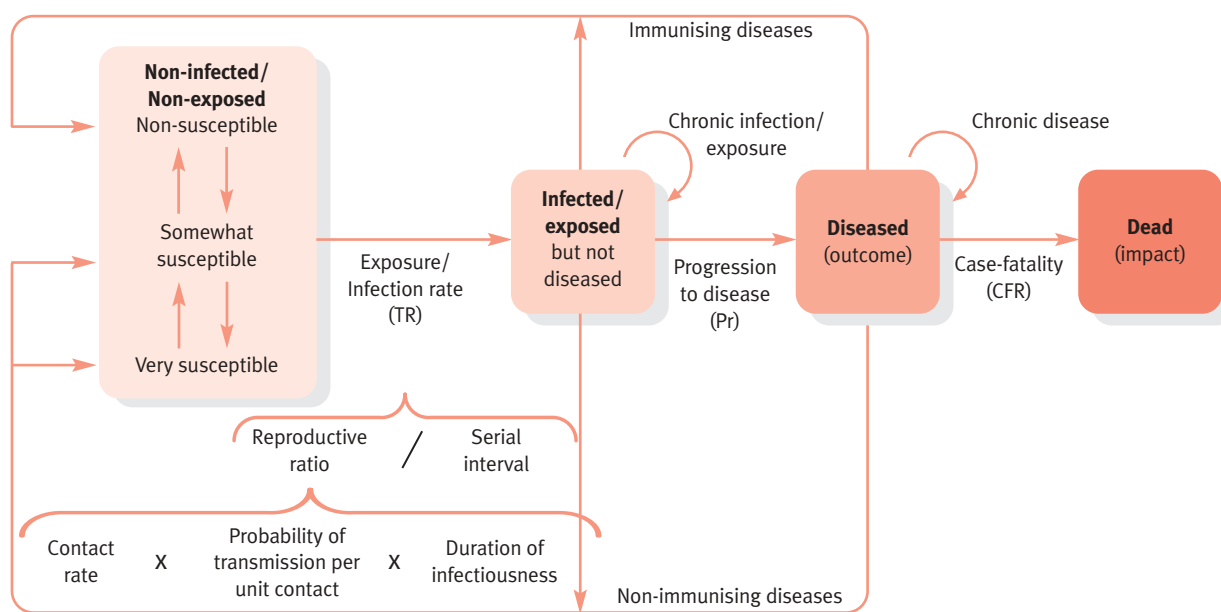
The greater the proportion of susceptibles, the greater the contact rate 'c' and thus R (only applicable to infectious diseases). R divided by the serial interval equals the rate of new infections (infection rate or TR), namely the flow from the uninfected to the infected boxes (TR is our own abbreviation). For non-infectious diseases, TR is the rate at which people are being exposed (e.g. to a cancer-causing agent or to a PTSD-causing event).

A proportion Pr of those infected develop symptomatic, or clinical, disease. Others remain chronically infected/exposed without any symptoms. The rest clear their infection spontaneously or following treatment: if the infection confers immunity, they return to the non-infected, non-susceptible box; if the infection is non-immunising, they will be susceptible to a new episode.

Out of those who develop symptoms, a proportion dies because of the disease (unless the disease is completely benign): this proportion is simply the CFR. Survivors return to the susceptible/non-susceptible box, as above. Some diseases, however, neither kill nor heal, but persist chronically (e.g. non-infectious conditions like hypertension, diabetes, asthma and mental disorders).

Figure 7

Simplified framework of the dynamics of disease in a population



Lag time between infection and death in slow-onset diseases

For short-duration and short serial interval diseases (diarrhoea, measles, meningitis), flow from susceptibility to death takes days. For slow-onset diseases such as TB, HIV/AIDS or cancer, however, the delay between the TR step and death can be many years. This has two important implications:

- For slow-onset diseases, crises' effects on TR will mostly manifest themselves as clinical disease long after the acute emergency is over. For example, if conditions in a refugee camp cause increased TB transmission, the resulting increased caseload might mainly be borne by the health system of the country of origin years after repatriation. Minimising the TR of slow-onset diseases (TB, HIV, sleeping sickness, leishmaniasis) during a crisis is thus justifiable as a forward-looking preventive action (note, however, that for HIV there is currently no evidence that conflict increases TR).
- In an emergency, the *immediate* concern with TB, HIV and non-infectious diseases such as hypertension is mainly not their TR, but rather their Pr and CFR. As discussed later, many risk factors brought about by crises make Pr faster and more probable, and increase CFR. Thus, what matters here is the burden of already prevalent cases, which may have been contracted before the crisis (in diseases such as diabetes or cardiovascular problems, exposure is often genetic or takes place early in life). In emergencies, the main challenge is to minimise Pr and CFR by ensuring uninterrupted treatment (e.g. TB drugs, antibiotics and antiretrovirals, insulin, heart medication).

Tying it all together

The exposure/infection rate TR, multiplied by Pr, gives the incidence rate (i.e. the rate of new symptomatic cases), or IR (Equation 2). There is a lag time (incubation period) between infection/exposure and incidence. Similarly, $IR \times CFR = \text{mortality rate (MR)}$, with a further lag time. The sum of all MRs due to individual diseases gives the overall MR for the entire population (Equation 3; in reality there is more complexity, since death is often caused by more than one disease occurring together). These relationships are fundamental to understanding what health interventions can accomplish.

Excess morbidity and mortality

Definition of excess morbidity and mortality

Morbidity and mortality that occur directly or indirectly because of the crisis, i.e. that would not have occurred had the crisis not taken place, may be considered in *excess* of the norm. Excess morbidity and mortality due to any disease occurs if crisis-associated risk factors increase TR and/or Pr and/or CFR beyond their pre-crisis levels (unless an increase in one of the three is compensated by a decrease in the others – but this is implausible, at least in the acute emergency phase).

If 'crisis' is the exposure, and pre-crisis the baseline, we can rephrase this in more epidemiological terms: excess morbidity and/or mortality occur when the relative risk of TR, Pr and/or CFR in crisis versus pre-crisis exceeds 1. Alternatively, excess morbidity and mortality are the summation, at the population level, of crisis-attributable excess risk at the individual level.

Typical scenarios of the evolution of morbidity and mortality

We have already listed the different crisis conditions covered in this paper: (1) entrapment due to surrounding violence, (2) mass displacement into camps, (3) displacement into host communities, (4) natural disaster, and (5) food crisis.

When quantifying mortality, we are interested in its elevation compared to the pre-crisis baseline (excess mortality rate), the number of people experiencing this excess mortality rate and the amount of time that this elevation persists:⁹ these three elements determine the absolute number of excess deaths attributable to a given crisis. The same holds for incidence rate and morbidity. While there is no 'typical' blueprint for the chronological evolution of morbidity and mortality in crises, considering the above conditions helps to delineate rough rules of thumb.

- Condition 1 (entrapment) may not result in extremely high mortality rates. However, considerable elevations in mortality do occur over large populations and time periods (think of eastern DRC or eastern Burma). The result is often unfathomably large death tolls, as in DRC (more than 4 million).¹⁰
- Condition 2 (sudden mass displacement into camps) is the best documented. Generally, a dramatic spike in mortality occurs, and then after a few months death rates typically decline, eventually reaching pre-crisis levels.
- Condition 3 (displacement in the host community) is poorly documented, probably because refugees, IDPs and informal migrants disperse into the local community, where they are difficult to identify and track. As discussed later, this type of displacement should theoretically lead to better health outcomes, but this requires confirmation.¹¹
- Condition 4 (natural disasters) usually results in a dramatic peak in mortality and injury within the first 72 hours. Survival chances after this time are bleak. Evolution in ensuing weeks depends largely on the occurrence of epidemics, though these have usually been of modest proportions.¹²
- Condition 5 (food crisis) has an insidious onset, but progresses rapidly (i.e. a couple of months). It may result in very high mortality, especially among children. Mortality is intimately related to the prevalence of acute malnutrition, but will also be linked to the occurrence of epidemics favoured by malnutrition itself. Food crises generally accompany both Condition 1 and 2.

Equation 2**Relationship between infection, progression to disease, case-fatality ratio, incidence rate and mortality due to a given disease**

$$\text{MR due to disease X} = \text{TR} \times \text{Pr} \times \text{CFR}$$

[lag time: incubation period + duration of disease before death]

where

MR due to disease X = specific mortality (or death) rate due to a given disease

TR = transmission rate, i.e. rate at which the population is becoming infected with the pathogen responsible for the disease

Pr = probability an infected person will actually develop the disease†

CFR = case-fatality ratio of the disease, including treated and untreated cases†

and

$$\text{IR of disease X} = \text{TR} \times \text{Pr}$$

[lag time: incubation period]

where

IR due to disease X = incidence rate of a given disease

so that

$$\text{MR due to disease X} = \text{IR} \times \text{CFR}$$

Note that:

- If TR, Pr or CFR increase, so will MR.
- The lag time between infection (TR) and death (MR), namely the sum of the incubation period (how long it takes to progress to disease) and the duration of the disease before death, varies widely (compare ebola with HIV).

Example 1. A cholera epidemic breaks out in a war-torn city. Imagine that TR, if it could be measured, were 300 per 10,000 people per day. About one in ten cholera infections progress to disease (i.e. Pr = 10%), and the CFR in such a setting might be 5%. The incubation period is 2–3 days and people die within 2–3 days of disease onset. The MR due to cholera would thus be 1.5 deaths per 10,000 per day, with about a 4–6-day lag time.

Example 2. In a village, the incidence rate of African sleeping sickness is 5 per 1,000 per month. It is thought that all cases progress to disease (Pr = 100%) and that all die unless treated (CFR = 100%). It takes about 2 years to progress to disease, and 2 years from disease onset to death. The MR of sleeping sickness will also be 5 per 1,000 per month, but with a 4-year lag time.

† Strictly speaking, we should actually specify these quantities as the rate of progression from infection to disease, per unit time, and the rate of death among disease cases, per unit time. But this is a mathematical detail.

Equation 3**Relationship between mortality due to individual diseases and total mortality**

$$\text{CMR} = \text{MR due to cause/disease A} + \text{MR due to cause/disease B} + [\dots] + \text{MR due to cause/disease X}$$

where

CMR = (crude) mortality (or death) rate due to all causes/diseases and in all ages

Different demographic and epidemiological settings

The magnitude and causes of excess morbidity and mortality vary according to the underlying demographic and epidemiologic profile of the population, *irrespective* of crisis conditions. We can (very roughly) distinguish three types of settings (Table 5, page 20).

Chapter 4 discusses how to plan for an appropriate response based on the above classification. The shift from infectious to non-infectious disease in transition settings is insufficiently appreciated: during the 2005 Indian Ocean tsunami response, drug kits sent to Sri Lanka and India contained mostly anti-infective drugs, and not enough

Table 5: Types of demographic and epidemiological settings in which crises can occur

Type of setting	Main regions (as of 2007)	Demographic profile	Epidemiologic profile
Tropical and/or very poor	Sub-Saharan Africa except for South Africa, Haiti, rural/remote parts of Central and South America, Afghanistan, Nepal, Burma, Laos, Cambodia, Papua New Guinea, Timor Leste	Pre-transition: high birth and death rates, low life expectancy (<55y); typical population comprises 20% under 5y, 50% under 15y and 3% pregnant	Pre-transition: most disease and deaths due to infectious diseases, children and pregnant women particularly affected (under 5 years mortality rate usually twice or more all-age mortality rate); unintentional injury also important
Transition	South Africa, urban/wealthy regions of Central and South America, North Africa and Middle East, China, South and South-East Asia except above, Oceania except Australia and New Zealand	In transition: falling birth and death rates, rising life expectancy (55–75y)	In transition: pockets of high infectious-disease burden subsist, but non-infectious diseases are increasingly dominant due to the aging population trend; unintentional injuries important; large within-country inequalities
Industrialised	North America, Europe and ex-Soviet states, Japan, South Korea, Australia and New Zealand	Post-transition; low birth and death rates, life expectancy >75y (the exception is ex-Soviet states where birth rates remain low but death rates are increasing)	Post-transition: most disease and death due to non-infectious diseases (in some ex-Soviet states, infectious disease mortality is increasing again, especially due to HIV and TB)

See www.gapminder.org for further information.

treatments for common chronic conditions. Mental illness is a major health problem everywhere.

Different risk factors

Crises bring about or exacerbate certain risk factors. These operate at different levels of causality, i.e. more or less remotely from the outcome, which we can roughly categorise as distal, intermediate and proximate (Figure 8). For example, proximate factors causing higher diarrhoeal incidence among babies in eastern DRC could be poor water and sanitation and high malnutrition prevalence; however, looking more broadly, we might identify intermediate factors such as food insecurity due to militia attacks. Further, we might conclude that, ultimately, underlying distal factors such as regional instability are to blame.¹³

Risk factors can be organised into conceptual frameworks, useful for organising studies, but sometimes very intricate. We will focus on proximate factors, namely those which relief *can and should* do something about, and discuss only the most important, i.e. those which are recognised to cause the vast majority of excess morbidity and mortality:

- overcrowding;
- inadequate shelter;
- insufficient nutrient intake;
- insufficient vaccination coverage;
- poor water, sanitation and hygiene conditions;
- high exposure to and/or proliferation of disease vectors;

- lack of and/or delay in treatment; and
- warfare among combatants and armed attacks against civilians.

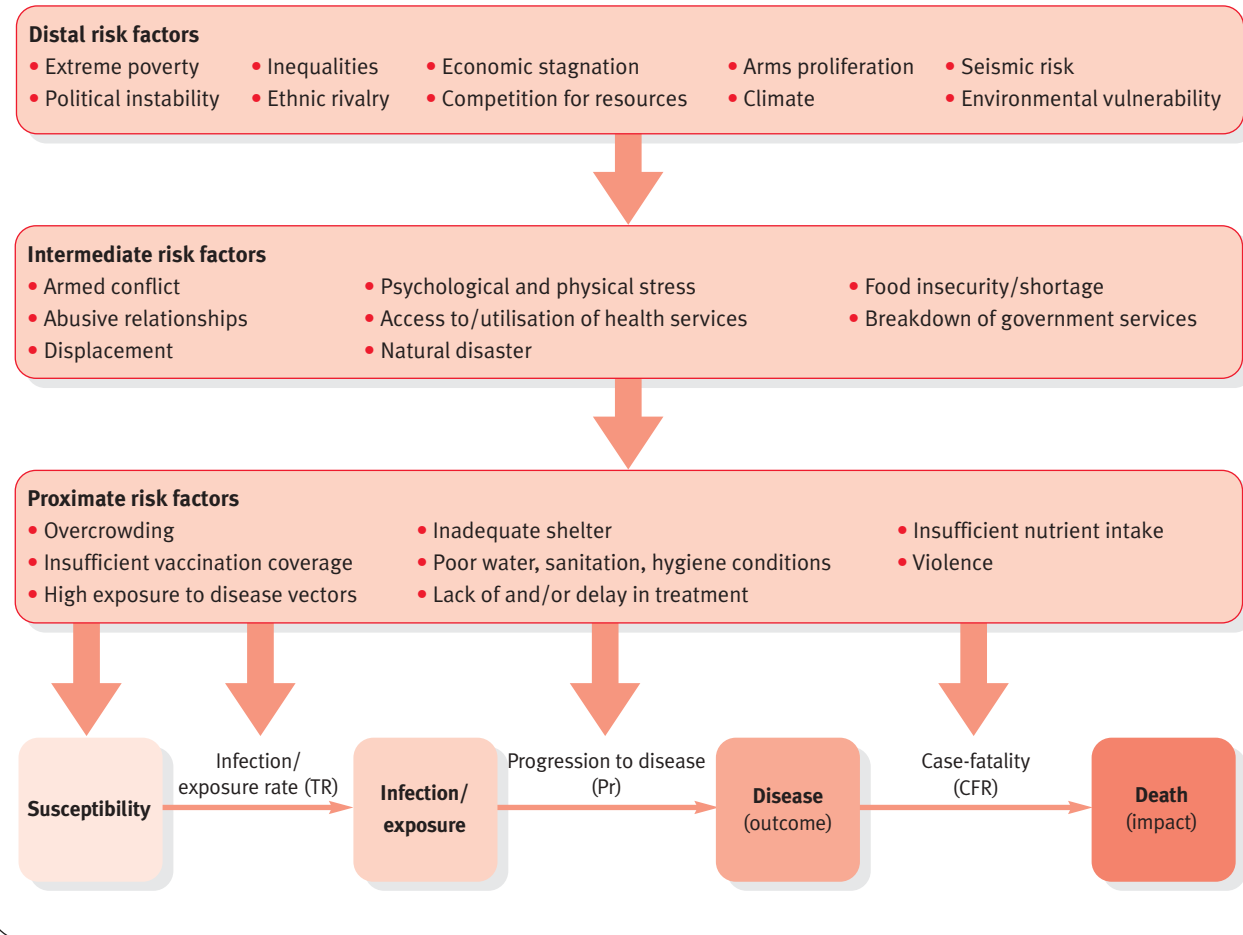
A similar list is found in other guidelines on emergency health interventions. Violations of international humanitarian law (such as forced displacement and destruction of food supplies) experienced by households are probably an intermediate factor, but have been shown to increase the risk of all-cause mortality.¹⁴ As further evidence accumulates, these will merit a more in-depth discussion.

An alternative framework, common in epidemiology, is to consider risk factors that affect the host (humans), the agent (the pathogen) and the environment within which hosts and agents interact (Box 4, page 22).

Notes on specific risk factors

Overcrowding

There are two kinds of overcrowding, which usually occur together: high population density (many people per unit area), as well as a large population concentration (say, tens of thousands or more) sharing the same settlement. In addition to psychological effects (e.g. lack of privacy), high population density increases contact between infectious cases and susceptible people: this is the ‘c’ component of R, and explains why the R of many diseases is higher in camps than in open settings. This inherently higher risk due to overcrowding is perhaps insufficiently appreciated

Figure 8**Examples of distal, intermediate and proximate risk factors of excess morbidity (disease) and mortality (death) in a crisis**

by relief agencies, but is implicitly recognised by Sphere guidelines, which specify that temporary or self-settled camps should offer at least 45m² surface area per person.

We can illustrate this effect through mathematical modelling (contact Rebecca Grais for details on this analysis: rebecca.grais@epicentre.msf.org). Figure 9 (page 23) shows the evolution of a measles epidemic in a population of 10,000 children according to different population densities. We assume everyone is susceptible at the start, and that a case is introduced. As in any model, this illustration oversimplifies reality, but highlights essential lessons.

As expected, it takes some time before the epidemic takes off, due to the length of the serial interval. Eventually, the attack rate reaches 100% in all cases, but the epidemic progresses much faster in situations of higher population density, leaving far less time for reaction.

Large population concentrations also favour faster epidemic spread, and place a larger number of people at risk. Compare a sprawling IDP camp in northern Uganda

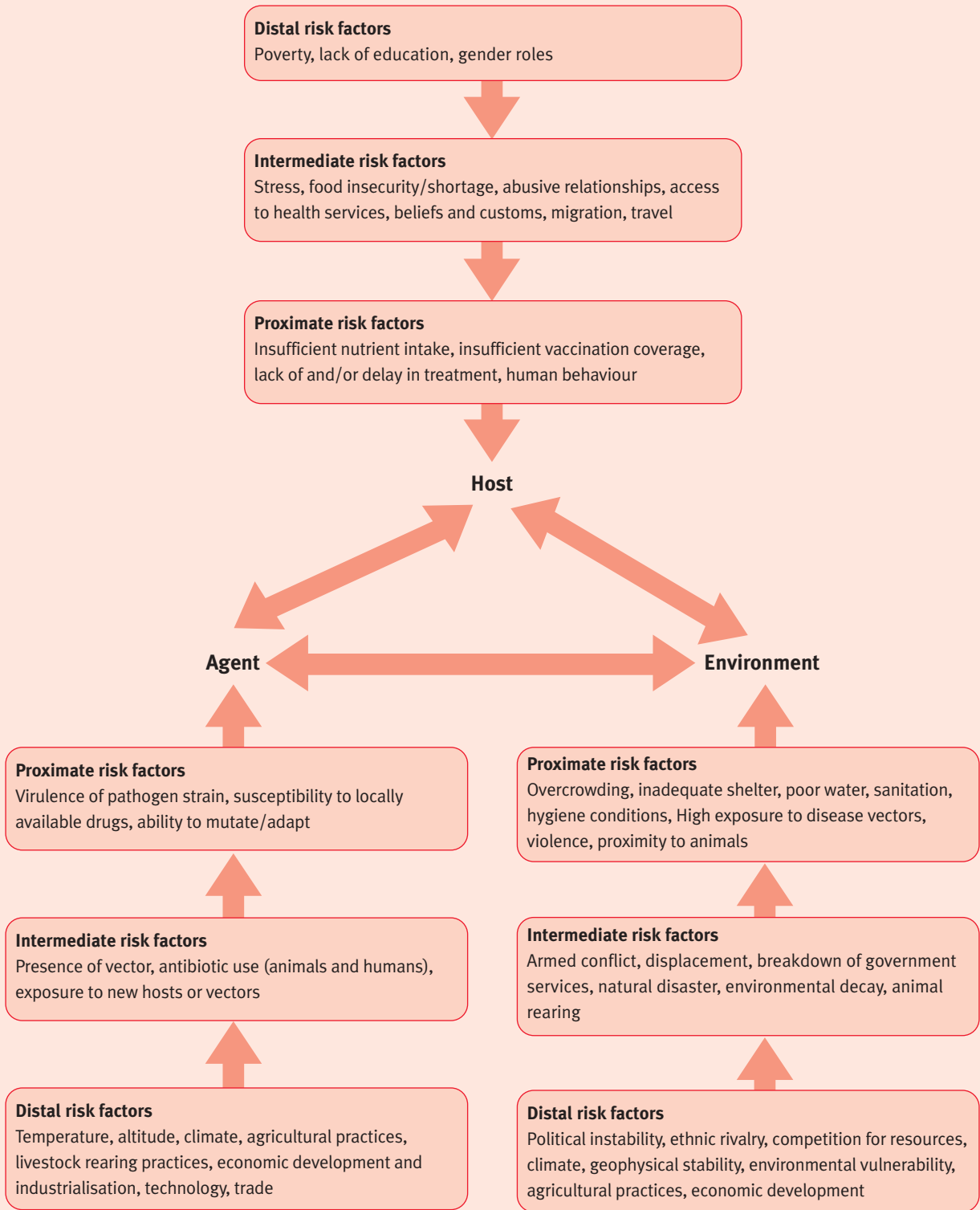
with isolated mountain villages in Nepal: the distance between villages acts like a firebreak, delaying or blocking disease transmission from one village to the next.

Which diseases' TR is affected by overcrowding? Mainly those spread by air droplet (particularly Acute Respiratory Infections or ARI, measles, meningitis, TB and flu) and faecal-oral (all the diarrhoeal diseases including shigella and cholera) routes. Vector-borne diseases such as malaria do not particularly depend on overcrowding. Furthermore, overcrowding does not affect Pr or CFR.

Overcrowding is advantageous for certain mass preventive interventions such as vaccination, since people can easily be reached. However, because of overcrowding such interventions must achieve a higher coverage than they would in more spread-out settings. In summary, forcing people to live in overcrowded conditions, while sometimes dictated by insecurity, places them at an unavoidable risk of contracting disease, which can lead to excess morbidity and mortality and increases requirements for disease control.

Box 4

Host-agent-environment framework of disease



Inadequate shelter

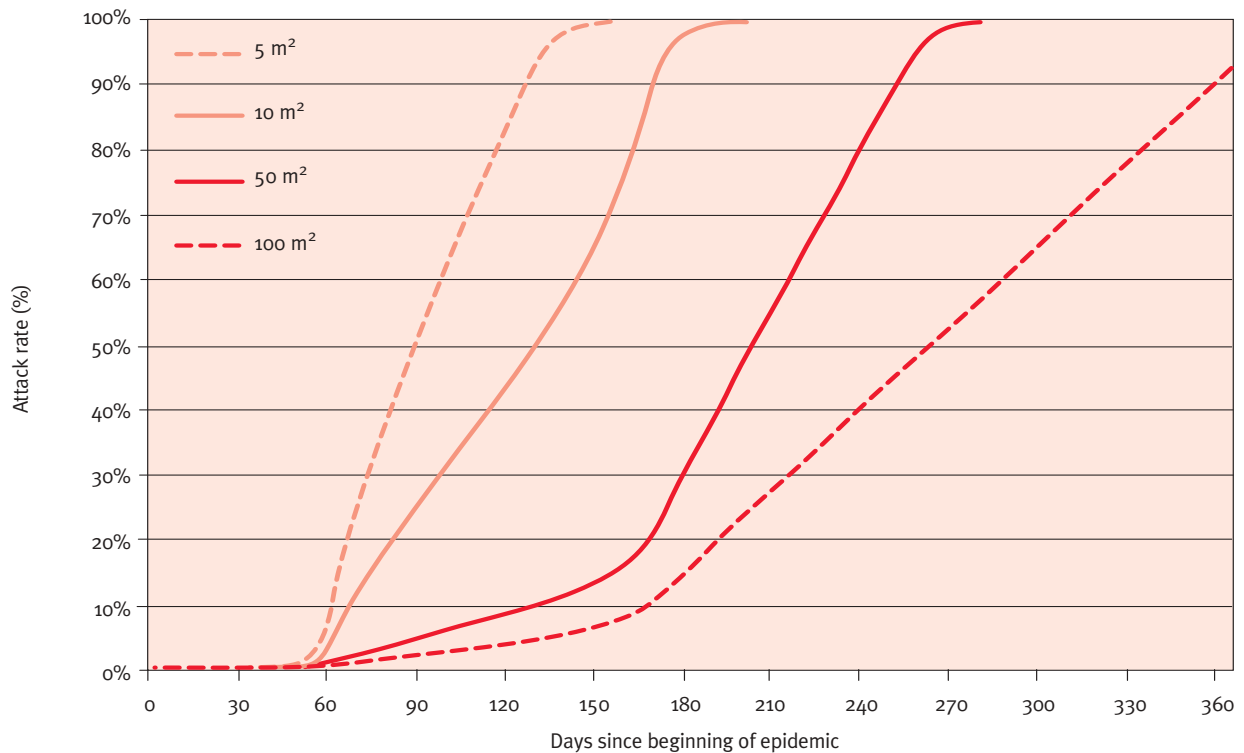
Poor shelter increases disease risk in at least three ways:

- Exposure to the elements: in hot climates, lack of shade will make people, especially children, dehydrated,

which can compound other dehydrating diseases, notably diarrhoea, thus increasing their CFR. Exposure to rain and cold (or even cool) temperatures probably favours the Pr of ARI and other respiratory diseases.

Figure 9

Predicted progression of a measles epidemic, in the absence of interventions, according to different population densities (m² per person)



- Exposure to disease vectors: inadequate roofing or walls may increase the insect biting rate at night, and thus the TR of vector-borne diseases, particularly malaria. Furthermore, spraying insecticide or setting up bed nets is difficult in some temporary shelters.
- Indoor smoke due to cooking or heating inside the shelter, without proper air exhausts, is a major risk factor for ARI and asthma. Pr, causes excess burn injuries and increased risk of fires.¹⁵

Insufficient nutrient intake

Insufficient nutrient intake causes malnutrition, which in turn increases the Tr, Pr and CFR of almost all infectious diseases.¹⁶ Insufficient nutrient intake occurs when households cannot obtain food in sufficient quantities and variety, because they cannot farm, harvests fail, food is absent in markets or unaffordable, or markets are unreachable: the concept of food insecurity captures these determinants. Populations move from situations of food insecurity to outright nutritional emergencies and, rarely, to generalised famine, defined based on selected food security indicators and the prevalence of acute malnutrition in children.

Food insecurity is to food crises what high malnutrition prevalence is to nutritional crises, but food and nutritional crises are not equivalent. Food insecurity does lead to lower nutrient intake and a greater prevalence of

malnutrition, but is not their only cause. Disease and insufficient family and social care (incorrect infant feeding, neglect) can exacerbate nutritional crises. Epidemics of measles and diarrhoea can themselves trigger nutritional crises by sheer virtue of the high proportion of children who have incurred rapid weight loss while ill.

The relationship between malnutrition and (child) mortality is complex. Higher malnutrition prevalence usually results in higher mortality, with a short lag time (weeks): food crises are almost always compounded by health crises. Furthermore, as malnutrition increases, mortality rises not linearly, but exponentially. In some stable settings, especially in South Asia, high malnutrition prevalence occurs regularly due to seasonal food shortages but is not accompanied by alarming child mortality; why this is so is unclear. Similarly, the absence of a food crisis does not mean mortality will remain low: disease or intentional injury could still cause high death tolls.

In entrapment conditions, food insecurity may evolve slowly. Populations may never completely lose access to food, but nutritional status gradually worsens. Furthermore, large populations may be affected, so that, even if malnutrition prevalence remains below emergency levels, the population risk attributable to insufficient nutrient intake is huge, and the mortality price staggering.

Table 6: Risks at the community and individual levels according to different scenarios of vaccination/immunisation status

		Status of individual X:	
		Not vaccinated and not immune due to previous exposure to disease	Vaccinated or immune due to previous exposure to disease
Status of community: Proportion in community who are vaccinated or have acquired immunity due to previous exposure to disease	Low	High risk of outbreak occurring Outbreak will have high attack rate Very high risk for individual X	High risk of outbreak occurring Outbreak will have high attack rate Very low risk for individual X
	High	Low risk of outbreak occurring Any outbreak is likely to feature a low attack rate Low risk for individual X overall (but high should an outbreak occur)	Low risk of outbreak occurring Any outbreak is likely to feature a low attack rate Very low risk for individual X

In mass displacement, populations suddenly find themselves in unfamiliar territory, having brought along limited or no food supplies. Here, malnutrition behaves like a fulminating epidemic, unless food distributions are implemented within days.

Breakdown of vaccination services

In any pre-crisis setting, vaccination is a routine government health service: caregivers are encouraged to bring children to health facilities to receive vaccines for diseases covered under the Expanded Programme on Immunization (EPI). The list of EPI vaccines varies by country, but currently comprises at least the following: the BCG vaccine against TB, an oral polio vaccine (OPV), diphtheria, pertussis and tetanus (DPT) and measles. Vaccines against rotavirus, *Haemophilus influenzae* type b, and various pneumococcal strains are now available, and are increasingly being included in EPI. Pregnant women also receive tetanus shots, to prevent neonatal tetanus.

Vaccination services aim to achieve a coverage of the population sufficient to prevent or minimise outbreaks (this critical level is known as the *herd immunity threshold*). Crises reduce vaccination coverage, i.e. increase the pool of susceptible individuals, and thus 'c'. The lower the vaccination coverage, the higher the chance that any imported case will cause an outbreak, and the higher the R of the outbreak, meaning higher incidence and attack rate. There is thus a heightened risk at the community level (more chance of an epidemic breaking out) and at the individual level (more chance of contracting a vaccine-preventable disease and dying from it; Table 6).

The effects of a breakdown in vaccination vary depending on the crisis condition:

- Entrapment generally leads to a gradual deterioration of EPI services. The effects could take weeks or months. As time progresses, new births occur in the community: because of poor vaccination services, these newborns swell the ranks of the susceptible, eventually bringing coverage below the herd immunity threshold.

- With mass displacement into camps, the most pressing issue is overcrowding, which raises the R of many vaccine-preventable diseases, especially measles and pertussis (whooping cough). Vaccination coverage sufficient to prevent outbreaks in the communities of origin suddenly is no longer adequate, explaining why outbreaks can strike within two weeks of a camp's creation, even in populations with relatively high coverage (e.g. >80%). Furthermore, the large population concentration exposes more people to the outbreak. Hence the absolute requirement to vaccinate 100% of children against measles as soon as possible and *no matter what*.
- Displacement into host communities may expose locals to cases introduced by unvaccinated IDPs or refugees, or vice versa. However, the overcrowding effects are not present. A displaced, unvaccinated person living in a host community may be shielded from infection by local, vaccinated people surrounding them, who provide a virtual 'epidemiological barrier'. The main risk is at the individual rather than community level, due to unregistered, 'invisible' IDPs not accessing local EPI or vaccination campaigns by relief agencies. This is however speculative due to insufficient evidence on health outcomes among host community IDPs.
- Natural disasters have not been associated with dramatic epidemics of vaccine-preventable diseases, mainly because local health systems are able to resume EPI fairly quickly. An exception is when disasters lead to displacement into camps.
- Food crises do not in themselves decrease vaccination coverage, but malnourished children have a compromised immune system, which may reduce their ability to develop immunity following vaccination, and increase TR and Pr. Thus, poor vaccination coverage interacts synergistically with malnutrition.

The nightmare scenario is that of a malnourished population that has lived in entrapment conditions for months or years (i.e. has decreased immunity and low vaccination coverage) and suddenly moves en masse to an overcrowded camp.

Resuming routine EPI-style vaccination is important, but implementing special ‘catch-up’ vaccination campaigns so as to rapidly restore coverage above the herd immunity threshold is paramount. Obviously, one-dose vaccines (e.g. measles, meningitis, yellow fever) are more amenable to vaccination campaigns.

Some vaccines confer protection not only against TR, but also partly against Pr and CFR. For example, measles vaccination will reduce the number of infections, but also the proportion of infections that result in severe disease.

Poor water, sanitation and hygiene conditions

Inadequate water, sanitation and hygienic behaviours mainly increase the TR of faecal-oral diseases, by increasing the likelihood that a faecal pathogen expelled by a person or animal will be ingested by another person, through one of the following routes:

- Water is already contaminated at the point of collection:
 - Surface water (pools, lakes, rivers) contaminated by excreta from defecation runoff.
 - Wells, pumping systems or aqueducts contaminated by surface water or faeces, which seep in when floodwater overwhelms defences; wells are unprotected (meaning surface water can flow into them, or people must dip hands or containers into them); and when aqueducts have holes or low pressure.
 - Aqueducts or pumping systems drawing on insufficiently purified water (through chlorination or other methods). Note that most deep wells/boreholes draw from microbiologically pure groundwater.
- Water is contaminated after collection, e.g. when containers cannot be sealed and have a wide neck, or hands are dipped into them.
- Food is contaminated during preparation:
 - Food is washed with contaminated water.
 - Market sellers, cooks or consumers handle food with contaminated hands.
 - Flies smear themselves with pathogens while feeding on faeces, and deposit them on food. Proliferation of flies is a telling sign of grossly insufficient sanitation.
- Hands are contaminated during visits to unsanitary latrines or defecation fields, while washing babies or handling animals, while playing, etc.

Unplanned mass displacement leaves people without adequate water and excreta disposal. Water and sanitation also deteriorate, albeit more slowly, in entrapment conditions: sources may be inaccessible due to insecurity; aqueducts, pumps and latrines fall into disrepair. Floods are a hazard for water availability mainly because contaminated floodwater can enter aqueducts or wells. Food crises resulting from drought obviously entail water quantity problems.

Interventions that interrupt transmission routes include hygiene promotion (soap and utensil distribution, education, market improvements); water supply (borehole drilling, water pumping, etc.); water quality improvements at the collection point and afterwards (chlorination, provision of appropriate containers and fuel for boiling drinking water); and sanitation (latrines). Water quantity takes priority over quality: if water is scarce, people cannot wash, thus facilitating the hands route. Providing latrines for each household decreases disease transmission compared to latrines shared among many households. All these interventions have a considerable impact on diarrhoeal disease in stable settings, and this effect is likely to be greater in crises.¹⁷

Inadequate water, sanitation and hygiene also lead to higher TR of air droplet (due to poor facial and hand hygiene) and vector-borne (due to stagnant water pools) infections.

High exposure to and/or proliferation of disease vectors

Four properties of vectors influence disease control: how many there are per human being, how often they bite humans (‘c’), how efficiently they transmit or acquire the infection during bites (‘p’) and how long they remain alive and infectious (‘d’). Increases in all of the above raise the R and thus TR of vector-borne diseases, such as malaria (by far the most important), dengue, yellow fever and sleeping sickness.

Because of poor nutrition and treatment, vector-borne disease will be more severe (higher Pr and CFR) in a crisis-affected population, even if TR does not increase. However, only some crisis conditions could result in proliferation and high exposure to vectors:

- Flooding can result in vector proliferation (particularly mosquitoes) in stagnant water. However, flooding could also wash away insect larvae. Salty floodwater, as after a tsunami, may actually inhibit larval breeding.
- Shelters of people displaced into camps or host communities may be more exposed to vector bites. It may also suddenly expose them to a vector-borne disease that is not endemic to their area of origin, and to which they have no immunity, resulting in severe epidemics, irrespective of whether vector proliferation is unusual.

The main reasons for sudden vector proliferation are unrelated to crises, and have to do with climate abnormalities or changes in the environment, and whether vectors for a given pathogen can actually survive in the local environment. Vector ecology is very delicate and complex: for example, mosquitoes’ lifespan, larval cycle and incubation of malaria parasites in the gut all depend on temperature and humidity. If an increase in disease-carrying vectors is suspected, an evaluation by an experienced entomologist is useful to accurately assess risk.

Lack of and/or delay in treatment

Treatment primarily decreases the CFR of diseases. The difference between a disease's CFR with and without treatment can be dramatic: for example, severe malaria and ARI may kill about 30–50% of those affected, but this CFR can be <5% with timely treatment. Many diseases are almost never lethal (e.g. scabies), but some can nonetheless cause disabilities (e.g. limb amputation with filariasis; blindness with trachoma) that make treatment imperative. CFR usually increases with delay in treatment. Delayed care-seeking also increases the proportion of cases requiring inpatient treatment, and thus costs.

Some chronic diseases of great concern in industrialised and transition settings, such as heart disease and diabetes, require regular prophylactic (i.e. preventive) treatment: treatment interruptions due to inaccessible health facilities or drug shortages will increase their Pr.

Treating infections can also reduce TR, since it reduces the duration of infectiousness ('d'), an added benefit of prompt care. However, this TR reduction effect will probably not be substantial unless a considerable proportion of infections accesses health facilities.

Warfare among combatants and armed attacks against civilians

Whether deliberately targeted or victims of indiscriminate weapons use, civilians are frequent victims of physical and mental injuries caused by weapons, physical attacks and exposure to traumatic events. The epidemiology of war-related injuries is difficult to describe as data are hard to collect during active fighting. In International Committee of the Red Cross surgical hospitals, at least 19% to 35% of patients admitted for war injury were civilians, depending on injury cause. About 45% of injuries were due to bullets, 30% to fragments of shells, bombs and mortars and 25% to mines. These figures do not reflect patterns among casualties who never reached a hospital.¹⁸

Weaponry can range from knives and swords to small arms (handguns, shotguns) to heavy artillery and aerial bombing. Apart from Iraq, Lebanon and Sri Lanka, current wars are mainly unconventional and low intensity, and small arms predominate, causing perhaps 60–90% of global war injury deaths (see <http://www.smallarms-survey.org>). In certain wars, such as Sierra Leone, northern Uganda and Rwanda, amputations and other atrocities have been extremely common, leaving a considerable proportion of the population disabled for life. During war, the frequency of exposure to traumatic events can be shocking and underappreciated.¹⁹ In Freetown, for example, more than 80% experienced traumatic events (attacks on villages, crossfire, mine explosions, bombings, burning and destruction).²⁰ Unfortunately, little information is available from many African crises. Relief workers often arrive when relative security has been restored, or assist populations that have fled to safety. Because of communication difficulties and detachment from beneficiaries' daily life, they may not perceive the degree to which war trauma has

affected and modified people's lives. Behaviours displayed by populations traumatised on a mass scale may be explained in part by their exposure to trauma.

Notes on some important direct causes of death

From a humanitarian standpoint, reducing excess mortality is the immediate goal. The main focus is thus on diseases that are (i) life-threatening and (ii) cause considerable excess mortality now, or may do so soon if allowed to spread unchecked. Globally, most excess mortality due to crises occurs in tropical and/or very poor settings, where the following list of *immediate* causes of death usually accounts for the majority of excess avoidable deaths:

1. Acute respiratory infections (ARI).
2. Diarrhoeal diseases.
3. Maternal and neonatal causes.
4. Tuberculosis (somewhat dependent on local TB and HIV prevalence).
5. Other AIDS-defining opportunistic infections (very dependent on local HIV prevalence).
6. Malaria (very dependent on local malaria transmission).
7. Intentional injury due to acts of war (dependent on intensity and patterns of warfare).

We intentionally leave out malnutrition, measles and HIV, and instead discuss these as underlying conditions. In transition and industrialised settings, heart disease and diabetes would feature among the top direct causes.

Acute respiratory infections

Acute respiratory infections (ARI), of which pneumonia is the most common life-threatening manifestation, cause more deaths annually (about 4 million) than any other infectious disease; most ARI deaths occur among young children. ARI is certainly among the top causes of death in most emergencies in tropical and/or poor settings. Various pathogens can cause ARI, the most important being *Streptococcus pneumoniae* and *Haemophilus influenzae*.²¹ Despite scarce evidence, it appears that these pathogens infect children in the first months of life, settling in the nose and throat without necessarily causing illness. However, the pathogens can then invade the lungs, causing pneumonia, as a result of a concurrent infection (for example, with the flu), environmental risk factors or an immunity decline due to malnutrition, HIV infection or measles.

While preventing infection of the upper airways (the TR step) is important, probably the most striking effect of crises on ARI is at the level of Pr and CFR. Because most ARI is bacterial in origin, it can be treated with antibiotics. If not treated promptly, cases can deteriorate to severe ARI over days, requiring hospitalisation.

Apart from crisis, risk factors for ARI in children include Vitamin A deficiencies (Vitamin A supplements are very effective to reduce the risk of ARI among children, as they address one of the key micronutrient deficiencies in

children with insufficient nutritional intake), lack of or partial breastfeeding and exposure to indoor smoke.²²

ARI has received far less attention in crisis settings than other leading diseases. This attitude must urgently be reversed. Specifically, systematic mass vaccination of infants with *Haemophilus influenzae* type B and pneumococcal conjugate vaccines, both available, cheap and highly effective, should be considered.

Diarrhoeal diseases

These can be due to viruses, bacteria or other micro-organisms, but, as with ARI, treatment does not require identification of the causative pathogen. Some diarrhoeal pathogens cause watery, prolific diarrhoea, others bloody or mucous excretions. Diarrhoeal deaths are among the easiest to prevent: death occurs due to dehydration, following rapid fluid loss because of vomiting and diarrhoea. The primary goal of treatment is not to eliminate the infection, but simply to keep the patient hydrated until their immune system successfully fights off the infection. Patient evaluation is based on the degree of dehydration: mild or moderate cases are treatable as outpatients with oral rehydration salts (ORS), but severe cases must be referred, and receive both ORS and intravenous fluids. The greater the delay between onset and diagnosis, the harder the treatment, and the higher the CFR. Dehydration can kill within hours, especially in an already weakened individual.

Diarrhoeal diseases are mainly life-threatening for children, but epidemic shigella (bloody dysentery) and cholera also affect adults: the occurrence of severe dehydration deaths among adults indicates a possible epidemic of either pathogen. Shigella is treatable with antibiotics, but bacterial resistance is widespread, and strains must be tested for susceptibility to various drugs to optimise treatment. Rotavirus and giardia can also cause severe epidemics.

Maternal and neonatal causes

These are grouped together because they stem from common problems, and because outcomes in the mother influence the risk in the newborn child. Maternal deaths (i.e. related to pregnancy) have infectious and non-infectious causes, including haemorrhage, hypertension, infection, obstructed labour, unsafe abortion and underlying causes such as HIV infection, malaria and domestic violence or suicide directly linked to the pregnancy.

Neonatal deaths (deaths in the first 28 days of life) represent about 40% of all mortality under 5 years. Almost half are due to infection, chiefly tetanus, ARI and diarrhoea; the remainder are mainly due to premature birth and asphyxia (about one-fourth each).

Most maternal and neonatal deaths occur within the first few days after delivery, underscoring the importance of prompt access to referral hospitals. The medical journal *The Lancet* recently hosted reviews of neonatal and maternal mortality.²³

Tuberculosis

About one in three humans is infected with TB bacteria. However, only a small, vulnerable proportion develops active TB disease, mostly pulmonary. Untreated TB progresses over months and years but has a CFR of about 30–50%. Drug resistance is a major problem: treatment consists of combinations of at least three antibiotics, lasts at least six months, and should be observed. TB treatment programmes are vertical. Repatriated refugee cases are often hard to manage because treatment policies vary across countries. Interrupting treatment worsens the prognosis, and greatly increases the chances of the TB strain becoming resistant to more drugs. Chiefly for this reason, relief agencies have traditionally been reluctant to initiate TB programmes among unstable or inaccessible populations.²⁴ The evolution of TB infection is greatly influenced by concurrent HIV infection.

Malaria

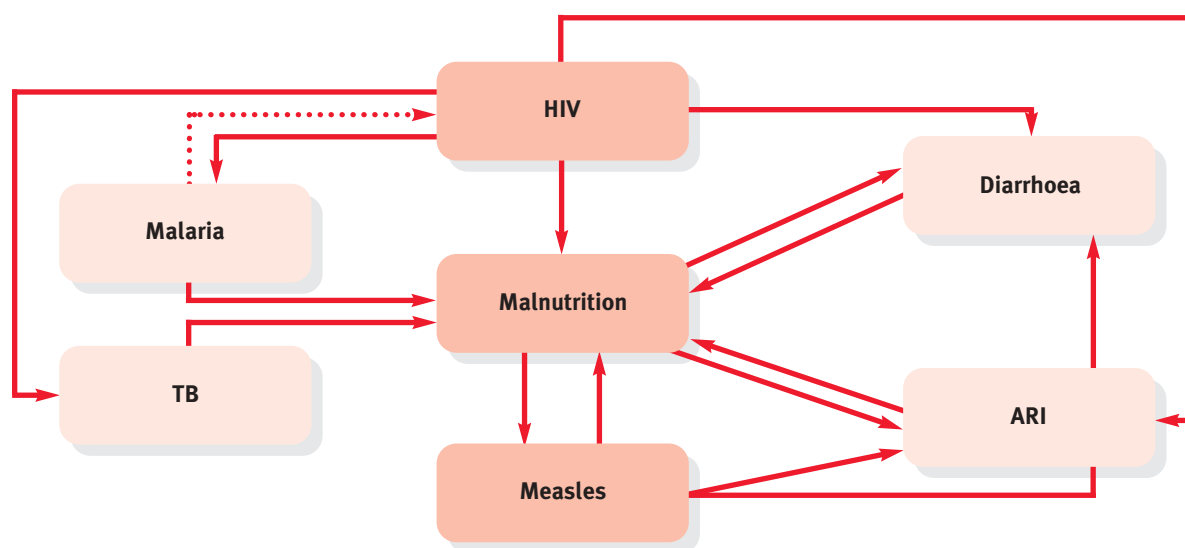
Transmitted by mosquitoes, malaria is heavily affected by climate and environmental changes. Epidemics have occurred in crisis-affected populations, causing staggering death tolls.²⁵ In malaria-endemic settings, the disease is mainly life-threatening for children, pregnant women and AIDS patients; other groups enjoy partial immunity derived from frequent exposure to the parasite (in much of sub-Saharan Africa, infection prevalence is over 50%). Where malaria is sporadic or absent, immunity does not build up with age and the entire population is susceptible. The sudden displacement of non-immune populations into malaria-endemic areas is a major epidemic risk factor. Again, early diagnosis and treatment greatly improves the chance of survival. Parasite resistance to old-generation drugs is a major issue: recently developed artemisinin-based combination therapies (ACT) are being deployed worldwide and are highly effective, though more expensive.

Intentional injury

Deaths due to intentional injuries are usually clustered in space and time, corresponding with battles and massacres. During periods of acute violence, injury deaths can dominate the mortality profile: in West Darfur, up to 93% of deaths before IDPs arrived in camps were violent, constituting a 20-fold increase from baseline mortality rates.²⁶ In industrialised or transition settings such as the former Yugoslavia, Iraq and Lebanon, most excess mortality has been due to injury. Elsewhere, the contribution of injury is smaller, although usually higher than combatants are prepared to admit. The observed ratio of fatal to non-fatal injuries ranges between 2 and 13, though it is typically around 3–4.²⁷

HIV, measles and malnutrition: killers by proxy

HIV infection, measles and malnutrition are three key underlying conditions responsible wholly or partly for many other diseases, and act similarly. The HIV virus itself does not kill anyone; the measles virus is very rarely fatal;

Figure 10**Known important interactions between underlying and immediate causes of disease and death**

.....➔ : Evidence has recently emerged that malaria may speed up HIV/AIDS progression

Note: Direction of arrow indicates cause and effect link. Note that more interactions may be discovered in the future

and malnourished children rarely ‘starve to death’. All three conditions, however, deeply compromise victims’ immune system. Immunity loss facilitates TR, results in faster and higher Pr and increases CFR. Various infections exploit this increased vulnerability (see Figure 10 for the most important relationships).

HIV infection

HIV is a subject of its own, and its role in crises has been considered elsewhere.²⁸ Knowledge and policy on HIV/AIDS are evolving rapidly, but, at the time of writing, the following points can be stressed:

- Individual risk of HIV infection can increase because of conflict and forced displacement for certain population groups (e.g. victims of sexual violence; women who survive through commercial sex). However, there is presently no evidence that conflict *per se* increases HIV TR at the population level, and the reverse may in fact sometimes hold.
- However, preventing HIV infection during crises is an extremely important activity with long-term benefits.
- HIV-positive individuals have a severely compromised immune system, and are more at risk of easily treatable infections such as diarrhoea, ARI and malaria, as well as certain opportunistic infections (e.g. cryptococcal meningitis, *Pneumocystis carinii* pneumonia, toxoplasmosis) that are almost unheard-of in HIV-negative individuals. TB is actually the most important HIV opportunistic infection, and is the leading immediate cause of AIDS death: HIV-positives have a relative risk of 6–7 of developing active TB.

- In crises, prevalent HIV infections will progress even faster (Pr) to full-blown AIDS due to risk factors that favour the TR of common and opportunistic infections, or the Pr of latent opportunistic pathogens (especially TB) which patients are already infected with. The AIDS burden will depend on the HIV infection prevalence in the crisis-affected population (thus, AIDS is a far more important underlying cause of death in the IDP camps of northern Uganda, where HIV prevalence is about 9%, than in Afghanistan, where it is probably very low).
- Most infections in an HIV-positive individual carry a higher CFR than in HIV-negatives.
- Many HIV opportunistic infections are difficult to treat, requiring drugs usually not included in standard emergency kits.
- Antiretroviral therapy has a marked reconstituting effect on the immune system; patients receiving these drugs experience fewer common and opportunistic diseases and much improved survival. So far, antiretrovirals have mostly not been offered in relief operations, for various objective or perceived reasons (need for sophisticated diagnostics and patient follow-up; fear of treatment interruptions). Operationally feasible models for delivering antiretrovirals in times of crisis need to be developed.

Measles

Measles is usually considered an immediate cause of death: 345,000 child deaths were attributed to measles in 2005, despite a cheap, easily administered vaccine (see www.measlesinitiative.org). The virus unleashes a cascade of harmful events: many cases develop ARI (about

70–80%) and/or diarrhoea, blindness and/or they become malnourished. Malnutrition in turn increases disease risk.

Measles is highly contagious: almost all non-immune children become ill if exposed to an infectious contact (high 'p', high Pr). Outbreaks thus expand very rapidly, underscoring the need to vaccinate 100% of children (usually six to 59 months, sometimes up to 14 years old) *before* outbreaks, and maintaining a high vaccination coverage thereafter. Main risk factors for outbreaks are discussed below (see Table 7). At the individual level, apart from vaccination itself, malnutrition is the greatest risk, considerably increasing CFR.

Once outbreaks start, reactive mass-vaccination campaigns can have considerable impact on transmission and thus mortality even if conducted late.²⁹ However, even the fastest campaigns will take 7–15 days to get underway, which, combined with the usual delay in detecting an outbreak, plus the time required for those vaccinated to develop antibodies to the virus (about 15 days), means the epidemic will run unchecked for at least 1.5 months. Pre-emptive vaccination is thus best.

Malnutrition

An in-depth discussion of malnutrition and nutritional crises is beyond the scope of this paper, and we refer readers to other sources.³⁰ However, the following points should be emphasised:

- About half of deaths among children under five years of age worldwide can be attributed to malnutrition, even in non-crisis conditions. This proportion is similar for the top diseases (ARI, diarrhoea, malaria).
- Insufficient nutrient intake leads to protein-energy malnutrition. Insufficient intake of certain micronutrients (e.g. Vitamin A, zinc) causes micronutrient deficiencies. Micronutrient deficiencies also increase the risk of protein-energy malnutrition, and vice versa.
- Protein-energy malnutrition can be chronic or acute. Chronic malnutrition (stunting) results from insufficient nutrient intake over a protracted period, and entails numerous long-term consequences for the child's development. Acute malnutrition (wasting) results from rapid weight loss or failure to gain weight, and is the focus of nutritional attention in crises.
- There are two manifestations of acute malnutrition: marasmus (fat and muscle depletion; children appear very thin), and kwashiorkor (fat and muscle depletion plus oedema, i.e. body swelling, due to fluid loss from tissues). Marasmus is more common, but the two can occur together. Classifications of acute malnutrition into severe and moderate are based on the weight/height ratio, oedema, and/or mid-upper arm circumference in children, and Body Mass Index in adults.
- Acute malnutrition has a profound, immediate effect on immunity, far increasing the TR, Pr and CFR of most infectious diseases, especially of diarrhoea and ARI.³¹ Severely malnourished children mostly die because of common infections such as diarrhoea, ARI, and

malaria. In turn, disease causes malnutrition due to poor nutrient intake during illness (Figure 11).

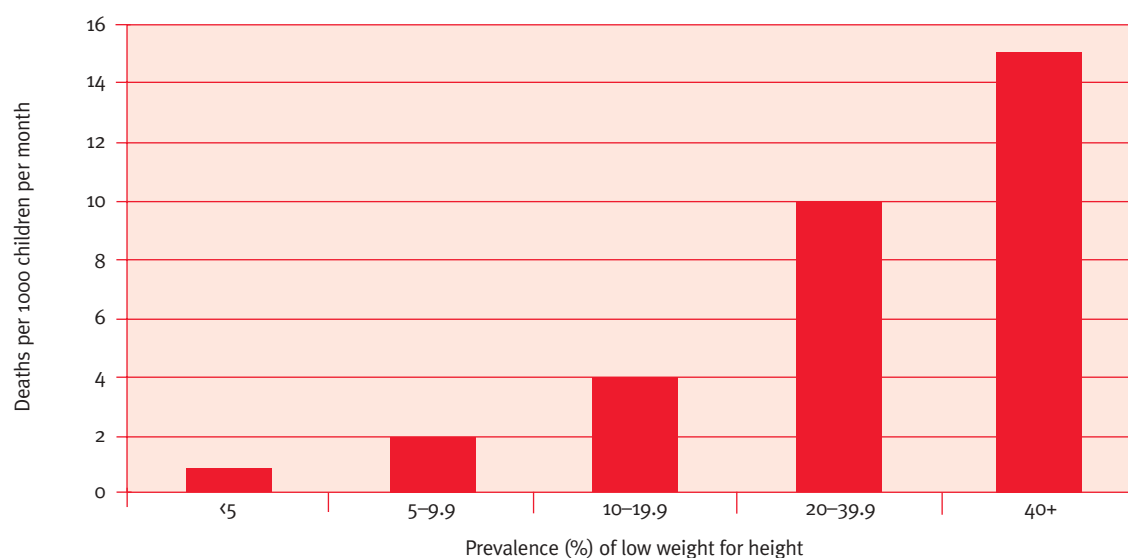
- Acute malnutrition is a 'prevalent' disease, i.e. it develops over several weeks. The more malnourished a child is, but also the *longer* a child remains malnourished, the higher the disease risk.
- Because of the above, there is usually an association between acute malnutrition prevalence and under-5 mortality. Severe acute malnutrition prevalence (SAM) and global (severe + moderate) acute malnutrition prevalence (GAM) are sensitive indicators of an unfolding emergency (GAM prevalence \geq 15% or 20% depending on agency) before mortality rates rise, and should be measured often.
- A high SAM and/or GAM already denote a failure of humanitarian relief and international protection to forestall the progression from food insecurity to nutritional crisis: preventing malnutrition through general or targeted food distributions, or other interventions, is the *sine qua non* aim of nutritional interventions in crises.
- GAM does not tell the entire story: in nutritional crises, the average nutritional status of all individuals declines – the relative risk of disease and death will increase by a lot for a few very malnourished, but also by some for many more.
- Both severe and moderate malnutrition are treatable through nutritional rehabilitation programmes. Community-based therapeutic care is increasingly the consensus approach, whereby only severely malnourished children with serious concomitant infections are treated on an inpatient basis; the remainder receive home-based care.³² Treatment relies on therapeutic foods and drugs for common infections.
- Next to children, pregnant women, HIV-infected persons and the elderly are most vulnerable to malnutrition. When conditions approach famine, malnutrition impacts on all population groups, reflected in shocking all-age mortality rates. The management of adult malnutrition is less standardised.
- Micronutrient deficiencies are less visible, and hard to measure, but they also greatly increase disease susceptibility (especially vitamin A and zinc deficiencies). 'Epidemics' of micronutrient diseases such as scurvy (due to vitamin C deficiency), pellagra (vitamin B₃ or niacin), and beri beri (vitamin B₁ or thiamine) have occasionally constituted serious public health problems in emergencies.

Epidemics versus endemic diseases

Risk factors for epidemics

During periods of crisis, morbidity and mortality may be driven mainly by epidemics, occasionally so severe that a single disease accounts for most community deaths.

Table 7 reviews the main epidemic risks as a function of crisis conditions, helping to show why entrapment and mass displacement into camps (which features simultaneous overcrowding, interruption of services

Figure 11**Correlation between mortality rate and prevalence of acute malnutrition among children under 5 years in refugee camps**

Reproduced from M. J. Toole and R. M. Malkki, *Famine-Affected, Refugee, and Displaced Populations: Recommendations for Public Health Issues*, US Department of Health and Human Services, Public Health Service, Centers for Disease Control, Atlanta, GA, 1992.

Table 7: Known main risk factors for major epidemics in crisis-affected communities

Main risk factor	Crisis condition which can produce risk factor					Main epidemic diseases of concern	Timing after onset of risk factor (time window to act)
	1	2	3	4	5		
Flooding Intense rainy season Temperature abnormalities				X		Malaria Dengue Rift Valley Fever	At least 1 month
Movement of people from non-endemic into disease-endemic region		X	X			Malaria	At least 1 month
Dry season						Meningitis	About 2 weeks
Overcrowding		X				Measles Meningitis	As little as 2 weeks
Insufficient water	X	X	?			Cholera	As little as 2 weeks
Contaminated water	X	X	?	X		Shigella (bloody dysentery)	
Very poor sanitation	X	X	?			Rotavirus	
Poor nutrient intake					X	Measles Cholera Shigella (bloody dysentery) Rotavirus	Starting about 1-2 months
Interruption of routine vaccination activities	X	X	?			Measles	A few months

Note: Much depends on whether the disease can be transmitted in the local environment

Condition 1: Progressive loss of livelihoods and deterioration of essential services, with entrapment in one's community due to the ever-present risk of violence. **Condition 2:** Mass displacement into a regimented or camp-like settlement of large population size. **Condition 3:** Displacement into neighbouring host communities. **Condition 4:** Sudden loss of livelihoods and rapid environmental change (including flooding) due to a natural disaster. **Condition 5:** Nutritional crisis.

Box 5**Comparison of Goma (1994) and Aceh (2004–2005) crises****Sudden refugee influx into Goma, Zaire (1994)**

About 500,000 to 800,000 Rwandan Hutu refugees entered the North Kivu region of then Zaire in July 1994. About 10% died within one month, mainly because of a cholera epidemic in which nearly all were probably infected, and a subsequent bloody diarrhoea (due to *Shigella dysenteriae* type 1) epidemic with at least 15,000 symptomatic cases. Several risk factors were present: GAM of 18–23%; only 1 L of purified water per person per day being available, most drank from the pathogen-infested Lake Kivu; the soil was volcanic and latrines were difficult to construct; camps were massively overcrowded; health care was vastly insufficient; and the shigellosis strain was resistant to many antibiotics.

Mortality rates from Goma remain the highest ever recorded: this relief debacle prompted the establishment of the Sphere Project.

Tsunami in Aceh, Indonesia (2004–2005)

Over 500,000 people were displaced into temporary shelters or host families across Aceh province, Indonesia, following the tsunami of 26 December 2004.

Small clusters of waterborne diseases (cholera, shigellosis, typhoid, hepatitis A and E), vector-borne diseases (malaria, dengue), measles and meningitis occurred after the disaster. However, apart from several hundred tetanus cases due to unclean wounds, no large epidemic was noted (as in previous tsunamis).

IDP settlements were relatively small (some only 20–30 people), with no major overcrowding. Food, safe water and adequate sanitation, as well as health care through mobile clinics and field hospitals, were provided promptly. The population was accustomed to hand washing and boiling drinking water, and GAM was low. The salty floodwaters did not support mosquito proliferation. The disease surveillance/early warning system (EWARN) system implemented by WHO and the Provincial Health Office in collaboration with NGOs and other partners detected and rapidly responded to potential outbreaks.

including vaccination, and insufficient nutrition) are the rifest conditions for epidemics.

Box 5 compares the situation in Goma, 1994, with that in Aceh, 2004–2005, crises with strikingly different conditions and occurrence of epidemics.

Recent epidemics in crisis settings have included several cholera epidemics following floods (Uganda, Mozambique, India, Malawi), influenza in DRC, measles in Afghanistan, DRC and Ethiopia, meningitis in DRC, and malaria in Burundi.³³

Contrary to popular belief, natural disasters carry a far lower (though not negligible) risk of epidemics than man-made crises. The list of epidemic-prone diseases that displacement or entrapment can favour is considerably longer than for natural disasters (Table 7), of which flooding is the main concern. A particular misconception, which never fails to circulate when a major disaster strikes, is that dead bodies and animal carcasses entail a high disease risk. In fact this risk is small, although disposal of dead bodies is important for mental health reasons.³⁴

Greater endemic burden

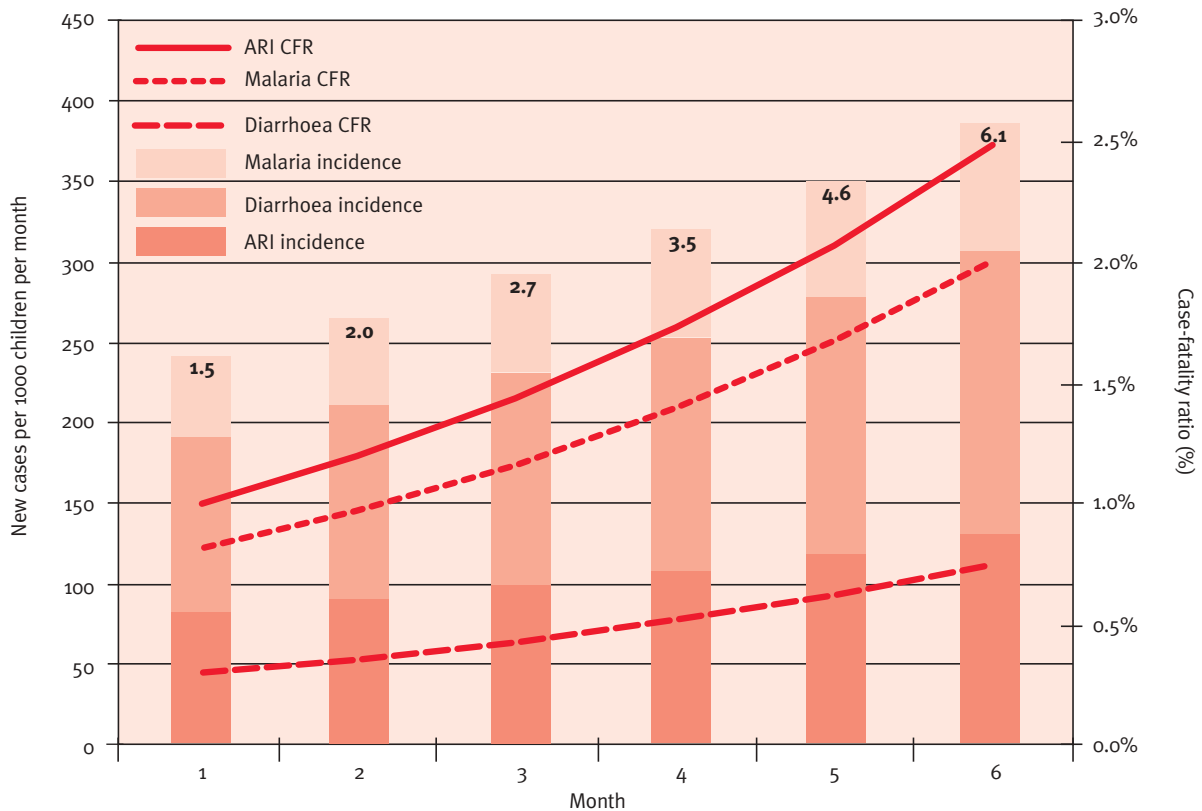
The absence of (detected or detectable) epidemics does not mean that there is no health emergency. Many risk factors will result in an increase in IR, Pr or CFR for a variety of endemic diseases. For example, insufficient water and sanitation will increase the risk of all faecal-oral diseases. While the increase in the burden of each such infection may not constitute an epidemic, altogether they can sum up to a staggering burden (Figure 12) that deserves as much attention as a single-disease epidemic, and should *not* be treated as routine.

Epidemic versus endemic diseases: which should we fear most?

In 2005, a widely publicised outbreak of Marburg haemorrhagic fever affected Uige province of Angola, killing about 300 people. The Marburg virus is normally carried by animals, but rare human outbreaks occur when people come into close contact with sick animals. Marburg has a very high CFR (about 80–90%), and there is no cure. At close range, it is extremely contagious, requiring strict isolation. Its R_0 , however, is probably low like that of Ebola, and cannot sustain widespread transmission in humans. Nevertheless,

Figure 12

Illustration of the greater incidence and lethality (CFR) of endemic diseases as a result of crisis



The graph shows a hypothetical, slowly unfolding crisis.

Month 1 shows the pre-crisis baseline. As conditions progressively deteriorate starting in month 2, the incidence of common, endemic childhood diseases such as ARI, diarrhoea and malaria increases due to a variety of proximate risk factors.

The CFR of all three diseases also gradually increases. The two effects combined (higher incidence and higher CFR) will result in considerable excess mortality, even in the absence of an epidemic. Numbers in bold show the mortality rate due to ARI, malaria and diarrhoea combined (as deaths per 1,000 children per month) obtained by multiplying incidence x CFR for each disease, and summing the three products. By month 6, the relative risk of dying compared to month 1 is about four-fold (=6.1/1.5).

the outbreak struck fear in the local population and attracted to Uige more than 100 international experts.

In 2006, Angola was struck by two countrywide epidemics of cholera, with 2,900 reported deaths as of April 2007. Cholera is an easily treatable and preventable disease, but this outbreak attracted far less international support, and was hardly mentioned in the international news. A handful of agencies battled it alone.

The point here is not to steal attention from eye-catching diseases such as Ebola, Marburg and SARS, but rather to highlight two grossly overlooked points:

- Perception of relative disease importance often drives resource allocation, but is itself driven by how easily

detectable some epidemics are. Many epidemics certainly go entirely unnoticed, especially in the early, chaotic emergency phase. This happens because no one is there to witness the epidemic; because necessary diagnostic tools are not available and/or because disease symptoms are non-specific (e.g. rotavirus can currently only be distinguished from other diarrhoeal diseases through tests in referral laboratories); and because agencies do not realise the potential risk of certain diseases, and fail to set up even minimal surveillance as soon as possible.

- Many endemic diseases carry a far higher burden of morbidity and mortality than some much-feared epidemic diseases, and yet are often dramatically neglected during emergency responses. ARI and neonatal infections are prime examples.

Mental illness: overlooked but omnipresent

Mental illness is a leading cause of disability worldwide, causing a greater disease burden than cancer or heart disease. Anxiety disorders (including PTSD) are the most common mental illness (global prevalence 2.4% to 18.2%), followed by mood disorders (0.8% to 9.6%), substance disorders (0.1%–6.4%) and impulse-control disorders (0.0%–6.8%). Nearly 800,000 commit suicide annually,³⁵ and 76.3% to 85.4% of cases in less-developed countries receive no care.³⁶

The prevalence of mental illness³⁷ is consistently elevated in crises, roughly two to four times that in stable settings.³⁸ In post-war Afghanistan, more than two thirds showed signs of depression and other mental illnesses. Prevalence of PTSD alone can range between 20% and 30% even years after wars end.³⁹ The more violent trauma individuals are exposed to, the higher their risk of mental illness.⁴⁰ Specific sub-groups may be particularly affected, and feature specific types and severity of conditions. Children, mothers and the elderly may have different coping mechanisms and progression of illness than others: mental health assessment within these groups should be an early intervention. The impact of mental illness on general health cannot be overstated. PTSD and depression may present as apathy, disregard for family members, suicide and unexplained early death.⁴¹

Measuring mental illness within disparate populations is challenging. Questionnaires specific to developing countries have yet to be developed; researchers use tools developed for Western societies (for example, the Harvard

Trauma Questionnaire), but there is little consensus on their applicability to non-Western settings.⁴²

Predicting the extent and patterns of mental illness in different crisis conditions is difficult. In entrapment settings, populations may have suffered more torture or severe physical or mental injuries, causing long-lasting effects on mental health.⁴³ But displaced populations may suffer elevated depression and PTSD due to the squalor of camp life or poor expectations of their future. Among Bhutanese refugees in Nepal, camp life increased suicide risk.⁴⁴ The association between intensity of trauma and mental illness is not obvious: certain cultures can effectively deal with mental trauma through coping mechanisms such as religion, meditation and education.⁴⁵ Victims of sexual violence may have severe mental illness, but these cases often go unrecognised because of stigma.

Interventions to prevent and treat mental illness in crises have been few and are poorly documented. The evidence base is slim, especially for crisis settings. Aid agencies may initiate immediate debriefing interventions and ad hoc counselling, but there is not yet compelling evidence to provide these services and some studies suggest they may be harmful.⁴⁶

Population-wide interventions such as community-based counselling are an area of considerable attention, but also lack evidence. Population-wide interventions or the integration of mental health in other basic health services require coordination among agencies and the development of culturally specific and acceptable interventions.⁴⁷

Chapter 4

Determining priority health problems and interventions

Which health problems?

General points

The process of prioritising health problems to be tackled should occur systematically. We distinguish between two phases: the onset of a new crisis, necessitating a new relief response; and the monitoring of an evolving crisis after relief has arrived.

Following Chapter 3's framework, questions to ask when first hearing about a new or ongoing crisis could be:

- Which of the three demographic and epidemiologic settings is the crisis taking place in? Is any health crisis going to be driven mainly by infectious or non-infectious diseases?
- Which of the five crisis conditions are present?
- Is there any information on the extent to which the main proximate risk factors are present?
- In this setting and these crisis conditions, how will these risk factors generally affect the TR, Pr and CFR of the various classes of infectious and non-infectious diseases?

An estimate of the size of the population that is being targeted either for exploratory assessment or for intervention is a further necessary starting point. Methods to estimate population size are reviewed elsewhere.⁴⁸

The above provides at least a frame of reference for short-listing likely health problems to consider. For example, the 2006 Israel–Hezbollah war in Lebanon took place in a setting with residual infectious disease threats, especially among children, but mainly chronic disease problems, including mental illness due to trauma. Displacement into neighbouring communities or very small camps was the main crisis condition, though non-displaced communities in the south experienced short-lived entrapment conditions. The main proximate risk factors in the immediate term appeared to be lack of and/or delay in treatment, and warfare itself (especially bombing), but a food crisis was possible within weeks in the trapped communities of the south if relief was not allowed through.

The next step is to identify and rank existing health problems as well as imminent threats, so as to select and prioritise interventions.

As soon as relief arrives, prospective surveillance becomes a critical tool to identify emergent health problems, and to adjust the relative weight of different interventions to address any observed gap.

Onset of a new crisis: epidemiological risk assessment

A systematic assessment of disease risk (*risk assessment*) is necessary to guide interventions designed to mitigate this increased risk (*risk management*). The risk assessment process reviews available evidence and expert opinion to predict both the likelihood and present or potential impact of specific diseases. Here we describe, as an established example, the risk assessment approach of the WHO Communicable Diseases Working Group on Emergencies (CD-WGE).⁴⁹

In response to a new crisis, the CD-WGE meets urgently to systematically review available data (including outbreak notifications, relevant Ministry of Health data from affected countries, published literature, reports and surveys) in order to better define the risk of infectious diseases and generate risk profiles for the crisis (see examples at http://www.who.int/diseasecontrol_emergencies/toolkits/en/).

The infectious disease risk assessment consists of three steps:

1. Event description is the process of systematically assessing the type of crisis and the characteristics of the population affected. This is analogous to considering the crisis conditions present (see above).
2. Threat/vulnerability assessment identifies potential interactions between the emergency-affected population (host factors), likely pathogens (agents) and exposures (environment) that determine the presence of risk factors for infectious disease. An assessment of the likely pathogens and vectors is critical. Both endemic and epidemic-prone diseases, as well as any disease control programmes operating in the area, are considered in terms of incidence, prevalence, usual CFR in similar populations, seasonality and history of recent outbreaks.
3. Risk characterisation combines the above information for each disease into a matrix grading both the potential magnitude of the health impact and the likelihood of the event occurring (Box 6).

The above exercise can apply for any disease, not just infections. For example, circumstantial evidence could indicate that intentional injury is an expected major cause of morbidity and mortality, or that levels of exposure to violence (for example, reports that many newly arrived refugees have been raped) are consistent with a high frequency of PTSD, which would urgently need to be addressed. Risk assessment also enables crude calculations of drugs and supplies for case management.

Data sources include:

- Evidence about the morbidity profile of similar past crises (e.g. other mass displacement crises in tropical or poor settings).
- Maps of the worldwide distribution of given diseases (e.g. one would not consider dengue fever a risk in the Middle East since the vector mosquito is absent).
- An online keyword search for academic papers on given diseases in the country of interest (e.g. through Google Scholar or PubMed: <http://www.ncbi.nlm.nih.gov/sites/entrez?db=pubmed>).
- A global dataset of past epidemics provided by Promed (<http://www.promedmail.org/pls/promed/>).
- Yearly/quarterly epidemiological digests from the surveillance branch of the local Ministry of Health, going back a number of years.
- WHO databases and archives.
- Relief agency situation reports.
- Media reports.

The aim at this stage is not to accurately predict the occurrence of each disease, but to deploy a response that combines both preventive and curative interventions against the likely major current and possible threats. A very in-depth assessment would be too time-consuming and might not yield a justifiably greater amount of certainty.

After the onset: epidemiological surveillance

Data from health structures serving the affected population is useful, but not necessarily for setting initial priorities. Rather, it should become part of a surveillance system, to be implemented *as soon as possible* (i.e. days, not weeks) after a relief operation gets underway.

In crises, the main purpose of *surveillance* is to detect epidemics with minimum delay. The implementation of surveillance systems requires expert coordination and is beyond our scope. However, all such systems consist of the following components:

- A *surveillance network*, consisting of actors involved in health services delivery (government, NGOs etc.), that shares information informally and reports morbidity data regularly: often, all facilities will report (*exhaustive surveillance*); sometimes, only a representative sample (*sentinel surveillance*) will be included in the reporting system, so as to lessen workload and enhance quality by concentrating on a few 'sentinel' facilities only.
- A *list of diseases* that each facility must report on: this should be short and essentially determined by the risk assessment; there is usually a breakdown by age groups and gender, on standardised forms especially provided.
- Standardised *case definitions* for each disease in the list (e.g. suspected cholera = acute watery diarrhoea in a person over 14 years old). A balance must be struck between definitions that create too many false alerts (i.e. insufficiently specific), and definitions that result in missed or delayed outbreak detection (i.e.

insufficiently sensitive; see Chapter 5 for definitions of sensitivity and specificity): generally, false alerts are the lesser evil. Definitions may vary by type of health facility (e.g. in a hospital with a laboratory, the malaria definition might be 'malaria symptoms with a positive blood slide'; in a health post, 'fever with no other evident causes of infection').

- A *mode of reporting*: active if surveillance staff call on facilities to obtain the data (this is done routinely for polio), and *passive* if facilities send reports without prompting; rarely (in dramatic epidemics), surveillance for a specific disease (e.g. cholera) occurs on a community basis, i.e. community health workers visit households daily, enumerating and triaging possible cases.
- A set *frequency of reporting and analysis* (daily during a severe epidemic of an acute disease such as cholera; weekly otherwise).
- A *reporting mechanism* (e.g. health posts hand-carry to health centres which fax to referral hospitals; NGO clinics e-mail to WHO sub-office).
- *Alert and epidemic thresholds*, which specify not just the definition of an event (e.g. '≥3 bloody diarrhoea cases from site X during week Y = suspected cluster of shigellosis cases') but also action needed (e.g. 'if one or more cases of measles reported from site X during week Y, send a team to investigate, trace contacts, etc.').

The main outputs of surveillance systems are trends in incidence over time, and ad hoc investigations of alerts and confirmed outbreaks. Mortality excepted, it is almost never necessary for surveillance to capture all events; rather, what matters is being able to monitor trends over time. Thus, collecting data from health facilities only (which rarely reflects true incidence, since only some cases access health care) is perfectly reasonable. However, data from health facilities can be skewed:

- Inpatient structures see more severe conditions, hence eliminating 'background noise' due to common diseases with similar symptoms: a malaria epidemic might first be detected via rising hospitalisations for severe malaria; any pandemic influenza epidemic might first become evident in hospitals (elsewhere the disease might be indistinguishable from common flu).
- Outpatient structures provide information on the range and relative importance (*proportionate morbidity*) of diseases in the community, but only if they are well attended, and if people seek care for each disease proportionately (e.g. communities may prefer traditional practitioners for certain conditions); these biases are insufficiently appreciated, especially when relief workers fail to explore the community.
- Community health workers may capture a more representative breakdown of proportionate morbidity, but their diagnostic capacity is generally limited, and may skew data (e.g. they may systematically classify most ARI as malaria).

Aside from surveillance, some ongoing risk assessment must be maintained, especially in protracted crises. For

Box 6**Risk assessment matrix for an infectious disease****Likelihood of disease occurring:**

- High (3+) Endemic disease with potential for epidemic transmission clearly present
Widespread exacerbation of conditions favourable for infectious disease transmission
Highly susceptible population
- Moderate (2+) Endemic disease with potential for epidemic transmission present
Some exacerbation of conditions favourable for infectious disease transmission
High background levels of immunity but large number of susceptible people
- Low (1+) Endemic disease with potential for epidemic transmission possibly present
Conditions favourable for infectious disease transmission possibly present
Population largely immune

Potential magnitude of health impact:

- Severe (3+) High morbidity/mortality
- Moderate (2+) Increased morbidity/mortality
- Low (1+) Minimal morbidity/mortality

Potential magnitude of health impact	3+			
	2+			
	1+			
	0			
		1+	2+	3+
	Likelihood of disease occurring			

Resulting classification

Code	Classification
	Very high risk (3+)
	High risk (2+)
	Low risk (+)
	No risk (-)
	No information (N)

example, a flow of new arrivals into a camp, heightened insecurity or the emergence of new epidemic risk factors should be included in epidemic intelligence, resulting in an updated risk profile and supplemental interventions.

Which interventions?***Vertical versus horizontal, preventive versus curative***

Existing guidelines already provide detail on specific interventions needed to address the major identified risks of excess morbidity and mortality. Here we merely suggest

broad rules for selecting and prioritising interventions based on quantitative and qualitative considerations.

Some interventions are *vertical*, meaning that they are entirely devoted to one disease and exist in parallel to routine health services; others are *horizontal*, meaning that they are integrated in existing health services and target several diseases simultaneously.

Furthermore, interventions are either *preventive* or *curative* (some are both). Preventive interventions mainly

reduce TR and Pr, i.e. incidence, although they sometimes have an intended or unintended effect on CFR. Curative interventions reduce CFR.

Debates around intervention choices often dichotomise preventive and curative interventions (e.g. vector control and case management for malaria), but both are indispensable. These dichotomies may arise simply because the bar is set too low: in wealthy countries, no government would deny citizens the combined benefits of prevention and treatment. In poor countries and especially crises, health targets are usually underwhelming. Even where Sphere standards are adhered to, it should be recognised that they reflect *minimum* targets, and that exceeding these is no waste of resources.

Curative interventions are indispensable as diseases cannot always be prevented. Furthermore, for some diseases the mainstay of intervention is curative (for example, TB) or effective preventive interventions are unavailable (e.g. the BCG vaccine confers limited protection against TB). Curative interventions can also reduce TR. Various levels of health care provision exist:

- Community health workers' functions can include screening and referring cases, diagnosing a few diseases through simple case definitions, and administering treatment at the household.⁵⁰
- Primary health care involves outpatient treatment of a few high-burden diseases at basic health posts or centres, usually diagnosed without laboratory work and based on simple case definitions. Preventive interventions such as health education, EPI, and antenatal care also take place. Most patients are seen here.
- Secondary health care entails inpatient care of severe or other cases that could not be managed elsewhere (e.g. because of insufficient drug range).
- Tertiary health care involves large district or regional hospitals with specialised services (e.g. orthopaedics, gynaecology, advanced diagnostics, surgery). Few cases are seen here, but many would die or remain disabled without this level of care.

Referral facilities being overwhelmed with severe, very advanced cases can mean any of the following: (i) insufficient hospitalisation capacity; (ii) a failure to manage cases promptly at the primary health care level; and (iii) the success of referral procedures.

Preventive interventions tend to be time-bound, requiring regular re-administration (e.g. mass deworming might take one week and need to be repeated annually). Hence, they are often cheaper than curative interventions. They also relieve pressure on health structures by reducing the disease caseload, a key point underscoring their importance.

Criteria for selecting optimal interventions

Diseases may be grouped according to proximate risk factors (unsafe water or overcrowding, for instance) to

facilitate choosing interventions. General criteria for this choice are suggested:

- Potential for addressing the main health problems:
 - Interventions should address diseases identified in the risk assessment.
 - Interventions should be prioritised to address the high risk diseases first.
- Feasibility:
 - Implementing the intervention should theoretically be feasible given local logistics, human resources, security, etc.
- Maximum *opportunity benefit*:
 - Interventions should have the potential to target several diseases at once (e.g. water and sanitation; primary health care).
 - Interventions against one disease only (e.g. vector control against malaria; hepatitis A vaccination) should be prioritised if the disease is classified as very high risk, but not otherwise.
 - Interventions should if possible be synergistic (e.g. safe water + sanitation + hygiene promotion result in an overall impact that is more than the sum of each intervention's impact).
- Minimum *opportunity cost*:
 - Interventions should neither exclude nor delay the implementation of another priority intervention, and the opportunity cost of doing so should be considered.
- Maximum *effectiveness*:
 - The intervention with the greatest expected effectiveness should be selected (e.g. the better of two types of latrine).
 - When a package of interventions against a disease exists (e.g. water + sanitation + hygiene promotion + early rehydration against diarrhoea), the whole package should be implemented, not just one component.
- Maximum *cost-effectiveness*:
 - Interventions should entail the lowest cost possible for the greatest possible outcome/impact.
- Timeliness:
 - Preventive interventions should be implemented before the potential occurrence of an outbreak or increase in the burden of any disease.
 - Interventions against a specific epidemic disease should only be deployed after the onset of an epidemic if they are proven to reduce morbidity and/or mortality even after an epidemic starts. Examples are water and sanitation for diarrhoeal diseases, mass vaccination for meningitis, mosquito control and use of ITNs for yellow fever, and isolation for viral haemorrhagic fevers.
 - Other interventions should only occur in the time window during which they are likely to have an impact. For example, indoor residual spraying must be completed before the malaria season starts if it is to reduce TR. Oral cholera vaccine should not be implemented once an outbreak has started: the epidemic will usually run its course by the time the

campaign gets underway and those vaccinated build up immunity.

There is no easy formula to balance the above: all parameters must be considered simultaneously. However, some compromises are unacceptable. If no intervention can be selected that will enable reaching the corresponding Sphere standard, either humanitarian space or resource allocation must be insufficient. Instead of resignation, advocacy is needed to secure these, while implementing the minimum response possible.

Notes on some of the above criteria

Opportunity benefit is the saving inherent in deploying interventions together. Horizontal curative interventions (e.g. primary health care) tackle many diseases simultaneously and thus carry a high opportunity benefit. Preventive interventions targeting proximate risk factors will often do likewise: for example, providing adequate quantities of safe water will tackle many diseases at once (cholera, shigellosis, rotavirus, typhoid fever, etc.). Opportunity benefits increase cost-effectiveness (see below; for example, coupling two vaccines increases the cost-effectiveness of both).

Opportunity cost is the cost of foregoing or delaying an intervention so as to implement another intervention. For example, cholera vaccination, which requires two doses administered one week apart, could monopolise human resources for hygiene promotion or divert resources from water and sanitation interventions that would avert, not just cholera but other faecal-oral diseases.

Cost-effectiveness is an economic concept that considers the ratio between inputs (quantified financially) and the intervention's outcome/impact. For example, the cost-effectiveness ratio of voluntary counselling and testing for HIV in Africa is about \$450 per infection averted, while condom distribution plus sexually transmitted disease treatment for prostitutes costs about \$15 per infection averted.⁵¹ If two alternative interventions are equally effective, the more cost-effective is usually chosen. However, what if an intervention is more cost-effective than another, but less effective overall (for example, an antibiotic for ARI that costs \$0.50 and treats 80% of cases is more cost-effective than one which costs \$3 and treats 90%?). If resources are constrained, one might choose the less effective but more cost-effective intervention, which will enable high coverage. However, such cruel choices should not obfuscate the fundamental problem, namely that most crisis-affected populations do not receive the same standards of service that people in wealthy countries expect as a given.

The top ten interventions

The following is an internationally recognised list of ten key interventions to reduce mortality in the acute phase of an emergency and beyond, in no order of priority:

1. Adequate and appropriately spaced and sited shelter.
2. Sufficient and safe food.
3. Sufficient and safe water.
4. Adequate sanitation facilities.
5. Environmental sanitation and waste disposal.
6. Mass vaccination, with measles vaccine as the first priority, and a restart of routine EPI vaccination in primary health care centres as soon as feasible.
7. Access to primary health care and referral hospital services for treatment of severe cases.
8. Disease surveillance, outbreak preparedness and control.
9. Vector control.
10. Health education and social mobilisation.

Most are preventive. The first five are universal and minimum standards (e.g. Sphere) exist: they should not be questioned without scientific evidence. The last five require adaptation to the local context, and are informed by the risk assessment exercise.

- Risk assessment can inform vaccination strategies. For example, if measles coverage is >90% and the setting is not a camp, a mass campaign is unwarranted and efforts can be directed towards routine EPI. If a mass campaign is deemed necessary, risk assessment can suggest the opportunistic inclusion of other vaccines. During the Horn of Africa floods of 2006, given the ongoing poliomyelitis transmission in the area, oral poliovirus vaccine was added to measles vaccination and vitamin A supplementation. National protocols and ongoing outbreaks must also be considered when selecting vaccines to administer. For example, during the 2006 Israel–Hezbollah war, a combined measles-rubella vaccine was used, taking into consideration the vaccination schedule in Lebanon (which included MMR at 13 months and at 4–5 years).
- Disease surveillance/outbreak preparedness and control measures should target priority diseases identified in the risk assessment. Disease-specific outbreak preparedness measures include writing response plans for each priority disease, outlining prevention and control strategies, assigning roles and responsibilities, stockpiling appropriate drugs and supplies, identifying laboratories for diagnostic confirmation, updating standard treatment protocols and ensuring their use in health care facilities, reinforcing infection control precautions and identifying potential isolation wards.
- Vector control techniques will vary based on local entomological conditions.
- Health education will consist of generic hygiene-related messages, but risk assessment may result in messages specific to certain diseases being developed. Expectation of potential outbreaks helps in preparing community messages and pamphlets in advance.

The order of priority of interventions changes with time and can be tiered. For example, considerable infrastructure needs to be in place before implementing a vertical TB programme (e.g. primary health care for detection and laboratory for diagnosis). Similarly, promoting hand washing is useless if people have extremely little water.

Chapter 5

Maximising impact

A framework for interventions

Steps in the intervention cycle

Box 7 shows a typical framework used to conceptualise the various steps in the intervention flow, and establish a monitoring and evaluation strategy. Inputs are the goods and human resources fed into the intervention. Processes are the actions whereby the service is actually delivered to beneficiaries. Outputs are the immediate results of the services, which then become positive health outcomes (reduction of disease risk) and impacts (reduction of mortality risk).

The key quantities: coverage and effectiveness

Coverage and effectiveness are the two key epidemiological quantities determining interventions' impact. Definitions vary: ours merely serve our purposes. Roughly, coverage is the proportion of individuals in need of an intervention who actually get it. Effectiveness is the proportion of individuals who, having received the intervention, experience the intended positive health outcome or impact. Within our

intervention framework, coverage is the proportion of targeted beneficiaries who receive the intervention's output, whilst effectiveness determines the outcome and impact, conditional on the achievement of this output.

Coverage issues

The coverage concept is straightforward but risks oversimplification. First, coverage definitions used in crisis monitoring reports are often very crude. For example, IDPs may be simplistically considered to have access to water if their camp has protected water points, or a community's health care needs may be considered covered simply because it has a clinic. This is fallacious not only because the clinic's effectiveness may be very low, but also because a clinic's physical presence does not guarantee fulfilment of objective needs. Instead, any coverage statement should refer to a specific intervention and quantitative target (e.g. not simply 'water and sanitation', but rather 'at least 15 L of water per person per day' or 'improved pit latrines at a ratio of no more than 20 persons per latrine'). The resulting picture may be sobering, but more realistic.

Box 7

Steps in the intervention flow, with examples of activities/events corresponding to each

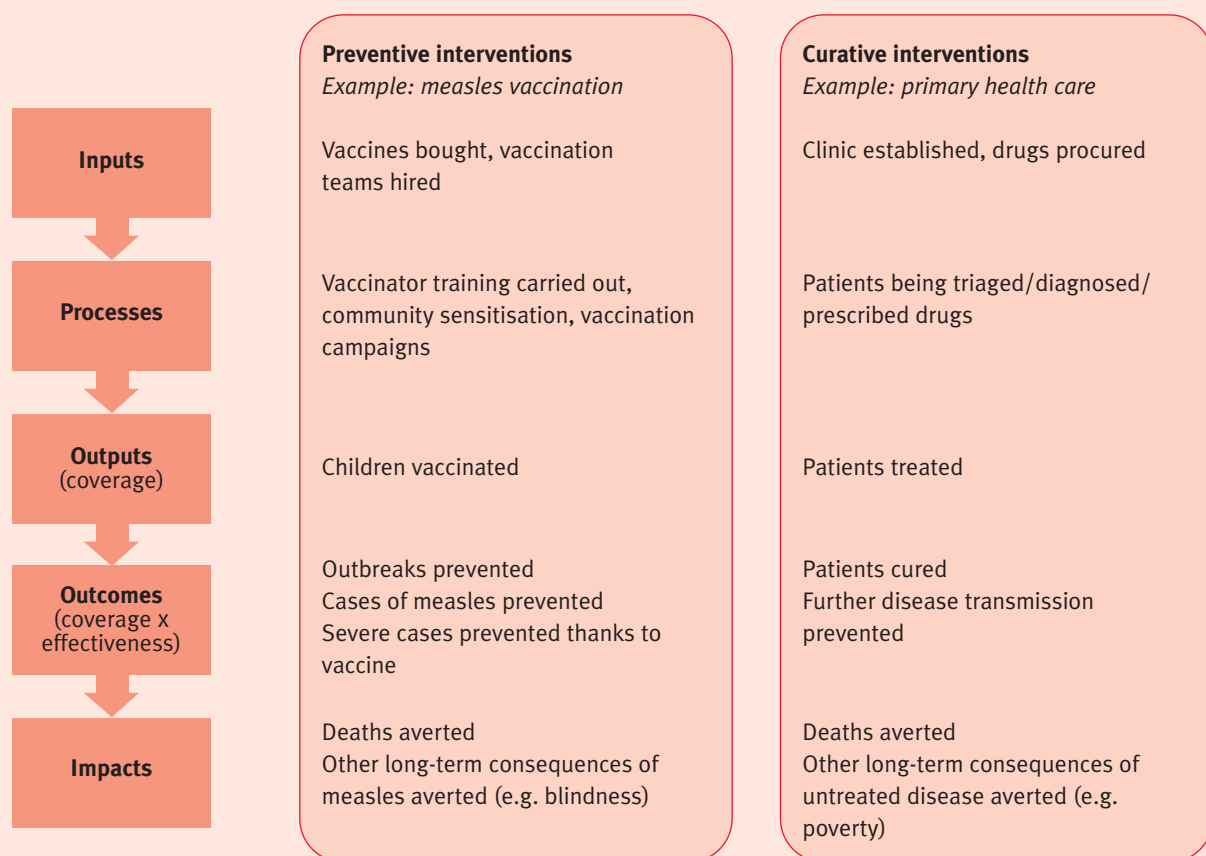
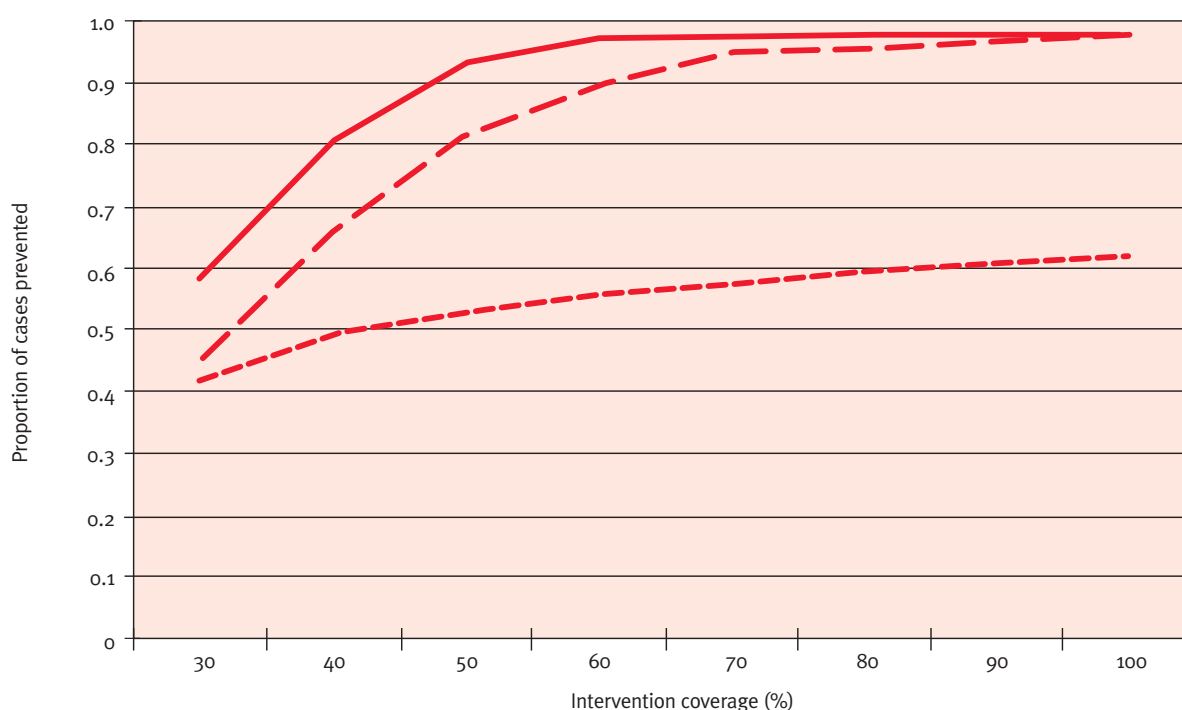


Figure 13**Predicted impact of measles vaccination⁵²**

Note: The vaccination intervention targeted children aged six months to 15 years for a vaccination intervention lasting ten days. Solid line = intervention at 60 days, large dashes = 90 days, short dashes = 120 days.

Second, the assumption that one unit of output will result in one unit of outcome/impact is usually incorrect. The relationship between coverage and impact is rarely linear, but instead governed by ‘threshold effects’, whereby a considerable impact is only achieved once a given coverage is reached. This has mainly to do with individual versus community risk: consider the herd immunity example given above. Similarly, if 50% of dwellings in a camp are sprayed with insecticide, 50% of individuals will be somewhat protected against malaria. If 80% of tents are sprayed, this could in some settings be enough to reduce ‘c’ and thus R below 1, thus causing a decrease in TR not only among protected individuals, but among the unprotected as well.

Third, the timing of coverage matters greatly. For example, Grais et al. have predicted the impact of measles vaccinations following an outbreak, showing that the proportion of cases averted declines with delays between outbreak onset and vaccination (Figure 13). Notice here too a non-linear coverage–outcome relationship.

Effectiveness issues

Effectiveness, like coverage, must be defined explicitly for each intervention. A statement such as ‘the meningitis vaccine is 80% effective’ means little unless one also specifies the population group for whom this effectiveness has been demonstrated, and the indicator of health effect (outcome or impact). Published evidence often contains

different effectiveness definitions for the same intervention. The definition adopted depends on how ‘downstream’ the effect to be observed is, and how easily measurable it is. For example, when evaluating indoor insecticide spraying of shelters to prevent malaria, one could define the intervention’s effectiveness as:

- Reduction in the prevalence of malaria infection in the general population (outcome).
- Reduction in the incidence of clinical malaria episodes among children under 15 years old (outcome).
- Reduction in under-five mortality due to malaria (impact).
- Reduction in child mortality due to all causes (ultimate impact).

While all are theoretically correct definitions, the choice depends on the questions, ‘effective against *what?*’, ‘effective in *whom?*’, and ‘can it be measured?’.

Most evidence on interventions comes from randomised trials, in which a group of people is allocated the proposed new intervention, and a comparable group (the control) receives either nothing or the current standard intervention.

Most trials are conducted in ideal conditions, meaning that state-of-the-art techniques and conditions are used to deliver the intervention. This might involve supervising every injection of a vaccine being tested, ensuring that

households know how to use the new type of bed net, diagnosing trial patients with the best available tests and supervising the treatment to ensure that all doses are correctly administered. Such trials produce a measure of *efficacy* (or *cure rate* if treatments are being tested), which reflects how the intervention will achieve its intended outcome/impact in ideal study conditions. Once interventions are implemented in real-life conditions, we speak of effectiveness. Effectiveness in field conditions is almost always lower than efficacy in studies.

Issues with (i) the field quality of the prevention tool; (ii) its acceptance, understanding and utilisation by beneficiaries; and (iii) its durability in field conditions contribute to effectiveness being lower than measured efficacy. For example:

- A small break in the cold chain might reduce the effectiveness of certain vaccines.
- If a certain model of latrine is built for a community that is not familiar with it, utilisation might be low or inappropriate.
- Routine wear and rats might tear bed nets, allowing mosquitoes to penetrate them.

In general, the efficacy–effectiveness gap will be even greater in crises. Pre-empting such losses in effectiveness, for example through quality assurance management, is very important.

Imagine a network of primary health care clinics, a community ARI treatment programme, or an emergency rehydration intervention to minimise CFR during a cholera outbreak. When designing and monitoring such curative projects, it is useful to not consider the treatment alone, but rather the entire ‘package of care’, from when patients enter the health structure to when they leave, and beyond this to the days during which they are supposed to take the drugs. Different sub-steps comprise this package of care:

- Diagnosis and assessment of disease severity (which sometimes includes a triage point at the clinic entrance).
- Prescription of treatment.
- Dispensing of the treatment, including an explanation of how to take it.
- Adherence to (or compliance with) the prescription by patients or caregivers (for outpatients) or by nursing staff tasked with administering it (for inpatients).
- Outcome of the treatment (cure versus continued and/or worsened disease).

At each sub-step, opportunities for appropriate care can be missed. We illustrate this in Figure 14, where we hypothesise that, at each sub-step, 80% of patients receive appropriate care.

The diagnosis step merits particular attention. Diagnostic accuracy will either rely on purely clinical signs and symptoms, or be aided by a laboratory test. The succession of clinical and/or laboratory tests that determines diagnosis is the *diagnostic algorithm*. Two possible diagnostic algorithms for malaria are provided in Table 8. Algorithm 1 could be performed by very unskilled health workers, and will capture the near-entirety of malaria cases, i.e. its *sensitivity* approaches 100%. However, it will also diagnose other febrile diseases (e.g. flu, ARI) as malaria, i.e. its *specificity* will be poor. Algorithm 2 is more resource-intensive, and will miss quite a few cases because some malaria patients have fever $<38.5^{\circ}\text{C}$ and undetectable parasite levels. This algorithm’s sensitivity will be $<100\%$. However, its specificity will be fairly high, as other febrile illnesses will seldom be misclassified as malaria given the criteria (high fever, positive blood test, rule out clinically any other febrile disease).

There is almost always a trade-off between high sensitivity and high specificity. Striking a compromise depends on various considerations:

- How deadly is the disease if left untreated? High sensitivity will minimise cases missed and thus CFR.
- How safe is the treatment? High specificity will minimise the prescription of toxic treatments to patients who do not need them.
- How expensive or complex is the treatment? Specificity will ensure that treatment is only administered to those who need it.
- Does over-treatment favour the development of drug resistance? If this risk is high, specificity should be maximised.
- How expensive or complex is the diagnostic algorithm? Going for simple tests might reduce sensitivity and/or specificity, but allow for much larger caseloads to be processed.

In field practice, especially in crises, the first consideration understandably overrides the others, i.e. sensitivity is generally privileged over specificity.

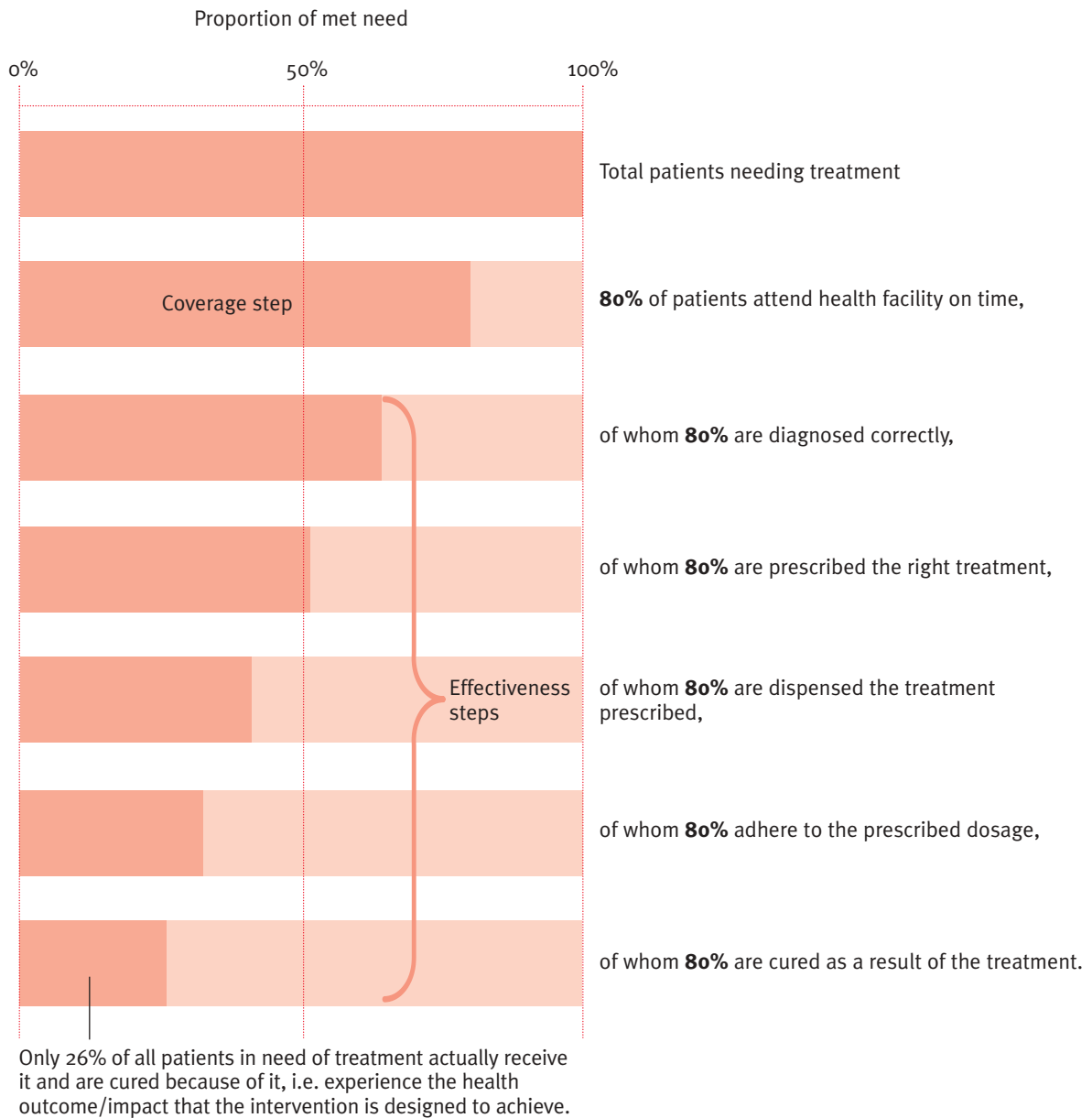
Simple, high-sensitivity case definitions such as ‘fever = malaria’ will lead clinicians to treat all fever cases as malaria, thus overlooking life-threatening episodes of ARI.

Table 8: Two possible diagnostic algorithms for malaria

Diagnostic algorithm	Sensitivity	Specificity
1) Malaria = fever or history of fever	high	low
2) Malaria = fever $>38.5^{\circ}\text{C}$ + positive blood test + no sign of other febrile infections such as ARI	moderate	high

Figure 14

Illustration of coverage and effectiveness steps in curative interventions



The danger is thus that a diagnostic algorithm that is highly sensitive but poorly specific for a given disease will result in low sensitivity for an equally important disease with similar symptoms. Over-diagnosis and consequent over-treatment of malaria is an enormous problem in sub-Saharan Africa.⁵³

A solution to the above conundrum is Integrated Management of Childhood Illness (IMCI), a WHO and UNICEF initiative offering diagnostic and treatment algorithms that can enable staff with limited skills to simultaneously tackle all main childhood infections. According to IMCI, in a high malaria-transmission country a febrile child receives antimalarials, but also antibiotics if

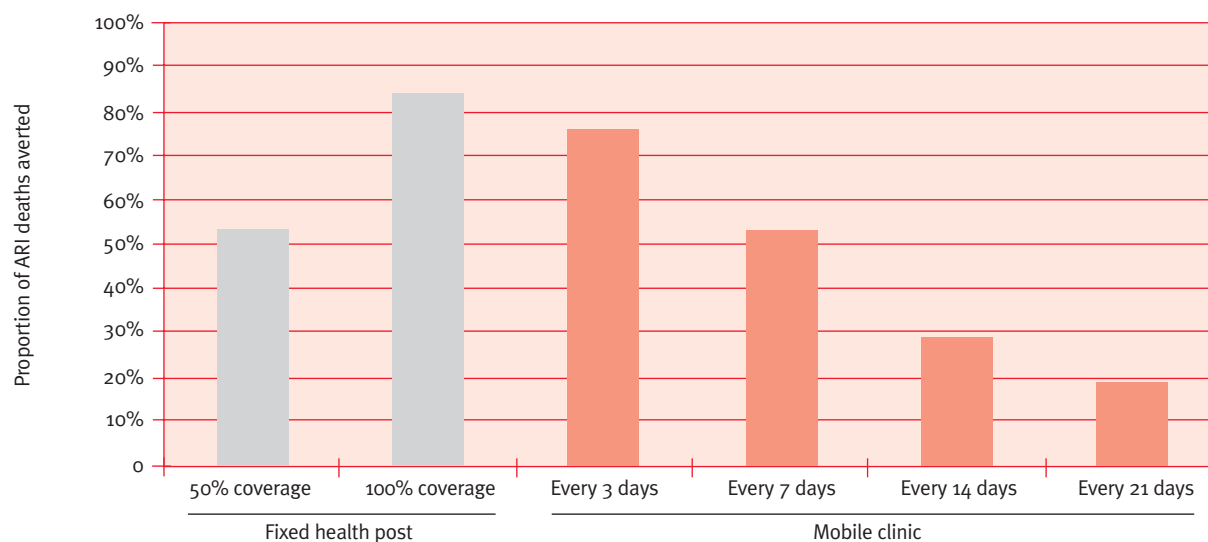
displaying signs of possible ARI. IMCI results in over-treatment overall, but covers the main causes of child illness. Evidence on its impact is at present mixed.⁵⁴

Coverage versus effectiveness

An intervention's impact will be greatest when both coverage and effectiveness are maximised, i.e. when nearly everyone benefits from the best possible intervention. In practice, however, there is often a tension between high coverage and high effectiveness, mainly due to the constraints imposed on relief: scarce funding (e.g. compared to what a crisis-stricken person in Europe would be offered), lack of skilled human resources, difficult working conditions

Figure 15

Predicted proportion of ARI deaths averted over 90 days in a population of 1,000 children, according to different treatment scenarios



and denial of access by combatants. This often creates a difficult choice between providing very sophisticated treatment to few, or very basic treatment to many.

We illustrate this quantitatively by comparing mobile clinics to fixed health posts in an isolated community where 1,000 children are experiencing a given incidence rate of ARI (contact Francesco Checchi for details on the mathematical model: francesco.checchi@lshtm.ac.uk). The goal is to reduce CFR of ARI. Mobile clinics are better equipped and thus offer higher effectiveness; they can also refer severe cases for hospital treatment, but they only visit the community every x days. By comparison, a fixed health post delivers daily care, but with lower effectiveness due to a cruder diagnostic algorithm, less efficacious antibiotics, and the inability to treat severe cases. Two scenarios of health post coverage are considered: 100% of cases attend the health post, or only 50%.

A health post which sees all ARI cases will prevent more deaths than one that sees only half, and indeed could be superior to a mobile clinic visiting as often as every three days, despite lower effectiveness. As the frequency of mobile clinic visits declines, so does their impact.⁵⁵

Sometimes triage can be used to identify cases most at risk of dying (i.e. maintain high coverage among those who need effective and complex treatment), and minimise workload. During a shigella epidemic in Sierra Leone, MSF teams hospitalised cases with signs of severity or cases aged below 5 years or 50 and above, as these were known to be at high risk; all others received oral rehydration solution.⁵⁶

Monitoring and evaluation

Why monitor and evaluate health interventions?

How will we know we helped? We most likely won't, unless a proportion of programme resources is set aside for monitoring and evaluation (M&E) activities. The prices of M&E efforts may seem exorbitant, partly due to increasing reliance on consultants. However, the costs of collecting data for M&E must be considered in light of the actual cost of the interventions themselves: the right question to ask is: does it make sense to spend 5% or 10% of the budget to decide whether the other 95% or 90% has had the intended effect? M&E can lead to improvements in programme quality, thus increasing impact, adding to existing knowledge on implementation, enhancing staff competence and morale, increasing the efficiency, cost-effectiveness and appropriateness of future programmes and raising the standards and expectations of relief worldwide.

There is perhaps some confusion among donors and agencies about the purposes of M&E. Its ultimate goal is to document and maximise impact. However, establishing whether a single intervention led to a given outcome on morbidity or impact on mortality is enormously difficult outside of a research context.

- A reduction in the health outcome (e.g. incidence of diarrhoea) or impact (e.g. infant mortality) that one might wish to monitor may be due to many different factors apart from the intervention itself. Typically, several interventions are deployed simultaneously: for example, an NGO may construct latrines and another wells. Attributing any reduction in diarrhoeal mortality

to either would require sophisticated research. A worsening of morbidity/mortality can also occur despite an intervention having some impact, if the intervention is deployed against a backdrop of increasing disease risk.

- Measurement of health outcomes and impacts of individual interventions requires measuring disease-specific incidence and mortality in the community (since health facilities only capture a fraction of cases). Current tools to classify causes of disease and death at the household level are not very accurate and require expertise.⁵⁷ Verbal autopsies, whereby next of kin report the signs and symptoms of their dead family member, are the best method, but only work well for some diseases.⁵⁸
- Measuring impact (mortality) specifically, while preferable because of its ultimate, downstream nature, requires studies with extremely large sample size.

What step of the intervention should one monitor?

Impact of individual interventions is a mirage in most circumstances – but is it really necessary to measure it? We believe that, for single interventions that have proven effective in studies, it is not. Which intervention step should M&E focus on? We believe that:

- For single preventive interventions (e.g. vaccination, vector control, hygiene promotion), provided that evidence on their effectiveness is available, it is sufficient to demonstrate output (coverage): outcome and impact (effectiveness) may be assumed. However:
 - Factors that could reduce effectiveness below that measured in published studies (e.g. logistics conditions; beliefs specific to the community, such as fear of vaccines) should be identified: their role should be anticipated in planning, and considered at least qualitatively during M&E.
 - Deviations from standards of implementation should be appraised. For example, a hand washing promotion campaign is shown to be effective when one hygiene promoter is present per 1,000 people, but the agency is only able to field one promoter per 5,000 people.
 - Efforts to maximise effectiveness should be included among project activities, and must also be evaluated. For example, bed net distributions should include community training on net maintenance.
- For single curative interventions, outcome and impact cannot be assumed, since factors that may modify their effectiveness are very context-specific. The effectiveness of care is very difficult to measure without following up patients in a research study. Nevertheless, outcome can reasonably be assumed if the following conditions are met:
 - The diagnostic algorithm is highly sensitive. Alternatively, the sensitivity is not very high but is known, thus allowing some quantification of the likely outcome, given a certain output.
 - The treatment is known or can safely be assumed to

be efficacious in the intervention setting (for example, drug resistance is very site-specific).

- Routine spot check evaluations at health facilities are carried out on a sufficient sample of patient visits, showing that key steps in the care package are functioning well.
- The outcomes and impact of sector-wide (e.g. water and sanitation) or crisis-wide relief operations are also difficult to measure, but cautious conclusions can be drawn based on evidence from past relief operations, and pre- and post-type comparisons within the crisis-affected community (not the ideal epidemiological set-up for establishing causality between relief and impact, but probably the only one available in most circumstances). The ultimate impact indicator is mortality (among all age groups and among children under five years). Outcome indicators that relate more or less to specific relief sectors include acute malnutrition prevalence, occurrence of disease outbreaks and disease attack rates. If a temporal association can be shown between the trend in coverage of essential relief services (output indicators), and the trend in such outcome or impact indicators, cautious conclusions about the relief operation's impact may be drawn, provided other factors not related to relief that could explain the observed outcome and impact are also appraised.

What about interventions without evidence?

Unless conditions make it the only option, no intervention should be introduced without first studying its effectiveness.⁵⁹ Nevertheless, untested interventions (for example, some mental and food security interventions) have been implemented in crises for perceived humanitarian reasons. From a humanitarian and medical standpoint it is difficult to abstain from attempting to relieve suffering.

Lack of evidence supporting proposed interventions for high-priority health problems should spur urgent research. In the interim, a solution might be to dedicate a greater part of the budget for untested interventions to M&E, so as to build some empirical evidence.

Data sources for monitoring and evaluation of interventions

Table 9 proposes data sources and types of studies needed to monitor each step of the intervention flow. Inputs and processes can usually be monitored using existing project data, and do not require working with the denominator (target population). Monitoring output (coverage) can be done:

- Roughly, using project data, assuming that population size is known (and it always should be), and a clear coverage target has been set (e.g. all children 6–59 months old to be vaccinated). For example, if the total population is 50,000, and 17% are in the age group 6–59 months, the target population for vaccination is 8,500; if 6,300 vaccine doses are administered, coverage should be $6,300/8,500 = 74\%$.
- More preferably, using household surveys,⁶⁰ whereby a

Table 9: Possible sources of data to monitor and evaluate each step of the intervention cycle

Steps in the intervention cycle	Data source/study needed
Inputs	Project reports (procurement records, accounts, etc.)
Processes	Project reports (procurement records, accounts, etc.) Project registers (health facility registers for curative interventions) Ad hoc data collection (ex. special reporting forms for each session/campaign/distribution) Spot checks to ensure processes are occurring as per project proposal
Outputs	Comparison of project data (ex. units of prevention services delivered, or persons attending health facilities) and known need (based on target population and target standard of service) Household surveys to estimate coverage, as in allocation of preventive intervention or access to curative care (better)
Outcomes/Impacts	Randomised trial (requires control group not receiving the intervention: unethical if intervention is proven to be beneficial, and very difficult to set up in emergencies) Case-control study (compares coverage of intervention among people who died or experienced any other outcome/impact, versus those who didn't, but were otherwise similar: ethical, but difficult to set up and cannot prove cause-effect relationship) Household surveys: one approach is to compare chosen outcome/impact indicators in households/individuals who received the intervention versus any who didn't (the problem is that those who didn't receive the intervention may be worse off in many other respects, which needs to be controlled for in the analysis); another approach is to do pre- and post-intervention surveys. Neither of these can decidedly establish cause-effect relationship Pre- and post- comparison of surveillance data: reported incidence of diseases against which the intervention was targeted is compared for the periods before and after the intervention's onset. This is a rough approach and cannot establish cause-effect relationship Usually, outcome and impact can be assumed from output

representative sample of beneficiaries is interviewed about access to the intervention, yielding estimates with associated confidence intervals (or margins of error).⁶¹ Various sampling strategies are available, some feasible in very chaotic settings. Surveys are the only option if an accurate population estimate is unavailable; the target population is hard to define (for example, the catchment area of a clinic is essentially determined by patients themselves, and may sometimes overlap with that of a nearby clinic); or people from outside the target population also access the intervention (e.g. food distributions in camps may attract neighbouring resident populations; here project data would overestimate coverage). Surveys also provide a more realistic picture, since 'services provided' may not always equal 'services received': engineers may estimate a borehole's theoretical output as 10 L per person per day, but if people must queue for hours to access it, the actual output at the household level will be less.

Outcome and impact measurement, if ever useful, requires ad hoc studies or expert analysis. The main challenge is establishing causality.⁶²

Defining good indicators

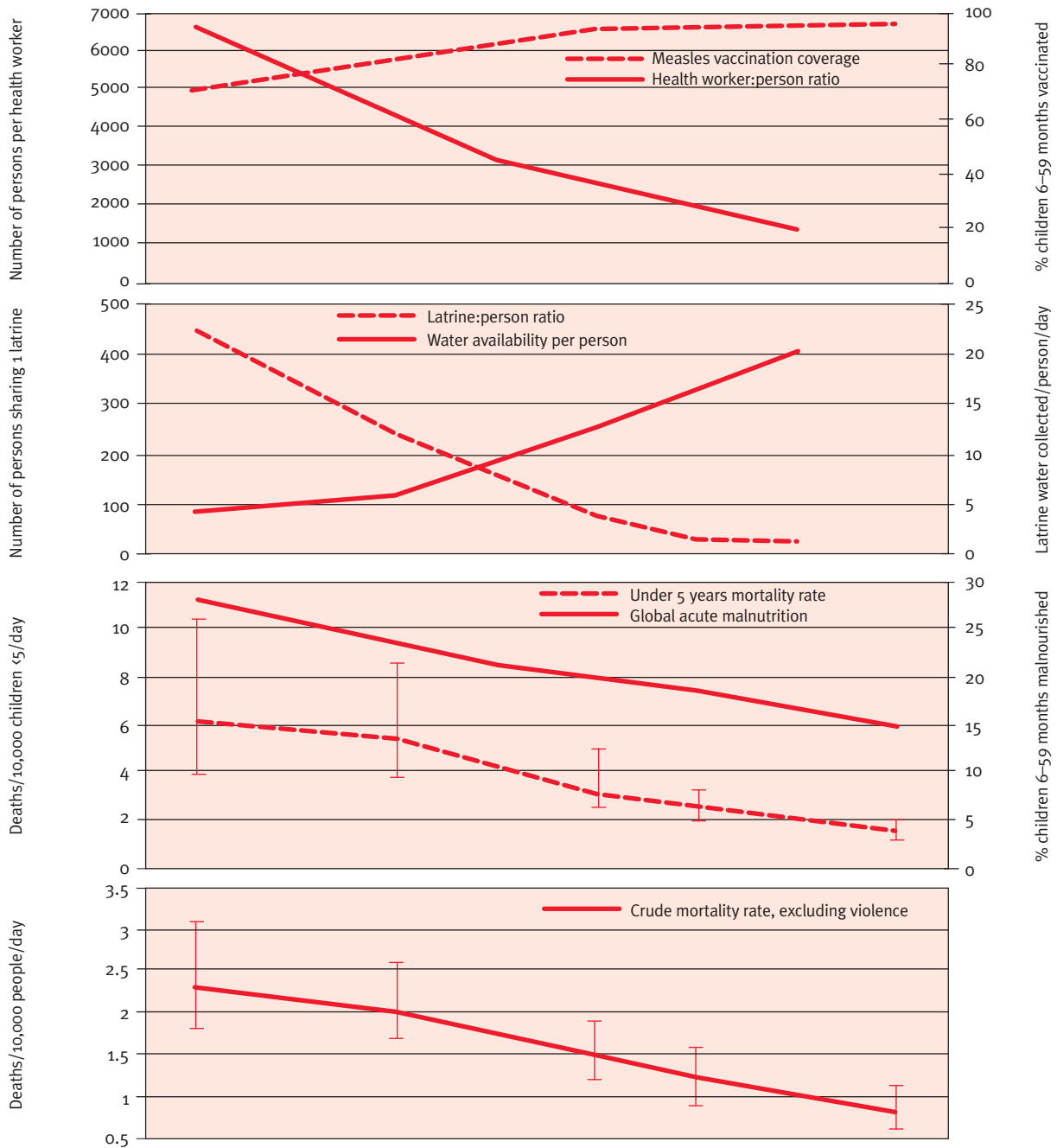
Developing good M&E indicators in crises is essential. The main criteria are S.M.A.R.T, or:

- **Specific:** any indicator must be expressed with time-person-place reference (e.g. 'proportion of patients admitted into the Clinic A mental health support programme who drop out before the end of therapy', not 'cases dropping out').
- **Measurable:** 'proportion of diarrhoeal cases due to viral versus non-viral pathogens in children under one year' would require unwieldy sample sizes and an enormous battery of lab tests.
- **Achievable:** 'time to eradication of acute respiratory infections' is very unrealistic; 'reduction in the CFR of ARI among hospitalised children under 5 from baseline' is reasonable.
- **Relevant:** the indicator should reflect what the intervention is expected to achieve (e.g. 'incidence of diarrhoea' would be pointless for monitoring access to oral rehydration since this intervention cannot reduce TR; CFR would be relevant).
- **Timebound:** the indicator should relate to an output/outcome/impact that can occur within the programme timeframe (for example, 'increase in the proportion of pregnant women who receive tetanus vaccination during antenatal visits to 80% by month 12 of the intervention').

M&E should not be burdensome: the list of indicators should be short.

Figure 16

Comparison of hypothetical trends in essential service, outcome (malnutrition) and impact (mortality) indicators



Note that data may be missing for some months, for some indicators. This exercise, however, can only be performed if all crucial crisis indicators are being measured on a nearly monthly basis: the trend is very important. Note also that, if surveys are being used, there will always be confidence intervals (error bounds) around some of the estimates, such as above for crude and under 5 mortality.

Chapter 6

Conclusion

Review of key principles

We have tried to present an overarching view of the epidemiology of crises, with a focus on physical health. Figure 17 (page 50) summarises the main concepts:

- Different crisis conditions (entrapment, mass displacement into camps, displacement into communities, natural disaster, food crisis) combine with different demographic and epidemiological settings (tropical and/or very poor, transitional, industrialised) to determine risk factors for infectious (either endemic or epidemic) and non-infectious diseases.
- Risk factors result in excess morbidity (outcomes) and mortality (impacts), both directly and indirectly attributable to the crisis: excess mortality is the ultimate indicator of crisis severity.

- Interventions achieve outputs with a given coverage, and the combination of coverage x effectiveness results in morbidity and mortality averted.
- Both risk factors and interventions modify the TR, Pr and CFR steps of disease progression.
- Risk assessment and disease surveillance, along with evidence, determine priority interventions.
- Interventions result in reduced excess morbidity and mortality: this reduction is the outcome/impact of relief.

Ten common fallacies

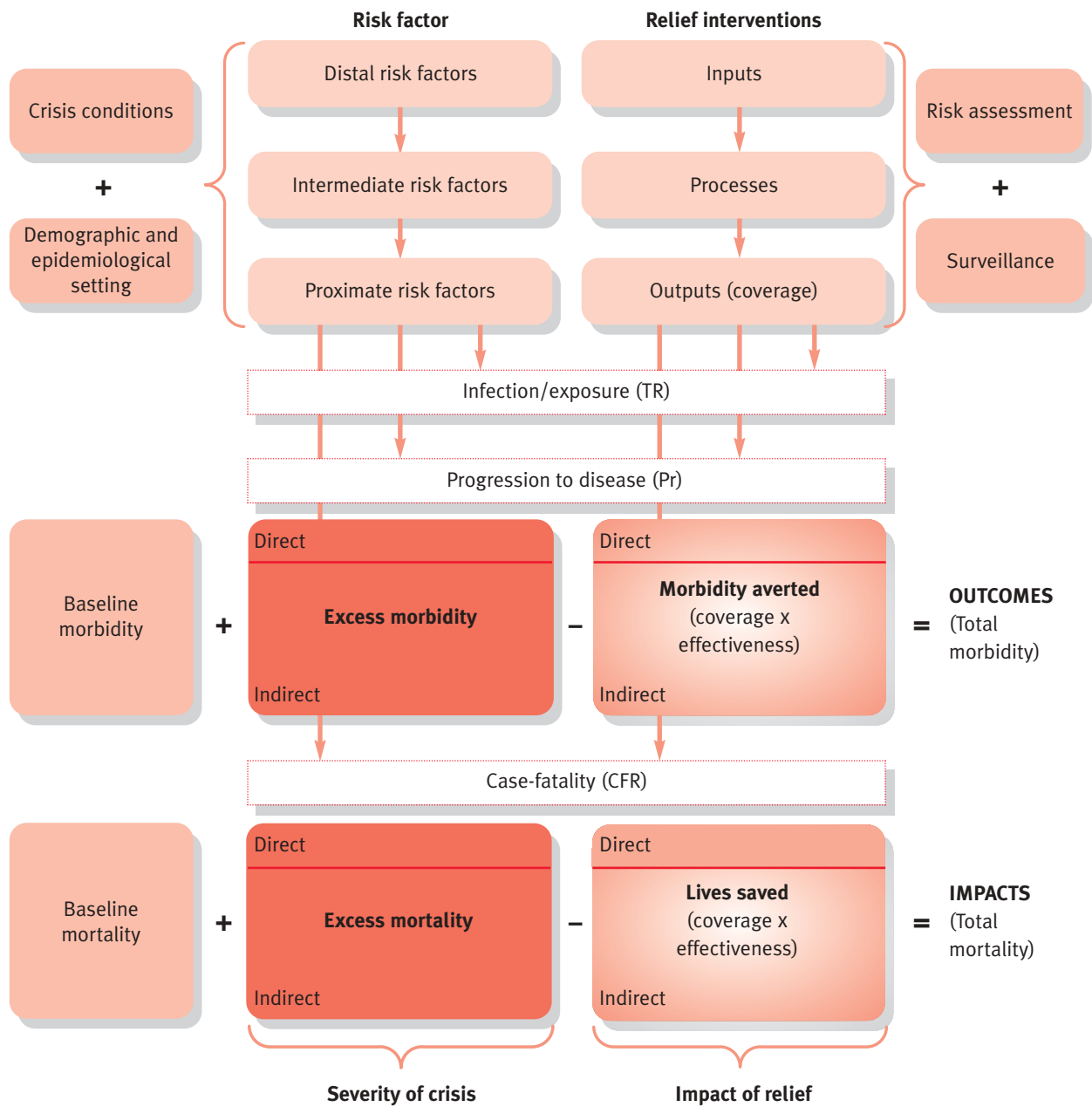
Box 8 suggests ten common fallacies in health relief operations, which underscore the importance of a widely shared quantitative, epidemiological grasp of how crises and interventions affect population health, and encapsulate key principles that we feel should guide public health responses to any crisis.

Box 8

Ten common epidemiological fallacies in public health responses to crises

- 1. Every crisis affects health in the same way.** There are very different types of crisis conditions, which result in different evolutions of morbidity and mortality. Slow-onset, long-duration situations of 'entrapment' due to insecurity may result in very large death tolls, even though mortality rates may not spike dramatically. Mass displacement into overcrowded camps is different from displacement into host communities or small settlements, and probably carries higher disease risks.
- 2. Epidemics kill more people than endemic diseases.** There is no evidence of this, and the inverse is more likely. Although epidemic prevention and control should remain a priority activity in relief operations, common endemic diseases such as diarrhoea, ARI, malaria and neonatal infections must not be neglected. A relatively small rise in the incidence of these diseases, coupled with a modest increase in their lethality, will lead to large increases in mortality, even if no epidemics occur.
- 3. Epidemics are very frequent in the aftermath of natural disasters.** Several studies and reviews have shown this not to be the case, at least compared to wars. However, some disasters (mainly flooding) have resulted in significant epidemics, mainly of diarrhoeal or vector-borne diseases, justifying surveillance and prevention activities.
- 4. Public health interventions cannot be implemented without first conducting a detailed situation and needs assessment.** One should not wait for detailed assessments to come in before implementing the basic package of Sphere guidelines, which is as much about controlling present problems as preventing new ones. These guidelines are meant to provide minimum acceptable living conditions. What makes crises different is the degree to which certain risk factors will emerge. However, all major risk factors for disease need to be pre-empted and addressed from the earliest stages of the relief operation.
- 5. Prevention is most important.** Preventive interventions have many advantages over curative ones, but one should avoid setting one above the other. Prevention will mainly decrease the degree of exposure/infection and the probability of developing disease; it will, however, do little to reduce case-fatality.
- 6. Treatment is most important.** As above. Treatment alone will result in health systems being overwhelmed by disease cases they have not been able to prevent. Both treatment and prevention are needed.
- 7. Numerators are enough for assessment and monitoring.** Numerators (number of cases, cases treated, number of services offered, etc.) are never sufficient to meaningfully assess needs, and monitor/evaluate interventions. They must always be compared to the denominator (i.e. the population at risk, or targeted for the intervention). Unless this is done, no conclusions about disease trends, coverage and effectiveness can reliably be drawn.
- 8. Coverage is enough.** Achieving and documenting high coverage is crucial. However, there are many reasons (especially in crises, and for curative care) for effectiveness to be much poorer than expected: poor effectiveness will negate the effects of high coverage.
- 9. Effectiveness is enough.** Even a very effective intervention will have little impact unless a considerable proportion of the target population is covered by it.
- 10. Each unit of output results in one unit of impact.** The relationship between coverage and outcome/impact (disease cured or averted/deaths averted) is rarely linear. More often there are 'threshold effects', whereby a certain level of coverage needs to be reached for considerable impact to be achieved. Vaccination is a prime example of this.

Figure 17
Schematic of physical effects of crises



- Existing standards (Sphere or other) reflect best available evidence. They must be used as guidance for intervention planning and monitoring, until better standards are introduced. These standards exist for a reason, and failure to achieve them, whilst sometimes inevitable due to insecurity, political or funding constraints, will generally result in poor health, and largely preventable death tolls.
- Failing to consider the specific risk factors present in a

- given crisis context, as well as the underlying epidemiological and demographic profile of the crisis-affected population, will tend to result in the implementation of inappropriate interventions.
- Maximising interventions' impact requires awareness of coverage as well as effectiveness issues. M&E should not be perceived as a stale exercise to satisfy donors or headquarters, but rather as a crucial opportunity to improve present and future practice.

Glossary

Note that other sources may offer different definitions. We have opted here for ones that seemed to us most understandable and pragmatic.

Active reporting: mode of reporting of surveillance data whereby staff contact health facilities to obtain data.

Age-specific mortality rate: **mortality rate** in a specific age group. See **under-5 mortality rate** for an example.

Alert threshold: similar to **epidemic threshold**, but of lesser severity (i.e. level of **incidence** which if exceeded may lead to on-site investigations or preparation for a possible **epidemic**).

Attack rate: **proportion** or **percentage** of the **population at risk** (or of the entire population) that experiences a new case of **infection** or **disease** during a given time period. Equivalent to the cumulative **incidence rate** over this same period. The main application is during **epidemics**. Note that an attack rate is not really a **rate**.

Basic reproductive ratio, often abbreviated as **R₀**: the **reproductive ratio** in conditions where all individuals are **susceptible** to the **infection**, and there is no treatment or control. These conditions often occur at the very beginning of an **epidemic**.

Burden (of disease): general term indicating the importance/severity of a specific disease or condition in the community. Burden can best be quantified as **incidence**, **attack rate**, **prevalence**, **mortality rate**, etc., depending on the disease in question.

Case-control study: a type of epidemiological study design in which **exposure** in **disease** or **infection** cases is compared to exposure in a comparable group of non-cases. A typical output of such a study is **odds ratios**.

Case-fatality ratio or **rate**, often abbreviated as **CFR**: **proportion** or **percentage** of **disease** cases that die as a result of the disease. Note that CFR is not a **ratio** or **rate**, but a proportion.

Case definition: in **epidemiology**, a standardised list of very precise criteria which, if fulfilled, result in a case being classified and reported as due to a given **disease**.

Causal association: link between a given factor and a given health event (**infection**, **disease**, death, etc.), whereby the factor is the only cause, or one of the possible causes, of increased risk of the event occurring. The Bradford-Hill criteria of causality are often invoked in **epidemiology** to decide whether an association is indeed causal.

Cause-specific mortality rate: **mortality rate** due to a specific **disease** (e.g. cholera) or exposure (e.g. intentional injury).

Child mortality ratio or **rate:** see the demographic definition of **under-5 mortality rate**. **Ratio** is a more accurate term than **rate** in this case.

Chronic disease: **disease** lasting a long time (e.g. years) relative to the individual's lifetime, or even one's whole life. Is sometimes used interchangeably with **non-infectious** or **non-communicable disease**, but in fact some chronic diseases can be **infectious**.

Clinical trial: a type of epidemiological study design in which two groups of people, one exposed to an intervention (i.e. **protective factor**) being tested, and another not exposed (or given the best currently available intervention), are observed over time, and the **relative risk** of the health event the intervention is designed to mitigate is measured in the exposed vs. non-exposed group: this **relative risk** is also a measure of **efficacy** or **effectiveness** of the intervention. More complex trial designs exist. Most clinical trials are **randomised**, meaning that allocation of the intervention is determined by chance, and that the two groups are similar to each other in all characteristics except for exposure to the intervention; they can be **blinded** or **double-blinded** depending on whether study staff and/or patients are aware of whether they received the intervention (or which).

Cohort study: a type of epidemiological study design in which a group of people exposed to a **risk** or **protective factor** and a group of people not exposed are observed over time, and the **relative risk** of a given health event in the exposed vs. non-exposed group is measured.

Communicable disease: used interchangeably with **infectious disease**.

Confounder: factor that appears associated with a given health event, simply because it is correlated with it in time and place, but which actually hides or distracts from a true causal association involving another factor which causes both the confounder and the health event.

Cost-effectiveness: **ratio** of an intervention's inputs (quantified financially) to its outcome/impact (quantified epidemiologically through any measure of **burden**); for example, US dollars per case averted.

Coverage: **proportion** or **percentage** of individuals in need of (or targeted as intended beneficiaries of) an intervention, who actually get it.

Cross-sectional study: a type of epidemiological study design in which the health event of interest is measured in a sample of the population or a given sub-group of interest, at a given point in time. Typical set-up for measuring **point prevalence**. See also **household survey**.

- Crude mortality rate**, often abbreviated as **CMR**: **mortality rate** among all age groups and due to all causes.
- Curative intervention**: an intervention whose main benefit consists of reducing the **risk** of death or other deleterious lasting consequences *after* the individual contracts the infection or disease.
- Cure rate**: **efficacy** of a treatment (or drug) regimen. It is not really a **rate**, but a **proportion**.
- Death rate**: see **mortality rate**.
- Diagnostic algorithm**: succession of clinical and/or laboratory examination and tests that results in a decision to diagnose the case as due to a given disease or not.
- Disease**: symptoms of illness and/or impairment of normal healthy bodily and/or mental functions due to an **infectious** or **non-infectious** cause.
- Duration of infection**: the time period elapsing between **infection** and the end of infection, which can be due to the body's immunological response, treatment administered, or death.
- Duration of infectiousness**: see **infectious period**.
- Effectiveness**: **proportion** or **percentage** of individuals who receive an intervention who experience the intended health outcome/impact the intervention is designed to achieve.
- Efficacy**: **effectiveness** in ideal study conditions (i.e. optimal administration of the intervention).
- Endemic disease**: **disease** that occurs year-round in the community, with **incidence** falling within an expected range. The distinction between endemic and **epidemic-prone diseases** is usually applied to **infectious/communicable diseases** only. Some diseases can behave as endemic or epidemic-prone depending on the context
- Epidemic** [*noun*]: occurrence of cases of a **disease** that is usually absent from the community; alternatively, a situation in which the disease is usually present, but suddenly reaches **incidence** levels in excess of the expected range.
- Epidemic** [*adjective*]: **disease** that causes an **epidemic**.
- Epidemic-prone disease**: **disease** that is normally absent from a community or present at low to moderate levels, but which can suddenly cause an **epidemic**.
- Epidemic threshold**: critical level of **incidence** specified *a priori*, which, if exceeded, triggers the declaration of an epidemic and/or pre-determined public health responses.
- Epidemiology**: the study of the distribution of **diseases** in the community, and of the factors affecting their frequency.
- Excess morbidity**: **morbidity** that would not have occurred if the crisis had not taken place. Can be quantified as excess **incidence** or using other indicators.
- Excess mortality**: **mortality** that would not have occurred if the crisis had not taken place. Can be quantified as excess **mortality rate** or an excess death toll.
- Exhaustive surveillance**: type of **surveillance** strategy whereby data is collected from all health facilities.
- Exposure**: the event and degree of an individual's coming into contact with an **infectious** pathogen or any substance/experience/other **risk factor** that increases the risk of a non-infectious disease (or a **protective factor** that decreases it).
- Group-specific mortality rate**: **mortality rate** in a given group (e.g. orphans).
- Herd immunity threshold**: critical level of **immunisation coverage** required to prevent **outbreaks** of a specific disease, in a given context.
- Horizontal intervention**: an intervention usually targeted at many diseases at once, and integrated within the existing health system.
- Household survey**: type of epidemiological study in which a representative sample of households is selected, and provides information on given health events of interest. Often analogous to a **cross-sectional study**.
- Incidence**: occurrence of *new* cases of **infection** or new cases of **disease**, depending on which event is being investigated. In this paper we have restricted the definition of incidence to signify the occurrence of new cases of disease.
- Incidence rate**: number of **incident** (i.e. new) cases of **infection** or **disease**, per unit **population at risk** and unit time (see also **rate**): for example, 'new cases of bloody diarrhoea per 1,000 children under 5 years per week'.
- Incubation period**: the time period elapsing between **infection** and the appearance of signs and symptoms of the **disease**.
- Infant mortality ratio** or **rate**, often abbreviated as **IMR**: number of infants below one year old dying out of 1,000 live births in a given year. Equivalent to the probability of dying in the first year of life. **Ratio** is a more accurate term than **rate** in this case.
- Infection**: colonisation of the body by foreign pathogens, including prions, viruses, bacteria, fungi and various micro- and macroscopic parasites. Infection does not necessarily lead to **disease**, and can even be beneficial (e.g. certain gut bacteria).
- Infectious disease**: **disease** that is caused by the **infection** of the body with foreign pathogens.
- Infectious period**: time period during which an infected individual is able to **transmit** the **infection** to others, via any **transmission route**. Also known as **duration of infectiousness**. The infectious period is often shorter than the **duration of infection**.
- Maternal mortality ratio**, often abbreviated as **MMR**: number of women dying due to pregnancy-related causes while pregnant or within 42 days of pregnancy termination, out of 100,000 live births in a given year.

Morbidity: the occurrence of **disease**.

Mortality: the occurrence of death.

Mortality rate: number of deaths occurring in a given **population at risk** per unit time, over a given time period (e.g. deaths per 10,000 people per day). Also known as **death rate**.

Neonatal mortality ratio or rate: number of infants below 28 days old dying out of 1,000 live births in a given year (sometimes 'below 30 days' is used instead). Equivalent to the probability of dying in the first month of life. **Ratio** would be a more accurate term than **rate** in this case.

Non-communicable disease: see **non-infectious disease**.

Non-infectious disease: **disease** not caused by **infections**. May include both physical and mental diseases. See also **chronic disease**.

Odds ratio: common expression of **relative risk**, indicating the **ratio** of people exposed to the given **risk** or **protective factor** to people not exposed, among cases, divided by the same ratio among non-cases (controls). Often used in **case-control** or **cross-sectional** studies and in **outbreak** investigations.

Opportunity benefit: saving inherent in deploying several interventions together.

Opportunity cost: cost of foregoing or delaying an intervention so as to implement another intervention.

Outbreak: equivalent to **epidemic**, but usually taken to refer to the very first cluster of epidemic cases, or to a small epidemic.

Passive reporting: mode of reporting of surveillance data whereby facilities report data without prompting.

Percentage: any **proportion** multiplied by 100 so as to provide the number out of 100 that fulfil a given condition (e.g. number of people out of 100, i.e. percent who are infected with malaria).

Period prevalence: **proportion** or **percentage** of the population (or sub-group) that had the infection or disease during a given period (usually a year). Uncommon.

Point prevalence: **proportion** or **percentage** of the population (or sub-group) that has the infection or disease at a specific point in time (e.g. today). This is the most common type of **prevalence** expressed, and the term prevalence accompanied by a proportion of percentage should be assumed to imply point prevalence.

Population at risk: fraction of the population that is **susceptible** to a given **infection/disease**, or to death. If not specified, the entire population is assumed to be at risk.

Population-attributable risk: **proportion** of the total **risk** of a given health event that the population experiences, which may be attributed to a given **risk factor**.

Prevalence: number of cases of infection or disease present in the population (or a specific sub-group).

This includes **incident** (new) as well as existing cases. See **point prevalence** and **period prevalence**.

Preventive intervention: an intervention whose main benefit consists of reducing the **risk** of **infection**, **exposure** or progression to **disease** or death *before* the individual contracts the infection or disease.

Proportion: quantity A over (i.e. divided by) quantity N, where A is a fraction of N (e.g. proportion of all people with malaria infection: A = malaria-infected people; N = all people).

Proportionate morbidity: **proportion** or **percentage** of all disease cases that is due to a given cause.

Proportionate mortality: **proportion** or **percentage** of all deaths that is due to a given cause.

Protective factor: factor that, when present, decreases the epidemiological **risk** of a given health event. We speak of **exposure** to a protective factor.

Rate: the number of events occurring per unit time (e.g. number of landslides per year). In **epidemiology**, rates are usually expressed as events per unit time *and* per unit people, i.e. as **incidence rates** (e.g. new cases of **disease** per 1,000 people per month). Other common uses are **mortality rates** or **birth rates** (e.g. births per 1,000 people per year).

Ratio: quantity A over (i.e. divided by) quantity B, where A is not part of B (e.g. male to female ratio; people to latrines ratio).

Relative risk, often abbreviated as **RR**: amount by which **risk** is increased (multiplied) or decreased (divided) in people **exposed** to a given **risk factor** or **protective factor**, compared to people not exposed (e.g. RR=3 means the risk is three-fold in those exposed to the risk factor; RR=0.1 means the risk is one tenth in those exposed to the protective factor). Also equivalent to the risk in the exposed divided by the risk in the unexposed. RR can be quantified as an incidence rate ratio, attack rate ratio, prevalence ratio, **odds ratio**, etc.

Reproductive number: see **reproductive ratio**.

Reproductive rate: see **reproductive ratio**.

Reproductive ratio, often abbreviated as **R**: the average number of **infections** that will result from any given case of infection (i.e. the average number of successful **transmissions** arising from each case of infection).

Risk: in **epidemiology**, a general term indicating the probability, for an individual or a community, that a given health event (**infection**, **disease**, death, etc.) will occur or is present.

Risk assessment: systematic assessment of disease **risk**.

Risk factor: factor that, when present, increases epidemiological **risk** of a given health event. We speak of **exposure** to a risk factor.

Risk management: planning and implementation of interventions designed to mitigate **risk**.

Risk ratio: see **relative risk**.

Sensitivity: ability of a **case definition** or **diagnostic algorithm** to correctly classify the true cases as cases, i.e. to capture as many of the real cases as possible. Expressed as a **percentage**.

Sentinel surveillance: type of **surveillance** strategy whereby data are collected only by some health facilities chosen to be representative of the population under surveillance.

Serial interval: average time elapsed between the onset of symptoms in the ‘primary’ case, and the onset of symptoms in the ‘secondary’ cases it **transmits** the **infection** to. It can also be computed as the time elapsed between the **infection** in the primary case and the infection in the secondary cases.

Specificity: ability of a **case definition** or **diagnostic algorithm** to correctly classify the true non-cases as non-cases, i.e. to minimise the number of false positive diagnoses. Expressed as a **percentage**.

Surveillance: systematic collection, analysis and interpretation of data on health events that is then used for defining and monitoring policy and interventions to mitigate them. In crises, surveillance efforts mostly concern **epidemic-prone** diseases. See also **sentinel surveillance** and **exhaustive surveillance, active reporting** and **passive reporting, case definition**.

Susceptible: able to contract the **infection**, i.e. at risk of **transmission**. Alternatively, one can speak of susceptibility to **disease**, i.e. the ability to fall ill once one is infected.

Systematic review: attempt to exhaustively search, review and summarise all of the published evidence on the **efficacy** or **effectiveness** of an intervention, or of the effect of a given **risk** or **protective factor**. A **meta-analysis** often supplements systematic reviews: this is an attempt to combine individual study findings into one summary figure.

Transmission: passage of the **infection** from one individual to the next. This can occur through various **transmission routes**.

Transmission route: mechanism through which an **infection** is **transmitted** (e.g. through airborne droplets, via a **vector** such as mosquitoes, etc.: see Table 1).

Under 5 years mortality ratio: see the demographic definition of **under-5 mortality rate**. **Ratio** is a more accurate term than **rate** in this case.

Under-5 mortality rate, often abbreviated as **U5MR**: in emergency **epidemiology**, this is understood as the **mortality rate** among children under 5 years (e.g. deaths among children under 5 years per 10,000 children under 5 years per day). In demography and more long-term development settings, this refers to the number of children under 5 years of age dying out of 1,000 live births in a given year, i.e. the probability of dying before age 5: this is also known as the **child mortality ratio** or **rate**, as commonly reported in UNICEF *State of the World's Children* publications.

Vector: organism (usually insects, more rarely snails or other small animals) that plays a role (usually crucial) in the **transmission** of an **infectious** pathogen from one individual to the next. Very often, pathogens can only survive and reproduce by infecting specific vectors (e.g. the female *Anopheles spp.* mosquito for malaria) and fulfilling part of their life cycle within them.

Vertical intervention: an intervention targeted specifically to one disease (or closely related group of diseases), and running in parallel to the routine health system.

Virulence: the ability of an **infectious** pathogen to result in successful **transmission** of the **infection, disease** and/or death, once a case comes into contact with a **susceptible** non-case. This property is inherent to the pathogen (e.g. to its ability to reproduce rapidly, secrete harmful compounds, etc.).

Notes

- 1 Médecins Sans Frontières, *Refugee Health: An Approach to Emergency Situations* (London: Macmillan Education Ltd, 1997); M. A. Connolly (ed.), *Communicable Disease Control in Emergencies: A Field Manual* (Geneva: WHO, 2005); Sphere Project, *Sphere Handbook*, revised ed., www.sphereproject.org, 2004.
- 2 United Nations, *Humanitarian Response Review* (Geneva: UN, 2005). See also the Active Learning Network for Accountability and Performance in Humanitarian Action: <http://www.odi.org.uk/ALNAP>.
- 3 Human Security Center, *The Human Security Report 2005: War and Peace in the 21st Century* (Boston, MA: Oxford University Press, 2005); H. A. Ghobarah, P. Huth and B. Russett, 'The Post-war Public Health Effects of Civil Conflict', *Soc Sci Med* 2004;**59**(4):869–84.
- 4 Only a handful of studies have quantified its extent in crises. We refer readers to a review of sexual violence in the 2005 *Human Security Report*, and a guide to sexual violence in humanitarian settings by the Inter-Agency Standing Committee: Inter-Agency Standing Committee, *Guidelines for Gender-based Violence Interventions in Humanitarian Settings: Focusing on Prevention of and Response to Sexual Violence in Emergencies* (Geneva: IASC, 2005).
- 5 F. Checchi and L. Roberts, *Interpreting and Using Mortality Data in Humanitarian Emergencies: A Primer for Non-epidemiologists*, Network Paper 52 (London: HPN, 2005).
- 6 P. J. Guerin, C. Brasher, E. Baron et al., 'Case Management of a Multidrug-resistant Shigella Dysenteriae Serotype 1 Outbreak in a Crisis Context in Sierra Leone, 1999–2000', *Trans R Soc Trop Med Hyg* 2004;**98**(11):635–43.
- 7 J. P. Guthmann, H. Klovstad, D. Boccia et al., 'A Large Outbreak of Hepatitis E Among a Displaced Population in Darfur, Sudan, 2004: The Role of Water Treatment Methods', *Clin Infect Dis* 2006;**42**(12):1685–91.
- 8 T. Grein, F. Checchi, J.M. Escribà et al., 'Mortality among displaced former UNITA members and their families in Angola: a retrospective cluster survey', *British Medical Journal* 2003;**327**(7416):650–655.
- 9 Checchi and Roberts, *Interpreting and Using Mortality Data in Humanitarian Emergencies*.
- 10 B. Coghlan, R. J. Brennan, P. Ngoy et al., 'Mortality in the Democratic Republic of Congo: A Nationwide Survey', *Lancet* 2006;**367**(9504):44–51.
- 11 Possibly the best documentation of community-dispersed IDPs comes from Guinea. W. Van Damme, Do Refugees Belong in Camps? Experiences from Goma and Guinea', *Lancet* 1995;**346**(8971):360–2.
- 12 J. T. Watson, M. Gayer and M. A. Connolly, 'Epidemics after natural disasters', *Emerg Infect Dis* 2007;**13**(1):1–5.
- 13 F. Stewart, 'Root Causes of Violent Conflict in Developing Countries', *Bmj* 2002;**324**(7333):342–5.
- 14 L. C. Mullany, A. K. Richards, C. Lee et al., 'Population-based Survey Methods To Quantify Associations Between Human Rights Violations and Health Outcomes Among Internally Displaced Persons in Eastern Burma', *J Epi Community Health* 2007;**61**(10):908–14.
- 15 World Health Organisation, *The Health Effects of Indoor Air Pollution in Developing Countries* (Geneva: WHO, 2002).
- 16 H. Young, A. Borrel, D. Holland and P. Salama, 'Public Nutrition in Complex Emergencies', *Lancet* 2004;**364**(9448):1899–909; H. Young and S. Jaspars, *The Meaning and Measurement of Acute Malnutrition in Emergencies: A Primer for Decision-Makers*, HPN Network Paper 56 (London: HPN, 2006).
- 17 L. Fewtrell, et al., 'Water, Sanitation and Hygiene Interventions To Reduce Diarrhoea in Less Developed Countries: A Systematic Review and Meta-analysis', *Lancet Infect Dis* 2005;**5**(1):42–52.
- 18 R. M. Coupland and H. O. Samnegaard, 'Effect of Type and Transfer of Conventional Weapons on Civilian Injuries: Retrospective Analysis of Prospective Data from Red Cross Hospitals', *Bmj* 319, 410–412.
- 19 K. de Jong et al., 'Psychological Trauma of the Civil War in Sri Lanka', *Lancet* 2002;**359**(9316):1517–8; K. de Jong et al., 'The Trauma of Ongoing Conflict and Displacement in Chechnya: Quantitative Assessment of Living Conditions, and Psychosocial and General Health Status among War Displaced in Chechnya and Ingushetia', *Confl Health* 2007;**1**:4; B. L. Cardozo, O. O. Bilukha, C. A. Crawford et al., 'Mental Health, Social Functioning, and Disability in Postwar Afghanistan', *Jama* 2004;**292**(5):575–84.
- 20 De Jong et al., 'Psychological Trauma of the Civil War in Sri Lanka'.
- 21 UNICEF/WHO, *Pneumonia: The Forgotten Killer of Children* (New York: UNICEF, 2006).
- 22 R. E. Black, S. S. Morris and J. Bryce, 'Where and Why Are 10 Million Children Dying Every Year?', *Lancet* 2003;**361**(9376):2226–34.
- 23 J. E. Lawn, S. Cousens and J. Zupan, '4 Million Neonatal Deaths: When? Where? Why?', *Lancet* 2005;**365**(9462):891–900; C. Ronsmans and W. J. Graham, 'Maternal Mortality: Who, When, Where, and Why', *Lancet* 2006;**368**(9542):1189–200.
- 24 R. Coninx, 'Tuberculosis in Complex Emergencies', *Bull World Health Organ* 2007;**85**(8):637–40.
- 25 F. Checchi, J. Cox, S. Balkan et al., 'Malaria Epidemics and Interventions, Kenya, Burundi, Southern Sudan, and Ethiopia, 1999–2004', *Emerg Infect Dis* 2006;**12**(10):1477–85.
- 26 E. Depoortere, F. Checchi and F. Broillet et al., 'Violence and Mortality in West Darfur, Sudan (2003–04): Epidemiological Evidence from Four Surveys', *Lancet* 2004;**364**(9442):1315–20.
- 27 R. M. Coupland and D. R. Meddings, 'Mortality Associated with Use of Weapons in Armed Conflicts, Wartime Atrocities, and Civilian Mass Shootings: Literature Review', *Bmj* 1999;**319**(7207):407–10.
- 28 E. J. Mills and J. B. Nachega, 'HIV Infection as a Weapon of War', *Lancet Infect Dis* 2006;**6**(12):752–3; E. J. Mills et al., 'The Impact of Conflict on HIV/AIDS in Sub-Saharan Africa', *Int J STD AIDS* 2006;**17**(11):713–7; P. B. Spiegel, A. R. Bennedsen, J. Claass et al., 'Prevalence of HIV Infection in

- Conflict-affected and Displaced People in Seven Sub-Saharan African Countries: A Systematic Review', *Lancet* 2007;**369**(9580):2187-95; N. B. Mock, S. Duale, L. F. Brown et al., 'Conflict and HIV: A Framework for Risk Assessment to Prevent HIV in Conflict-affected Settings in Africa', *Emerg Themes Epidemiol* 2004;**1**(1):6.
- 29 R. F. Grais, A. J. Conlan, M. J. Ferrari et al., 'Time Is of the Essence: Exploring a Measles Outbreak Response Vaccination in Niamey, Niger', *J R Soc Interface* 2007.
- 30 H. Young, A. Borrel, D. Holland and P. Salama, 'Public Nutrition in Complex Emergencies', *Lancet* 2004;**364**(9448):1899-909; Young and Jaspars, *The Meaning and Measurement of Acute Malnutrition in Emergencies*.
- 31 U. E. Schaible and S. H. Kaufmann, 'Malnutrition and Infection: Complex Mechanisms and Global Impacts', *PLoS Med* 2007;**4**(5):e115.
- 32 S. Collins et al., 'Management of Severe Acute Malnutrition in Children', *Lancet* 2006;**368**(9551):1992-2000; Emergency Nutrition Network, Special Supplement: Community-Based Therapeutic Care (CTC), in T. Khara and S. Collins (eds), ENN Special Supplement Series, 2004.
- 33 L. B. Spiegel, P. Le, M. T. Ververs and P. Salama, 'Occurrence and Overlap of Natural Disasters, Complex Emergencies and Epidemics During the Past Decade (1995-2004)', *Confl Health* 2007;**1**:2; J. P. Guthmann, M. Bonnet, L. Ahoua et al., 'Death Rates from Malaria Epidemics, Burundi and Ethiopia', *Emerg Infect Dis* 2007;**13**(1):140-3.
- 34 O. W. Morgan et al., 'Mass Fatality Management Following the South Asian Tsunami Disaster: Case Studies in Thailand, Indonesia, and Sri Lanka', *PLoS Med* 3, e195, 2006.
- 35 M. Prince, V. Patel, S. Saxena et al., 'No Health Without Mental Health', *Lancet* 2007;**370**(9590):859-77.
- 36 K. Demyttenaere, R. Bruffaerts, J. Posada-Villa et al., 'Prevalence, Severity, and Unmet Need for Treatment of Mental Disorders in the World Health Organization World Mental Health Surveys', *Jama* 2004;**291**(21):2581-90.
- 37 It should also be remembered that mental illness, PTSD and depression are medical conditions requiring medical diagnosis and treatment, and should be distinguished from psychosocial activities that many agencies provide in emergency settings, which are preventive and often aimed at reintroducing a sense of normalcy into the lives of affected persons, especially children.
- 38 K. de Jong et al., 'Psychological Trauma of the Civil War in Sri Lanka', *Lancet* 2002;**359**(9316):1517-8.; de Jong et al., 'The Trauma of Ongoing Conflict and Displacement in Chechnya; K. de Jong, N. Ford and R. Kleber, 'Mental Health Care for Refugees from Kosovo: The Experience of Medecins Sans Frontieres', *Lancet* 1999;**353**(9164):1616-7; de Jong et al., 'The Trauma of War in Sierra Leone'; K. de Jong, S. Prosser and N. Ford, 'Addressing Psychosocial Needs in the Aftermath of the Tsunami', *PLoS Med* 2005;**2**(6):e179; W. F. Scholte, M. Olf, P. Ventevogel et al., 'Mental Health Symptoms Following War and Repression in Eastern Afghanistan', *Jama* 2004;**292**(5):585-93.
- 39 J. T. de Jong, I. H. Komproe, M. Van Ommeren et al., 'Lifetime Events and Posttraumatic Stress Disorder in 4 Postconflict Settings', *Jama* 2001;**286**(5):555-62.
- 40 R. F. Mollica, K. McInnes, C. Poole and S. Tor, 'Dose-effect Relationships of Trauma to Symptoms of Depression and Post-traumatic Stress Disorder among Cambodian Survivors of Mass Violence', *Br J Psychiatry* 1998;**173**:482-8.
- 41 R. F. Mollica et al., 'Disability Associated with Psychiatric Comorbidity and Health Status in Bosnian Refugees Living in Croatia', *Jama* 1999;**282**(5):433-9; R. F. Mollica et al., 'Longitudinal Study of Psychiatric Symptoms, Disability, Mortality, and Emigration among Bosnian Refugees', *Jama* 2001;**286**(5):546-54.
- 42 D. Silove, V. Manicavasagar, R. Mollica et al., 'Screening for Depression and PTSD in a Cambodian Population Unaffected by War: Comparing the Hopkins Symptom Checklist and Harvard Trauma Questionnaire with the Structured Clinical Interview', *J Nerv Ment Dis* 2007;**195**(2):152-7.
- 43 De Jong et al., 'Lifetime Events'; E. J. Mills, S. Singh, T. H. Holtz et al., 'Prevalence of Mental Disorders and Torture Among Tibetan Refugees: A Systematic Review', *BMC Int Health Hum Rights* 2005;**5**:7.
- 44 S. B. Thapa et al., 'Psychiatric Disability Among Tortured Bhutanese Refugees in Nepal', *Am J Psychiatry* 2003;**160**(11):2032-7.
- 45 J. T. de Jong 'Commentary: Deconstructing Critiques on the Internationalization of PTSD', *Cult Med Psychiatry* 2005;**29**(3):361-70; discussion 371-6.
- 46 B. Raphael and J. P. Wilson (eds), *Psychological Debriefing: Theory, Practice, and Evidence* (New York: Cambridge University Press, 2000); S. Rose, J. Bisson, R. Churchill and S. Wessely, 'Psychological Debriefing for Preventing Post Traumatic Stress Disorder (PTSD)', *Cochrane Database Syst Rev* 2002(2):CD000560.
- 47 Mollica et al. have proposed a strategy against mental illness in crises. R. F. Mollica, B. L. Cardozo, H. J. Osofsky, B. Raphael, A. Ager and P. Salama, 'Mental Health in Complex Emergencies', *Lancet* 2004;**364**(9450):2058-67.
- 48 R. F. Grais, D. Coulombier, J. Ampuero et al., 'Are Rapid Population Estimates Accurate? A Field Trial of Two Different Assessment Methods', *Disasters* 2006;**30**(3):364-76.
- 49 The CD-WGE consists of technical experts drawn from the various disease and cross-cutting departments within WHO.
- 50 A. Haines, D. Sanders, U. Lehmann et al., 'Achieving Child Survival Goals: Potential Contribution of Community Health Workers', *Lancet* 2007;**369**(9579):2121-31.
- 51 A. Creese, K. Floyd, A. Alban and L. Guinness, 'Cost-effectiveness of HIV/AIDS Interventions in Africa: A Systematic Review of the Evidence', *Lancet* 2002;**359**(9318):1635-43.
- 52 R. F. Grais, A. J. Conlan, M. J. Ferrari, et al., 'Time Is of the Essence: Exploring a Measles Outbreak Response Vaccination in Niamey, Niger', *J. R. Soc Interface* 2007.
- 53 H. Reyburn, H. Mbakilwa, R. Mwangi et al., 'Rapid Diagnostic Tests Compared with Malaria Microscopy for Guiding Outpatient Treatment of Febrile Illness in Tanzania: Randomised Trial', *Bmj* 2007;**334**(7590):403.
- 54 WHO, *Handbook - Integrated Management of Childhood Illness* (Geneva: WHO, 2000); J. R. Armstrong et al., 'Effectiveness and Cost of Facility-based Integrated Management of Childhood Illness (IMCI) in Tanzania', *Lancet* 2004;**364**(9445):1583-94.
- 55 For a review of mobile health unit strategies, see S. du Mortier and R. Coninx, *Mobile Health Units in Emergency Operations: A Methodological Approach*, Network Paper 60 (London: HPN, 2007).
- 56 P. J. Guerin, C. Brasher, E. Baron et al., 'Case Management of a Multidrug-resistant Shigella Dysenteriae Serotype 1

- Outbreak in a Crisis Context in Sierra Leone, 1999–2000’, *Trans R Soc Trop Med Hyg* 2004;**98**(11):635–43.
- 57 V. Brown, F. Checchi, E. Depoortere et al., ‘Wanted: Studies on Mortality Estimation Methods for Humanitarian Emergencies, Suggestions for Future Research’, *Emerg Themes Epidemiol* 2007;**4**(1):9.
- 58 N. Soleman, D. Chandramohan and K. Shibuya, ‘Verbal Autopsy: Current Practices and Challenges’, *Bull World Health Organ* 2006;**84**(3):239–45.
- 59 N. Banatvala and A. B. Zwi, ‘Conflict and Health. Public Health and Humanitarian Interventions: Developing the Evidence Base’, *Bmj* 2000;**321**(7253):101–5.
- 60 K. Bostoen, O. O. Bilukha, B. Fenn et al., ‘Methods for Health Surveys in Difficult Settings: Charting Progress, Moving Forward’, *Emerg Themes Epidemiol* 2007;**4**:13; P. B. Spiegel, ‘Who Should Be Undertaking Population-based Surveys in Humanitarian Emergencies?’, *Emerg Themes Epidemiol* 2007;**4**:12.
- 61 Checchi and Roberts, *Interpreting and Using Mortality Data in Humanitarian Emergencies*.
- 62 L. Roberts and C.-A. Hofmann, ‘Assessing the Impact of Humanitarian Assistance in the Health Sector’, *Emerg Themes Epidemiol* 2004;**1**(1):3.

Network Papers 1997–2007

Network Papers are contributions on specific experiences or issues prepared either by HPN members or contributing specialists.

- 21 *Humanitarian Principles: The Southern Sudan Experience* by I. Levine (1997)
- 22 *The War Economy in Liberia: A Political Analysis* by P. Atkinson (1997)
- 23 *The Coordination of Humanitarian Action: the case of Sri Lanka* by K. Van Brabant (1997)
- 24 *Reproductive Health for Displaced Populations* by C. Palmer (1998)
- 25 *Humanitarian Action in Protracted Crises: the new relief 'agenda' and its limits* by D. Hendrickson (1998)
- 26 *The Food Economy Approach: a framework for understanding rural livelihoods* by T. Boudreau (1998)
- 27 *Between Relief and Development: targeting food aid for disaster prevention in Ethiopia* by K. Sharp (1998)
- 28 *North Korea: The Politics of Food Aid* by J. Bennett (1999)
- 29 *Participatory Review in Chronic Instability: The Experience of the IKAFF Refugee Settlement Programme, Uganda* by K. Neeffjes (1999)
- 30 *Protection in Practice: Field Level Strategies for Protecting Civilians from Deliberate Harm* by D. Paul (1999)
- 31 *The Impact of Economic Sanctions on Health and Well-being* by R. Garfield (1999)
- 32 *Humanitarian Mine Action: The First Decade of a New Sector in Humanitarian Aid* by C. Horwood (2000)
- 33 *The Political Economy of War: What Relief Agencies Need to Know* by P. Le Billon (2000)
- 34 *NGO Responses to Hurricane Mitch: Evaluations for Accountability and Learning* by F. Grunewald, V. de Geoffroy & S. Lister (2000)
- 35 *Cash Transfers in Emergencies: Evaluating Benefits and Assessing Risks* by D. Peppiatt, J. Mitchell and P. Holzmann (2001)
- 36 *Food-security Assessments in Emergencies: A Livelihoods Approach* by H. Young, S. Jaspars, R. Brown, J. Frize and H. Khogali (2001)
- 37 *A Bridge Too Far: Aid Agencies and the Military in Humanitarian Response* by J. Barry with A. Jefferys (2002)
- 38 *HIV/AIDS and Emergencies: Analysis and Recommendations for Practice* by A. Smith (2002)
- 39 *Reconsidering the tools of war: small arms and humanitarian action* by R. Muggah with M. Griffiths (2002)
- 40 *Drought, Livestock and Livelihoods: Lessons from the 1999-2001 Emergency Response in the Pastoral Sector in Kenya* by Yacob Aklilu and Mike Wekesa (2002)
- 41 *Politically Informed Humanitarian Programming: Using a Political Economy Approach* by Sarah Collinson (2002)
- 42 *The Role of Education in Protecting Children in Conflict* by Susan Nicolai and Carl Triplehorn (2003)
- 43 *Housing Reconstruction after Conflict and Disaster* by Sultan Barakat (2003)
- 44 *Livelihoods and Protection: Displacement and Vulnerable Communities in Kismaayo, Southern Somalia* by Simon Narbeth and Calum McLean (2003)
- 45 *Reproductive Health for Conflict-affected People: Policies, Research and Programmes* by Therese McGinn et al. (2004)
- 46 *Humanitarian futures: practical policy perspectives* by Randolph Kent (2004)
- 47 *Missing the point: an analysis of food security interventions in the Great Lakes* by S Levine and C Chastre with S Ntububa, J MacAskill, S LeJeune, Y Guluma, J Acidri and A Kirkwood
- 48 *Community-based therapeutic care: a new paradigm for selective feeding in nutritional crises* by Steve Collins
- 49 *Disaster preparedness programmes in India: a cost benefit analysis* by Courtenay Cabot Venton and Paul Venton (2004)
- 50 *Cash relief in a contested area: lessons from Somalia* by Degan Ali, Fanta Toure, Tilleke Kiewied (2005)
- 51 *Humanitarian engagement with non-state armed actors: the parameters of negotiated armed access* by Max Glaser (2005)
- 52 *Interpreting and using mortality data in humanitarian emergencies: a primer* by Francesco Checchi and Les Roberts (2005)
- 53 *Protecting and assisting older people in emergencies* by Jo Wells (2005)
- 54 *Housing reconstruction in post-earthquake Gujarat: a comparative analysis* by Jennifer Duyne Barenstein (2006)
- 55 *Understanding and addressing staff turnover in humanitarian agencies* by David Loquercio, Mark Hammersley and Ben Emmens (2006)
- 56 *The meaning and measurement of acute malnutrition in emergencies: a primer for decision-makers* by Helen Young and Susanne Jaspars (2006)
- 57 *Standards put to the test: Implementing the INEE Minimum Standards for Education in Emergencies, Chronic Crisis and Early Reconstruction* by Allison Anderson, Gerald Martone, Jenny Perlman Robinson, Eli Rognerud and Joan Sullivan-Owomoyela (2006)
- 58 *Concerning the accountability of humanitarian action* by Austen Davis (2007)
- 59 *Contingency planning and humanitarian action: a review of practice* by Richard Choularton (2007)
- 60 *Mobile Health Units in emergency operations: a methodological approach* by Stéphane Du Mortier and Rudi Coninx (2007)

Good Practice Reviews

Good Practice Reviews are major, peer-reviewed contributions to humanitarian practice. They are produced periodically.

- 1 *Water and Sanitation in Emergencies* by A. Chalinder (1994)
- 2 *Emergency Supplementary Feeding Programmes* by J. Shoham (1994)
- 3 *General Food Distribution in Emergencies: from Nutritional Needs to Political Priorities* by S. Jaspars and H. Young (1996)
- 4 *Seed Provision During and After Emergencies* by the ODI Seeds and Biodiversity Programme (1996)
- 5 *Counting and Identification of Beneficiary Populations in Emergency Operations: Registration and its Alternatives* by J. Telford (1997)
- 6 *Temporary Human Settlement Planning for Displaced Populations in Emergencies* by A. Chalinder (1998)
- 7 *The Evaluation of Humanitarian Assistance Programmes in Complex Emergencies* by A. Hallam (1998)
- 8 *Operational Security Management in Violent Environments* by K. Van Brabant (2000)
- 9 *Disaster Risk Reduction: Mitigation and Preparedness in Development and Emergency Programming* by John Twigg (2004)

A full list of HPN publications is available at the HPN website: www.odihpn.org. To order HPN publications, contact hpn@odi.org.uk.

Humanitarian Practice Network

The **Humanitarian Practice Network (HPN)** is an independent forum where field workers, managers and policymakers in the humanitarian sector share information, analysis and experience.

HPN's aim is to improve the performance of humanitarian action by contributing to individual and institutional learning.

HPN's activities include:

- A series of specialist publications: Good Practice Reviews, Network Papers and *Humanitarian Exchange* magazine.
- A resource website at www.odihpn.org.
- Occasional seminars and workshops to bring together practitioners, policymakers and analysts.

HPN's members and audience comprise individuals and organisations engaged in humanitarian action. They are in 80 countries worldwide, working in northern and southern NGOs, the UN and other multilateral agencies, governments and donors, academic institutions and consultancies. HPN's publications are written by a similarly wide range of contributors.

HPN's institutional location is the Humanitarian Policy Group (HPG) at the Overseas Development Institute (ODI), an independent think tank on humanitarian and development policy. HPN's publications are researched and written by a wide range of individuals and organisations, and are published by HPN in order to encourage and facilitate knowledge-sharing within the sector. *The views and opinions expressed in HPN's publications do not necessarily state or reflect those of the Humanitarian Policy Group or the Overseas Development Institute.*

Funding support is provided by institutional donors (AusAID, CIDA, DANIDA, DFID, Development Cooperation Ireland, MFA Netherlands, SIDA, USAID), non-governmental organisations (British Red Cross, CAFOD, Christian Aid, Concern, International Rescue Committee, MSF, Oxfam, Save the Children (UK), World Vision) and UN agencies (WFP).

To join HPN, complete and submit the form at www.odihpn.org or contact the Membership Administrator at:

Humanitarian Practice Network (HPN)
Overseas Development Institute
111 Westminster Bridge Road
London, SE1 7JD
United Kingdom

Tel: +44 (0)20 7922 0331/74

Fax: +44 (0)20 7922 0399

Email: hpn@odi.org.uk

Website: www.odihpn.org