'IT IS ESTIMATED THAT BETWEEN 2002 AND 2020, APPROXIMATELY 1000 MILLION PEOPLE WILL BE NEWLY INFECTED, OVER 150 MILLION PEOPLE WILL GET SICK, AND 36 MILLION WILL DIE OF TB ...' WORLD HEALTH ORGANIZATION

# THE RETURN OF THE WHITE PLAGUE

## GLOBAL POVERTY AND THE 'NEW' TUBERCULOSIS

EDITED BY MATTHEW GANDY AND ALIMUDDIN ZUMLA

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# 1

# Life without Germs: Contested Episodes in the History of Tuberculosis

Matthew Gandy

The understanding and control of tuberculosis is one of the most significant chapters in the history of humankind. In a detailed chronology of the disease from classical times until the twentieth century the Italian historian Arturo Castiglioni remarked on 'the marvelous progress from demonism to bacteriology'.[1] For Castiglioni, writing in 1933, the science of bacteriology marked a decisive advance in the progressive development of Western civilization. The decline of TB was a marker on a path towards a better future in which rational knowledge would prevail over the ignorance, superstition and neglect of the past. Yet Castiglioni's faith in the centrality of science to the eradication of disease masks the full complexity of the epidemiology of TB. The rapid decline of the disease from the middle decades of the nineteenth century, before the disease was fully understood, and its more recent resurgence point to an array of social, economic and political developments beyond the confines of the laboratory.

Few other diseases have caused human suffering on the scale of TB. The classical medical scholar Hippocrates (460–370 BCE) considered 'phthisis', or pulmonary TB as it is now known, to be the 'greatest and most terrible disease'.[2] A combination of historical and archaeological evidence shows that TB was widespread in early Hindu, Greek and Roman societies, and gradually spread for over 2,000 years.[3] By the early nineteenth century the 'White Plague' had become the principal cause of death in much of Europe and North America and it is estimated that the prevalence of TB infection neared 100 per cent in rapidly growing cities such as London and Paris.[4]

The history of TB is complicated, however, by the diversity of manifestations of the disease and the absence of definitive data before the introduction of mass screening in the late nineteenth century using the bacterial culture tuberculin and X-ray photography.[5] In 1726, for instance, the English physician Richard Blackmore was unable to clearly distinguish 'scrophulous tumours' from other 'Morbifick viscous coagulations'. 'So immense is the variety of Knots and Tumours to which all Parts of the Body, External and Internal, are obnoxious,' Blackmore wrote, 'that to reduce them to their proper Classes and assign the Limitations and essential Boundaries that discriminate and divide them, seems impracticable.'[6] Other manifestions of the disease in addition to 'scrofula' (glandular TB) include infections of the spine (widely referred to as 'gibbus' or 'Pott's disease'), infections of the skin (known as 'lupus') and infections of the brain. The disease can affect almost any part of the body and produce a variety of symptoms, which have often caused it to be confused with bronchitis, typhoid or a variety of other illnesses.[7] Indeed, the powerful stigma surrounding the diagnosis of TB, and its long-standing hereditarian associations, has fostered a degree of confusion and uncertainty concerning many medical records in the pre-bacteriological era before the advent of compulsory notification and the widespread adoption of other modern public health measures.

For much of medical history, TB proved not only difficult to classify but also mysterious in its apparently indiscriminate prevalence. During the nineteenth century we find a variety of different explanations for TB. In addition to hereditary and 'constitutional' causes, for example, the English physician Sir James Clark, writing in 1837, lists 'improper diet, impure air, deficient exercise, excessive labour, imperfect clothing, want of cleanliness, abuse of spiritous liquors, mental causes and contagion'. Clark despaired at the 'total inefficacy of all means hitherto adopted for diminishing the frequency or reducing the mortality of this class of diseases'.[8] The American physician William Beach, writing in 1840, dismissed the notion that TB might be contagious and chose to emphasize 'hereditary disposition' marked by features such as 'prominent shoulders' or a 'narrow chest'. Like many of his contemporaries Beach suggested travel as the only alternative for patients who had not responded to 'proper medicine'. 'It is well known,' Beach wrote, 'that warm climates have considerable influence in removing tubercles from the Lung, by the genial and uniform temperature imparted, hence the

Brazils, and West India climate, have sometimes benefited, when other means have failed'.[9]

The diversity of nineteenth-century perspectives on the disease belied the fact that medical science could offer no satisfactory explanation or cure. In 1868, for example, the founder of modern cellular pathology, Rudolf Virchov, considered TB to be a kind of tumour and fiercely rejected the newly emerging contagion theories developed by the French surgeon Jean-Antoine Villemin.[10] Villemin had succeeded in infecting animals with TB under laboratory conditions and presented a direct challenge to the prevailing 'hereditarian' consensus in northern Europe. In fact, modern theories of contagion can be traced to the Veronese physician Gerolamo Fracastoro (1483–1553) who warned against the 'seeds of contagion' lingering in the rooms and belongings of phthsis sufferers.[11] Fracastoro's views proved influential in Italy but failed to make much impact in northern Europe where hereditary and 'constitutional' explanations prevailed. Theories of contagion held implications for the responsibilities of municipal government which conflicted with the narrowly perceived self-interest of political and economic elites. In eighteenth-century Italy, for example, legislative measures to ensure the disinfection of infected homes in Lucca, Florence, Naples and Venice were short-lived in the face of opposition on the grounds of cost.[12]

In the last two decades of the nineteenth century the contagion theories developed by Fracastoro and Villemin were radically advanced by the emerging science of bacteriology which could now identify the role of microbial pathogens in the spread of disease. The discovery of the TB bacterium by the Prussian bacteriologist Robert Koch in 1882 marked a decisive shift in the balance of the epidemiological debate: Koch declared that in the 'battle with this horrible plague . . . we are no longer dealing with an indefinable Something, but with a definite parasite whose vital processes are, for the most part, known, and which can be studied further'.[13] Yet even after Koch's discovery of the TB bacterium, alternative theories of hereditary or 'constitutional' transmission persisted for many years. And Koch was himself the subject of fierce criticism for disputing the significance of bovine TB in transmitting the disease to humans through infected milk.[14] In fact the history of TB was marked from the outset by rival bodies of medical thought rooted in alternative explanations for the transmission of the disease. The gradual acceptance of various 'germ theories' undermined

the moralistic discourses of nineteenth-century medicine and strengthened the political salience of the public health movement. Yet by the middle decades of the twentieth century the recognition that TB was a social disease rooted in poverty and poor housing became gradually obscured by an emphasis on the success of new forms of biomedical intervention. The discovery of relatively cheap and individualized courses of antibiotic treatment such as streptomycin and isoniazid succeeded in virtually eliminating the disease from more affluent societies but also served to shift attention away from the structural factors that had contributed towards the spread of the disease in the past.

This chapter continues with an exploration of the intersections between Romanticism, anti-urban sentiment and the development of the sanatoria movement in the pre-bacteriological era. My starting point is the nineteenth century, which marks a transition between the aestheticization of tubercular mortality and an emerging recognition of the contagious nature of the disease. I turn next to the debate surrounding the McKeown thesis and rival explanations for the widespread decline of TB in the second half of the nineteenth century. My focus then moves to the twentieth century in order to evaluate explanations for persistent regional and social disparities in the incidence of TB. Evolving interpretations of the relations between race, class and disease reveal how hereditarian views became reconfigured within a racialized topography of illness. I conclude with an examination of the global resurgence of TB since the early 1980s, and suggest that the success of TB eradication programmes during the 1950s and 1960s in the developed world served to deflect attention from the global scale and political complexity of the problem. The return of TB has undermined this earlier wave of scientific optimism and fostered a renewed recognition of the connection between disease and poverty.

## Romanticism, anti-urbanism and the sanatoria movement

During the modern era, TB became widely perceived as 'a disease of humid and dank cities'.[15] Ever-greater numbers of people were crowded into dilapidated and makeshift housing in the rapidly growing cities of Europe and North America, and those who could find work were widely subjected to dusty, confined or physically exhausting working conditions in the textile, metalworking and other industries

which contributed towards a greater susceptibility of infection.[16] Yet the absence of any clear understanding of the epidemiology of the disease led to a generalized indictment of urban life rather than any systematic analysis of the changing living and working conditions that placed many people at much greater risk than hitherto of contracting respiratory diseases. As the literary critic Susan Sontag observes:

> When travel to a better climate was invented as a treatment for TB in the early nineteenth century, the most contradictory destinations were proposed. The south, mountains, deserts, islands – their very diversity suggests what they have in common: the rejection of the city.[17]

The association between TB and urban living conditions fed into long-standing medical discourses surrounding climate and pulmonary TB. From Hippocrates onwards many physicians had recommended changes in climate to alleviate the symptoms of consumption as a form of 'physiological therapy'.[18] Consequently, wealthier sufferers sought to leave cities in search of warm and dry climates where they might alleviate their symptoms.[19] Hereditarian and 'constitutional' conceptions of TB had allowed dual cultures of disease to emerge: for the poor TB was a disaster, yet for the rich the illness was transformed into an intense personal experience.[20] The nineteenth-century internalization of TB as, for wealthy sufferers, a disease of the self rests on an interplay between Romantic anti-urbanism and pre-bacteriological epidemiology. Romantic depictions of pulmonary TB contributed towards the aestheticization of death and at the same time emphasized the unique individual sensitivities of the tubercular self in a grotesque parody of the enhanced social and cultural subjectivity of the Enlightenment. The literary Romantic movement, which was especially influential between 1760 and 1830, sought to transform the moral stigma of the TB death into a profound experience of individual sensitivity 'which dissolved the gross body, etherealized the personality, expanded consciousness'.[21] The disappearance or 'consuming' of the body became a metaphor for spiritual transcendence, in contrast to the widespread revulsion associated with pulmonary TB by earlier scholars and physicians.[22] The 'consumptive' lover dying as a consequence of rejection is a recurring cultural trope in Romantic literature which served to trivialize the suffering associated with the disease.[23] The actual death from TB of leading figures within the Romantic movement

such as the poets Novalis (1801), Ernst Schulze (1817) and John Keats (1821) intensified the association between artistic genius and consumption, thereby separating the experience of illness (and creativity) from any wider social context. TB became associated with a heightened state of creativity in the shortened lives of poets and artists (a creativity believed by contemporaries to be caused by the intoxicating effects of the illness) yet widespread opiate addiction among wealthy sufferers is a more plausible explanation for this 'mental effervescence'.[24]

The search for a climatic cure fostered the development of specialized institutions devoted to the treatment of TB. The first of these sanatoria were private health institutions, catering for the wealthier classes, which originated in Germany in the late 1850s and early 1860s. The commercial success of Hermann Brehmer and other pioneers of this new treatment soon attracted international attention. The construction of sanatoria across Europe and North America formed part of a wider reaction against the insalubrity of modern cities and was closely allied with the back-to-nature movements that emerged in nineteenth-century Europe and North America with their emphasis on forests, mountains and open-air pursuits. A second phase of development occured in the early twentieth century with the widespread construction of public sanatoria in Europe, North America and elsewhere, as the state played a much more active role in the eradication of TB.[25] The sanatoria evolved into elaborate institutions where patients, of whom an increasing number were working class, undertook a combination of rest and exercise aimed at eliminating infection and building up bodily defences. Though sanatoria had originally emerged as a kind of 'safe haven' from the urban and industrial chaos of the nineteenth century they gradually became an extension of urban modernity itself through their adoption of the modernist principles of 'hygienist' urban design.[26] Esoteric treatments such as heliotherapy (sunbathing) formed part of a wider aim to reconnect mind, body and nature in a new mode of modern living.[27] With the rapid growth of the sanatoria movement the promotion of rigorous treatment regimes replaced the original emphasis on specific locations such as islands and mountains. The spread of institutionalized treatment 'swept the pine trees from the path' and challenged the therapeutic role of climate.[28]

Reports of patient improvement lent a perceived scientific rationale to the burgeoning sanatoria movement but such claims remained controversial. 'These albums of cured cases,' the physician Pierre

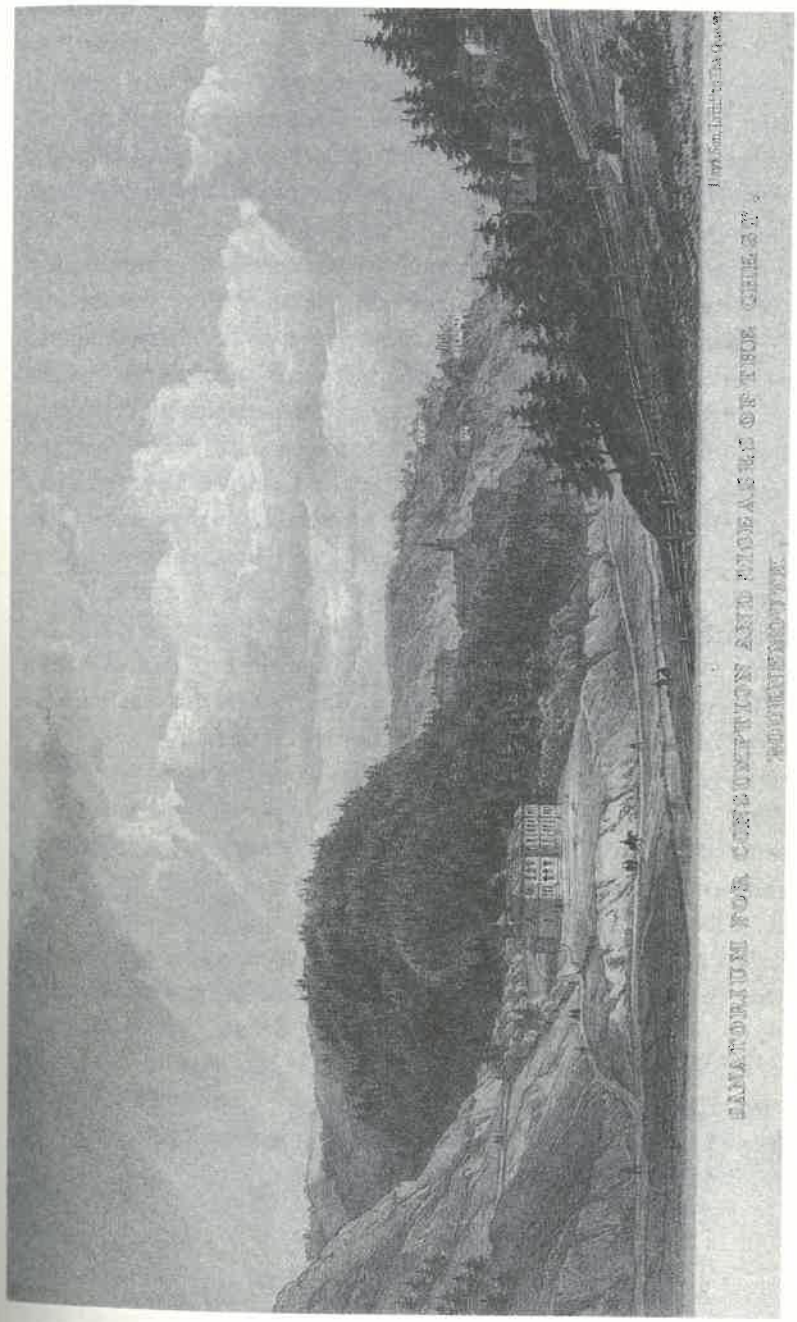

**Figure 1.1** The TB and chest disease sanatorium, Bournemouth, founded in 1855. Lithograph by Day and Son, n.d. Source: Wellcome Library for the History and Understanding of Medicine, London.

Hulliger mocked, 'have no scientific value: they are advertising results.'[29] By the 1930s a wide-ranging critique of sanatoria began to emerge based on their limited impact on the wider health problem, the unreliability of their medical records and the absence of any effective aftercare. Most critically, the provision of expensive and ineffective sanatoria treatment diverted attention and, more important, resources away from the more radical prescriptions that might have had any real impact on the disease.[30]

The second phase of the sanatoria movement grew out of the radical disjuncture in the experience of TB in nineteenth-century Europe and North America. Nineteenth-century conceptions of TB reworked existing disease metaphors within a specific historical and cultural context derived from the Romantic reaction to rapid urbanization. Yet by the late nineteenth century the 'perverted sentimentalism' of the past had been replaced by a renewed sense of fear and disgust.[31] No longer a 'romantic affliction', the disease was now unambiguously perceived by middle-class commentators as a menacing and disgusting stigma of poverty. In 1912, for example, the British physician de Carle Woodcock described TB as 'in truth a coarse, common disease, bred in foul breath, in dirt, in squalor. . . . The beautiful and the rich receive it from the unbeautiful poor. . . . The scrofula which deforms the already coarsened features of the stunted slum dweller is tubercle.'[32]

The development of the modern sanatorium reflected a shift in prevailing conceptions of TB from a constitutional affliction to be countered with a 'change of air' towards a contagionist emphasis on the eradication of disease through a programme of institutional segregation and intervention. Yet even when bacteriological insights into the disease began to be accepted in mainstream medical opinion, the idea that the disease was associated with specific categories of people continued to suffuse scientific and political debate.[33] The fatalistic spectre of 'tubercular urbanism' would blight the lives of the urban poor for many decades after the first tentative steps towards public health reform. And in much of the developing world the impact of TB was yet to be fully experienced.

## Social reform, the McKeown thesis and the epidemiological transition

Evidence from Europe and North America suggests that TB was in widespread decline from the middle decades of the nineteenth century onwards, before the introduction of any systematic attempts to prevent its spread (see Figure 1.2). This significant time lapse between the recorded decline of TB and coordinated control efforts is one of the most contentious themes in the history of disease: it forms the basis of the historian Thomas McKeown's influential view that TB and other infectious diseases declined as a by-product of wider social and economic advances rather than as a result of any specific medical interventions.[34] The core of the so-called 'McKeown thesis' is a rejection of the role of physicians, scientists and hospital administrators in the decline in morbidity and mortality – the so-called epidemiological

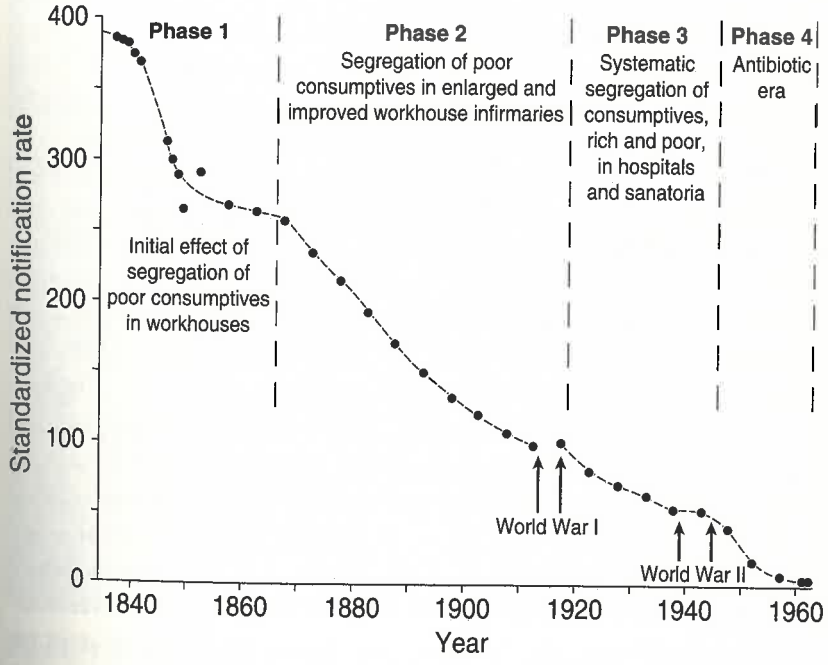


Figure 1.2 The historical decline in TB, 1840 to 1960. Source: Data derived from various sources including Thomas McKeown, *The Modern Rise of Population*, London: Edward Arnold 1976.

transition – that occurred during the second half of the nineteenth century:

> The history of TB illustrates, perhaps better than that of any other infection, a general point about the contribution of therapy. Effective clinical intervention came late in the history of the disease, and over the whole period of its decline the effect was small in relation to that of other influences.[35]

The principal flaw with McKeown's argument, however, is that the decline in TB mortality is difficult to attribute simply to improved nutrition or higher standards of living. Whilst McKeown is right to be sceptical about some of the claims of physicians, scientists and the 'heroic' genre of medical history, he overlooks the significance of wider public health reforms. Alternative perspectives on the history of disease have emphasized the critical importance of institutional and legislative change fostered by the political salience of the public health movement.[36] An emphasis on public health advocacy reveals the significance of specific measures aimed against TB such as effective patient segregation, housing improvements and the control of bovine TB.[37] The historian Leonard Wilson, for example, contests the McKeown thesis by emphasizing the role of segregation for infective TB patients. He points to the growing role of workhouse infirmaries, hospitals and other institutions during the nineteenth century using evidence from Britain, Prussia and the USA. From the late 1840s onwards, for instance, before the infectivity of the disease was understood, British workhouses admitted ever greater numbers of consumptive patients: the issue then, in the pre-bacteriological era, is not the effectiveness of treatment but the impact of containment on rates of infection.[38] Yet Wilson's emphasis on segregation does not fully explain why a higher proportion of those infected with TB, the majority of the population at the time, did not go on to develop the full symptoms of the disease. It is this complex relationship between infection, resistance and illness that fuels rival interpretations of the epidemiological transition in nineteenth-century Europe and North America.[39] The 'nutritionists', who adhere to the McKeown thesis, emphasize general improvements in resistance, whereas the 'contagionists' point to reduced sources of infection.

The uneven social and geographical incidence of the disease also works against any generalized mode of explanation. In France, for


Figure 1.3 A health visitor promoting a campaign against TB and infant mortality. Colour process print by Auguste Leroux, 1918. Source: Wellcome Library for the History and Understanding of Medicine, London.

instance, high mortality rates for TB, almost double those of Britain and Germany, persisted well into the twentieth century. 'France has a death rate from TB that is especially unfavourable,' observed Robert Koch in 1910; '. . . there can be no question of a general, regular, uniform decrease in mortality from consumption'.[40] Koch's analysis of the prevalence of TB in early-twentieth-century European cities showed that it was far higher in Paris than in Hamburg, Copenhagen or London. The historian Allan Mitchell has amplified these observations by showing that these regional and national differences are not explained by stark variations in general welfare and nutrition; instead, he attributes them to the failure of the French authorities to implement the kind of local sanitary measures and national health reforms that were being pursued in Britain and Germany. As Mitchell notes of early-twentieth-century France, 'The construction of inexpensive public housing (*habitations à bon marché*), still left in private hands, had scarcely begun to alleviate wretched conditions in the "dark spots" (*îlots noirs*) of French cities'.[41] In an interesting defence of the McKeown thesis, however, the historian David Barnes refutes the accuracy of available mortality data for late-nineteenth-century France. Barnes uses local rather than national data to question the argument made by Mitchell and others that levels of TB in France continued to increase at a time of falling rates in Britain and Germany. Barnes also suggests that French workers had a lower standard of living compared with Britain and Germany which may have limited their resistance to infection and hence led to the observed disparities in TB mortality. He emphasizes the role of workplace reform rather than municipal public health measures in order to provide a wider historical interpretation of the political dynamics of mortality decline in nineteenth-century Europe.[42] Thus the French case is especially interesting for the historiography of TB because it offers contradictory interpretations of the relationship between disease epidemiology and different aspects of social reform.

It appears that the McKeown thesis can be interpreted in two radically different ways. On the one hand the emphasis on general social and economic improvements behind the epidemiological transition can be perceived as a pretext to dismiss the role of medical science in large-scale improvements in human health. Robert Koch, for example, was quick to point out that the decline in TB since the middle of the nineteenth century could not be attributed to improved methods

of treatment.[43] On the other hand the scepticism towards the role of the medical sciences can be interpreted as an argument for structural reform in preference to a reliance on clinical intervention.[44] Here again, the observations of Koch are germane to contemporary debate. Koch contended that two critical factors explained the sharp variations in the incidence of the disease: the construction of hospital facilities and improvements in housing. Hospital facilities played a crucial role in segregating infective patients from the general population, and better housing reduced levels of overcrowding and infection within the home. Using the most detailed data available Koch was able to show that the relative increase in rates of infection in early-twentieth-century Norway, for instance, could be attributed to inadequate hospital provision. He also suggested that regional variations in the disease across northern Europe could be influenced by differences in housing design such as the use of cramped sleeping rooms or *Butzen*. Similar observations have been made in Britain where general improvements in TB mortality during the first half of the twentieth century mask stark regional disparities driven at least in part by differences in housing design. The overcrowded tenements of Glasgow, for example, saw rates of TB peak in 1949 despite forty years of attempts to control the disease.[45]

The generalized decline in TB since the middle decades of the nineteenth century – which began first in Britain, Germany and Belgium and occurred at a later stage in France, Ireland, Norway and elsewhere – cannot, then, be attributed to only one factor. If we add the possibility that widening resistance to infection may also have played a role then the picture becomes even more complicated.[46] The tension in the historical literature between 'nutritionist' and 'contagionist' perspectives has obscured the relationship between public health interventions and broader social and political developments. Legislative advances in fields such as workplace safety and housing provision must be viewed within the context of more general patterns of social and economic improvement.

Furthermore, the bacteriological insights from the 1880s onwards did begin to contribute towards a more focused and coordinated set of public health policies. The comprehensive efforts to fight TB in New York City in the 1890s, for example, were praised by Robert Koch for their efficiency, scientific rationale and willingness to challenge private interests in the pursuit of wider goals.[47] The city's new diagnostic research laboratory was 'a superb technical innovation, a medico-

scientific and political counterpart to the Brooklyn Bridge', and unlike any other facility yet constructed.[48] Similarly, the city's introduction of compulsory notification for the disease in 1897 marked a radical extension in the power of municipal government to override the objections of individual physicians and patients in the interest of public health.[49] Yet the nineteenth-century public health movement displayed a profound ambivalence towards the structural determinants of ill health since the purification of urban space it proposed was a process which sought simultaneously to transform both the social and the environmental characteristics of cities. Concern with housing design, sanitation and workplace safety was coupled with an emphasis on the morality and living habits of the working classes in the new industrial cities.

## Class, race and death in the twentieth century

The production of more accurate data on morbidity and mortality in the last decades of the nineteenth century began to reveal the disproportionate concentration of TB among specific social groups. The relationship between TB, race and social class became more evident not only because of the changing politics of public health but also because the partial retreat of the disease had highlighted its higher prevalence among the poor, non-whites and other marginalized groups.[50] A parallel discourse of differential susceptibility to TB began to develop alongside the sanitarian emphasis on improved hygiene. Instead of new bacteriological insights dispelling the earlier hereditarian emphasis of the nineteenth century, the 'constitutional' dimensions to disease epidemiology became reformulated in terms of different degrees of racial resistance to TB.

The recognition of differential mortality by race provoked a complex array of arguments which sought to use prevailing cultural and biological conceptions of human difference as a means to explain widening inequalities in human health. In the British colonies, for example, a theory of 'virgin soil' populations emerged which attributed high rates of TB mortality among non-whites to the lack of 'tuberculariza tion' or disease resistance amongst people who had not yet undergone the full effects of industrialization and urbanization. The influential pathologist Lyle Cummins posited that Europeans owed their higher survival rates to long-standing low-level exposure to the disease coupled with the

spread of new hygienic living practices which prevented the illness from taking root in all but the most weakened or intemperate individuals. In contrast, non-European peoples were characterized by Cummins as biologically inferior and culturally backward which rendered them susceptible to TB as a 'disease of civilization'. The high rates of TB experienced in Africa, India and elsewhere in the colonial world were widely seen as an inevitable and necessary marker on the path to a Westernized society which had achieved a more stable relationship between the human immune system and the virulence of the TB bacillus.[51] Yet at the heart of this 'tuberculariza tion' debate lay a profound confusion over whether the observed racial differences in mortality could be attributed to a Lamarckian process of acquired immunity or to a longer-term history of Darwinian evolution. Arguments about the relative significance of inherited or acquired immunological characteristics were supplemented by the proposal of a cultural hierarchy of disease resistance ranging from the 'childlike' susceptibility of colonized peoples to the supposedly hardier responses of urbanized European societies.

In the United States the control of TB amplified middle-class antipathy towards the 'lower classes' and heightened anxieties over immigration and racial mixing. Surveys carried out in the early twentieth century revealed that the rate of TB mortality was three times higher for black Americans than for white Americans.[52] These general figures mask even greater differences in many of the larger towns and cities. In Charleston, South Carolina, for example, the rate of TB mortality for blacks in 1900 was seven times higher than for whites. How were these disparities to be explained? A variant of the 'tuberculariza tion' thesis suggested that former slaves had been protected from the disease through their 'healthy' outdoor life on Southern plantations. The implication was that high rates of TB among African-Americans were an unfortunate yet inevitable consequence of their emanicipation.[53] Yet the social conditions under which most African-Americans lived could not be discounted altogether as a cause of their higher mortality. In 1920, for example, George E. Bushnell, a colonel in the US Medical Corps and director of the National Tuberculosis Association, combined a version of the tuberculariza tion thesis with a recognition of the impact of poverty on ill health. 'Because of his color the negro is barred from much productive industry,' Bushnell wrote. 'As he therefore cannot compete with the whites in earning capacity, he is relegated

to the worst habitations in the most insalubrious locations and to arduous or poorly paid toil everywhere.' But instead of calling for government action to improve living conditions, Bushnell chose instead to insist on improvements in the behaviour of poor blacks, the majority of whom he characterized as 'extraordinarily untrained, improvident and reckless; so that there must be taken into account not only poverty but a poverty which is tenfold worse because of the failure to make proper use of the scanty means at hand'.[54] A new concern with what the historian Marion Torchia describes as the 'Negro health problem' emerged which combined a mix of nineteenth-century moralism and white self-interest in order to avoid the possibility of infection by black maids, servants and other workers in daily contact with affluent middle-class homes.[55] As the historian Jessica Robbins notes of the shifting politics of TB in the United States:

> The acceptance of the germ theory had given TB a new and threatening social dimension. The tuberculous patient was not only an individual sufferer but also a potential source of infection and danger to others. The overwhelming majority of these patients were among the working-class poor. The prescription for health, physicians believed, was fresh air, good food, and plenty of rest – all of which were beyond the means of the urban poor. Professionals and reformers engaged in a protracted debate over what, if anything, should be done about this social problem.[56]

To acknowledge fully the structural causes of TB threatened to open up a much wider progressive agenda for social reform. For conservative health commentators it was essential, therefore, to persist with individualized modes of explanation which emphasized putative connections between 'immoral living habits' and susceptibility to infection. In Canada, for example, the chief medical officer, Peter Bryce, described TB as one of a group of social diseases along with alcoholism, syphilis and feeble-mindedness: 'We find them so often intermingled,' Bryce wrote, 'that it seems quite impossible to determine which disease is the determinant or dominant one.'[57] The idea of differential degrees of immunity provided a scientific veneer for official indifference towards much higher levels of TB among poor and marginalized sections of society. Evolutionary conceptions of TB implied that control of the disease would occur through a long-term immunological transition rather than through any kind of medical intervention.[58] At root,

however, anxieties over class, race and disease flowed from fears that these stigmatized groups would act as reservoirs for the contagion of wider society. Hereditarian views persisted in the post-bacteriological era as part of an emerging discourse of disease and national identity which would find its most virulent expressions under European fascism. Jews, for example, were denounced in Nazi Germany as 'a racial TB among nations'.[59] Thus even in the context of the widespread retreat of the disease across Europe and North America, TB retained a powerful metaphorical resonance for racial and ethnic hatred.

Improvements in diagnosis after the discovery of the TB bacillus in 1882 were not matched by major advances in treatment for many decades. Koch's claim, for example, to have discovered a cure for TB in 1890 through the production of the bacterial culture tuberculin was discredited. Although the incidence of TB declined during the twentieth century those who contracted the disease still suffered a high mortality rate. And even when improved treatments became available, the most vulnerable sections of society – principally the urban poor – were often unable to gain access to adequate medical care.[60] In the post-bacteriological era there were repeated innoculation experiments aimed at furthering scientific understanding of bacterial immunity. In 1914, for example, the American physician Guy Hinsdale predicted that 'future generations will be provided with a practical and efficient method of destroying this insatiate monster'. It would be some years, however, before the efforts of Trudeau, Gilliand, de Schweinitz and others would lead towards any definitive advance in the development of an effective vaccine. The most critical advance in TB inoculation was achieved by the French scientists Albert Calmette and Camille Guérin who began testing a bovine-derived vaccine in 1922. Initially the Bacille Calmette-Guérin (BCG) vaccine was only widely adopted in France and other countries – such as Spain and Canada – with strong cultural ties to French science.[61] In the USA, by contrast, the rejection of the BCG vaccine reflected a long-standing antipathy towards universal health interventions which might strengthen the role of the state in the advancement of public health (despite the pioneering early efforts of some municipal authorities). American scientists feared that the introduction of BCG would subvert the self-improvement ethos of the New Deal and at the same time draw attention to the persistence of the disease in spite of the twentieth-century rhetoric of scientific success.[62] In essence, the varied national responses to the

BCG vaccine reflected different conceptions of the relationship between health care and social policy, a tension which was temporarily obscured by advances in the antibiotic treatment of TB sufferers from the 1940s onwards.[63]

In 1944 the treatment for TB was transformed with the first use of the antibiotic streptomycin by Selman Waksman to cure infected patients. This was followed in 1951 by the use of another powerful antibiotic, isoniazid. Taken together, these new drugs revolutionized the treatment of TB and contributed towards a sharp decline in the environmental emphasis of the past. Unlike the BCG vaccine which aimed to prevent the progression from infection to illness, the widespread use of antibiotic drugs from the early 1950s onwards sought to cure patients who had already become ill. Within thirty years the rate of TB mortality in the developed world had fallen by more than 90 per cent. The discovery of streptomycin instituted a new phase in the control of TB which would increasingly emphasize issues of patient compliance over any wider discussion of the social and economic dimensions to disease epidemiology. The very success of the antibiotic revolution in health care served to disengage clinical medicine from the wider public health agendas of the past. The historical construction of the term 'non-compliance' reveals how bio-medical perceptions of patient deviance emphasized that particular groups are 'difficult' or 'recalcitrant' in terms of cooperating with medical authorities. Thus the issue of uneven access to adequate treatment in the antibiotic era became widely framed in terms of individual deficiencies or antirational belief systems rather than any acknowledgment of the social context in which the disease might be spread.[64] In apartheid South Africa, for example, persistent racial disparities in rates of infection were routinely dismissed on the grounds of cultural difference and a preference for traditional medicine. Yet a wealth of evidence from South Africa showed how poor housing and working conditions, combined with inequalities in access to medical care, had contributed towards widening health disparities in the twentieth century.[65] At a global scale, the antibiotic revolution of the 1940s and 1950s was highly uneven in its social and geographical impact, leaving much of the world's population languishing under high rates of infection with only haphazard access to treatment. Most critically, however, the early success of antibiotic drugs served to mask the continuing prevalence of TB infection. The 'magic bullet' of drug therapy diverted attention

from the social and economic conditions in which the TB bacillus could continue to thrive.

## Global poverty and the 'new' TB

The antibiotic revolution of the 1940s and 1950s led a range of leading public health campaigners, scientists and physicians confidently to predict the eradication of TB by the year 2000.[66] By the early 1980s TB appeared to be largely a disease of historical interest in the West, a consensus that indicated a dangerous complacency in the face of the continuing high prevalence of the disease in many developing countries. As recently as 1987, for example, the *Oxford Textbook of Medicine* predicted the virtual eradication of TB in 'most technically advanced countries' before the year 2050.[67] Yet those who considered TB in a global context were far less optimistic. In 1964, for instance, the executive director of the International Union against Tuberculosis, John Holm, issued this warning:

> For about half of the world's population no organized efforts are made to control tuberculosis, and this is the half where the problem is most serious. For the other half, efforts to control tuberculosis are conducted in a haphazard manner. Only a small fraction of the world's population is covered by well-organized programs in which the most modern means to control tuberculosis are systematically employed.[68]

The turning point in global efforts to control TB can be traced to the United States in the mid-1980s where a sudden increase in cases was observed in urban areas: between 1985 and 1992 there was a rise in TB cases of over 20 per cent.[69] Cities such as New York faced a rapid and unexpected spread of TB which quickly escalated into a public health emergency. This surge in reported cases can be attributed to increases in poverty and homelessness during the 1980s combined with the effects of HIV infection and the spread of drug-resistant TB strains. The emerging public health crisis facing deprived inner-city neighbourhoods represented a microcosm of the changing global incidence of the disease. It soon became apparent that the problems facing inner-city America were surfacing on a global scale in response to the combined effects of drug resistance, HIV and poverty.

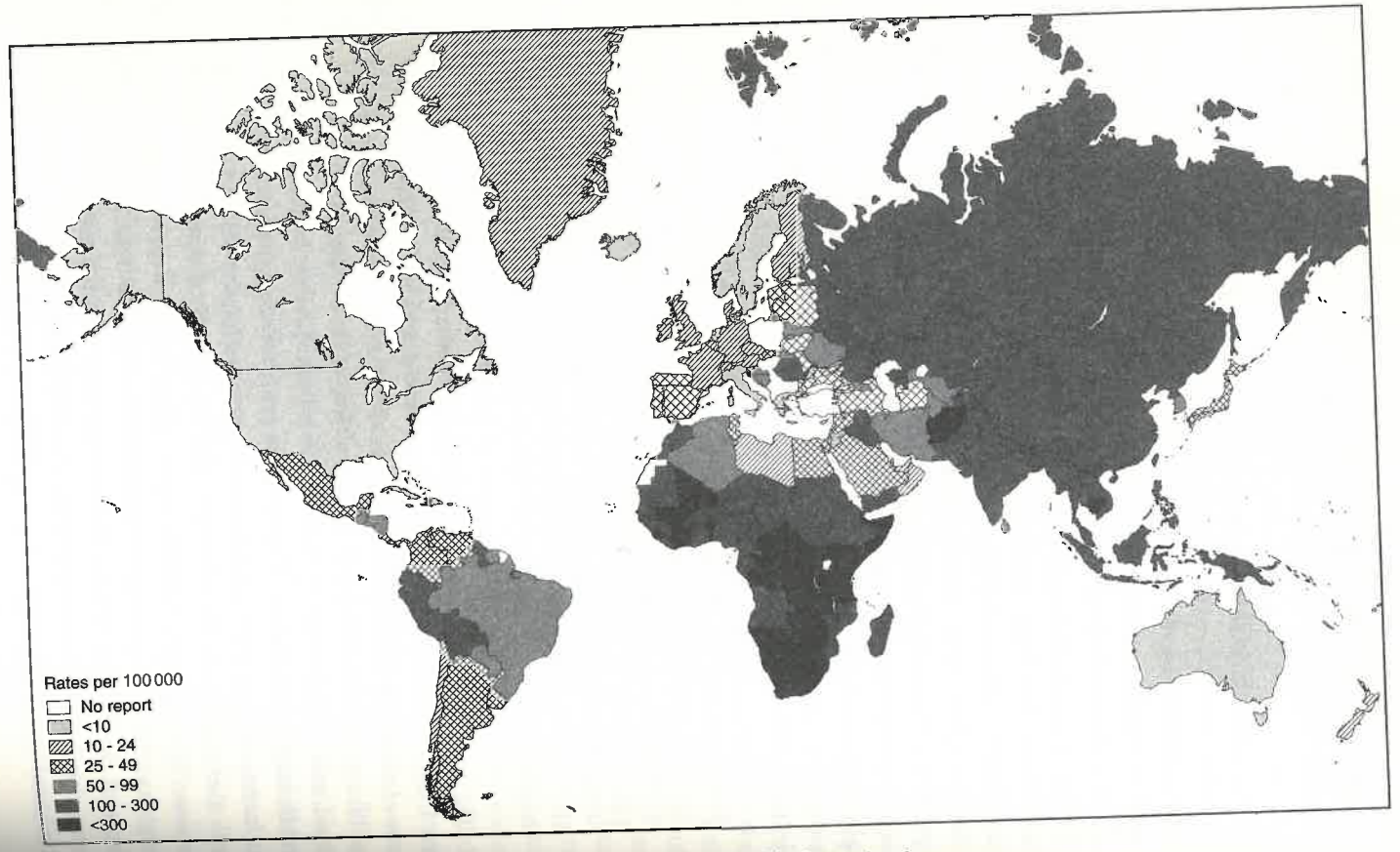


Figure 1.4 The global incidence of TB in 2001. Source: World Health Organization.

The development of drug resistance is thought to be responsible for around 10 per cent of new TB cases worldwide.[70] The problem of drug resistance was encountered soon after the discovery of streptomycin and other anti-TB drugs and led to the gradual emergence of multi-drug treatment programmes. Factors involved in the emergence of drug-resistance include the poor supervision of therapy, the use of badly prepared combination preparations, inconsistent prescribing practices, erratic drug supplies, and unregulated over-the-counter sales of drugs.[71] The most commonly encountered resistance is to a single drug, usually streptomycin or isoniazid, and most TB bacteria with such resistance respond adequately to a multi-drug treatment programme. The emergence of resistance to rifampicin is much more serious, however, as this is the most powerful anti-TB drug, with the ability to sterilize lesions by destroying near-dormant 'persister' bacilli. Furthermore, most rifampicin-resistant strains are also resistant to isoniazid; by convention, TB due to strains resistant to these two agents, with or without additional resistances, is said to be multi-drug-resistant. The use of standard short-course treatment becomes not only ineffectual but may even be positively harmful as resistance to other drugs such as pyrazinamide and ethambutol also develops as part of the so-called 'amplifier effect'.[72] In Russia and other states of the former Soviet Union, mutant forms of TB, variously referred to as multi-drug-resistant TB (MDR-TB), have been rapidly spreading in response to chronic overcrowding in the prison system and severe cutbacks in primary health care. The problems and costs of managing each case of MDR-TB are enormous. Successful therapy requires prolonged courses of less effective, more expensive and more toxic drugs, under long-term supervision.[73] The incidence of MDR-TB in New York City has been reduced by such a strategy, although at very great cost: the cost of the management of a single case can exceed US$250,000.[74] In the case of New York, the spread of MDR-TB was facilitated by reductions in public health expenditure during the 1980s, but the city ended up having to spend ten times more than it saved in order to bring TB under control.[75]

A second factor behind the resurgence of TB is the AIDS pandemic. This is estimated to contribute around 10 per cent of TB cases worldwide. In Africa, however, HIV is responsible for at least 20 per cent of TB cases.[76] Given that one third of the world's population carry quiescent TB infection the effects of immune system damage can be


Figure 1.4 The global incidence of TB in 2001. Source: World Health Organization.

expected to have devastating consequences: the most recent data suggest that in parts of sub-Saharan Africa, for example, more than one third of the adult population are now infected with HIV. Infection by HIV is currently the most important predisposing factor for the development of overt TB in those infected by TB before or after becoming HIV positive and by the late 1990s there were estimates of at least 11 million co-infected persons.[77] The increasing recognition of links between TB and HIV among patients has had the adverse effect of adding to the stigma of TB symptoms and has hindered cooperation between patients, health care workers and local communities.[78] The return of TB has also exposed tensions between different conceptions of medicine and individual liberty: in the USA, for example, the threat of MDR TB and co-infection with HIV has led to calls for punitive public health strategies based on mandatory screening and treatment, case notification to public agencies, aggressive contact tracing and the use of quarantine. Such measures, reminiscent of early-twentieth-century approaches to public health, are in conflict with contemporary conceptions of individual liberty.[79]

A third dimension to the 'new' TB is the effects of global social and economic change. Mass movements of people in response to war, increased economic insecurity, community breakdown and other factors have been involved in the spread of TB and other infectious diseases associated with overcrowding, makeshift housing and poor sanitation.[80] In addition to short-term disruption we must consider the longer-term social and economic shifts that have emerged since the early 1970s. There is now increasing evidence that growing poverty, infrastructural decay and declining health services have facilitated the spread of TB, diphtheria, sleeping sickness and other preventable diseases.[81] In the case of Vietnam, for instance, recent research has shown how the scaling down of established public health care systems during the 1990s has resulted in increased costs, more erratic drug availability and sinking morale among low-paid community health workers at the forefront of health care provision.[82] A substantial body of evidence suggests that TB has a disproportionate impact on the economically poor: 95 per cent of all TB cases and 98 per cent of TB deaths occur in the developing world where problems of ill health contribute towards cycles of economic hardship in the context of high unemployment and weak social security and health care provision. Similarly, the spread of TB and other preventable diseases in the so-

called de-developing enclaves of urban America and the poverty-stricken cities of the former Soviet Union can only be fully understood with reference to the dynamics of global political and economic change since the Second World War.[83] Changing patterns of economic and social investment have contributed towards a new geography of wealth and poverty with significant implications for the epidemiology of disease. With the advent of more diffuse patterns of urbanization and the greater mobility of capital investment it has become far easier for public health crises to be effectively ignored where they present no generalized threat to the overall well-being of an increasingly globalized economic system.

In 1948 the newly created World Health Organization defined health as 'a state of complete physical, mental and social well-being, not merely the absence of disease and infirmity'.[84] This definition rests on an explicit recognition of the connections between health care and wider ethical and political ideals, yet recent advances in bio-medicine have served to obscure any meaningful connection between health and social justice. The last thirty years has seen a shift from collective forms of health care to an increasing emphasis on health as an individualized dimension to personal development. The historical synergy between health reform and social justice has been displaced by an increasing emphasis on the individual patient (or consumer) rather than the wider social and political context for disease. The profit-driven restructuring of global health care has led to widening health inequalities as the world's poor find themselves unable to benefit from the latest technological and pharmaceutical advances. In comparison with other major health afflictions, TB remains relatively neglected: the funding of TB control worldwide, for example, continues to be very low in comparison with other infectious diseases: just $8 of external aid is spent for each patient death compared with $137 for malaria, $925 for AIDs and over $38,000 for leprosy.[85] Of the 1,240 new drugs that were licensed between 1975 and 1996, only thirteen dealt with the world's killer diseases that primarily afflict people from tropical and poor countries. In 1998, for example, the World Health Organization failed to persuade pharmaceutical leaders to collaborate over the development of a combined drug for TB to make public health campaigns simpler and more cost-effective because the potential profit margins were too low.[86]

The problems of poverty and community breakdown have had a

devastating effect on global public health and threaten to overwhelm the prospects for greater social cohesion and economic development. Whilst new technological advances may play a useful role in the treatment of TB the eventual eradication of the disease will rest on wider structural changes in modern societies. Most sufferers from TB have limited political and economic power and their plight remains of only marginal significance in global affairs. Yet the corrosive effect of ill health on social development threatens to expose the specious logic behind a new world order in which much of humanity is condemned to poverty and serfdom. If there is one lesson to be learned from the diseased cities of nineteenth-century Europe and North America, it is that the contemporary global public health crisis will be solved not by medical intervention but by political transformation.