GENERATING HEALTH FROM THE PATTERN OF DISEASE*

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AWAKENING OF INTEREST IN PUBLIC HEALTH AND SCOPE AND CENTRAL THEME OF THE LECTURE

At school, I was interested in politics, philosophy, economics and science, but in Medical School I was absorbed by biomedicine. By the fifth year of my studies, I had unexpectedly chosen a career in public health, so reviving earlier interests. The turning point came in my fourth year elective in Newfoundland and Labrador where I observed the stark contrast between the disease patterns of the poverty stricken Native Canadians (First Nations people) and those of the affluent European Canadians. In my fifth year elective in India I had the opportunity to contrast the relative contributions of clinical and public health medicine in serving rural and urban populations around New Delhi, cementing my resolve to work on disease prevention.

Simple observations on disease patterns can not only influence careers, but also yield deep insights of value to medicine and public health. Using three historical studies and three examples of my work, I will illustrate how epidemiology generates health from the pattern of disease. I will briefly trace the history of public health in Edinburgh. Finally, I will peek into the future of my speciality.

DEFINING BASIC CONCEPTS: HEALTH, PUBLIC HEALTH, EPIDEMIOLOGY

Defining health is problematic. Healthy people are alive (literally and metaphorically) and, ideally, energetic, functioning, free from pain and disease, feeling well and happy. The World Health Organisation's memorable rhetoric states that health is 'a state of complete, physical, mental, and social well-being and not merely the absence of disease or infirmity'. Public health is everything that is designed to improve health or, in the words of the Acheson Report: 'The science and art of preventing disease, prolonging life and promoting health through the organised efforts of society.'

These ideas are not new. My ancient Indian predecessor, Caraka, stated in the first century AD that concord is health, discord is disease; Susruta in the fourth century AD declared that his purpose was to prolong life and prevent disease. The word 'Ayurveda', a traditional and ancient form of Indian medicine, means knowledge on prolonging life. Many professions and institutions contribute to public health, and the speciality that takes the lead responsibility on behalf of the medical profession is, of course, public health medicine.

Epidemiology is the science that seeks to understand the causes of diseases by measuring their frequency and patterns in populations or, in the language of the profession, it is the study of the distribution and determinants of disease in populations, disease being any condition of the body in which its functions are disturbed or deranged.

A BRIEF HISTORY OF EPIDEMIOLOGY

Some 2,400 years ago Hippocrates wrote 'to know the cause of a disease and to understand the use of the various methods by which disease may be prevented amounts to the same thing in effect as being able to cure the malady'.[1] This was an astonishing statement, and remains a bedrock of modern medical science. Epidemiology was established as a discipline nearly 2,000 years later. Some early landmarks were the systematic recording in the sixteenth century of births, marriages and deaths in parish records; Graunt's (a haberdasher) examination of the pattern of mortality in 1662; Edmund Halley's (an astronomer) invention of life-tables in 1693; Villerme's (a surgeon) studies of inequalities in mortality in Paris in 1821 and the compulsory registration of births, deaths and marriages in 1836. Stark variations between populations in mortality were soon demonstrated. These could either be a result of differences in the constitution of the individual, in exposure to the agents of disease, or in the environment. The evidence pointed to the latter. Advocacy and action to reverse such inequalities, e.g. by making available clean water, sewage disposal and a safe working environment, founded modern public health. I have picked three historical examples to show how epidemiology transformed public health.

LIND ON SCURVY

Lind was born in Edinburgh and graduated MD (Edinburgh) in 1768. He reported in 1753 that 'scurvy alone, during the last war, proved a more destructive enemy, and cut off more valuable lives, than the united efforts of the French and Spanish wars' and that scurvy 'raged with great violence in some journeys, not at all in others'.[2] The first observation identified the immense size of the problem, the second told him that scurvy was preventable. He generated many hypotheses – including the sea climate and particularly the moist air – but chose to investigate diet and conducted his experiment on the ship *Salisbury* in 1747: he 'ordered' 12 sailors, divided in pairs, to take either cider, elixir vitriol, vinegar, sea-water, an electuary (made of garlic, mustard seed, radishes, balsam of Peru and gum myrrh), and oranges and lemons. He found that 'the most sudden and visible good effects were perceived from the use of the oranges and lemons'. Many lives were lost before his remedy was adopted. Vitamin C deficiency was later shown to be the cause of scurvy and this vitamin was synthesised in 1932. This story illustrates the importance of reflecting on the differing patterns of disease, and then generating and testing hypotheses. Lind's experiment is described on a plaque (donated by the Sunkist citrus grower of California and Arizona) in the Edinburgh Medical School quadrangle which proclaims him the Hippocrates of naval medicine.

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JENNER AND SMALLPOX

Smallpox, a viral infection which spreads via the respiratory tract, was a savage killer, the scourge of armies and the colonist's friend. The story of how Edward Jenner, a country practitioner in Gloucester, investigated the role of vaccination with cowpox virus is well known, but where did he get the idea? What was the observation that inspired him to take the cowpox virus from the hand of Sarah Nelmes and insert it into the arm of 'a lad of the name of Phipps' on 14 May 1796?[3] The observation was this: that milkmaids have clear complexions and are generally free of pockmarks and that it is hard to inoculate them using smallpox virus. Jenner investigated this and the local practice of exposing people to cowpox as a means of protecting against smallpox. He deduced that milkmaids' exposure to cowpox protected them from smallpox. If so, he thought, why not inoculate with cowpox, rather than with smallpox, the latter practice being widespread but risky. His gamble was to vaccinate Phipps, and then expose him to the smallpox six weeks later. Phipps did not react to the smallpox inoculation. Jenner was convinced he had demonstrated a new technique for the prevention of smallpox and he correctly forecast the disease would be eliminated. The World Health Organisation declared smallpox to be eradicated in 1980 – surely the supreme medical advance. Smallpox is the only disease yet eradicated through deliberate public health endeavour but guinea-worm infestation and poliomyelitis are likely to follow.

SNOW AND CHOLERA

The pandemics of cholera in the nineteenth century, sweeping from the East into Europe, led to wholesale riots, as thousands succumbed to this illness. At the time the miasma theory was favoured (i.e. atmospheric pollution arising from decaying organic matter). John Snow investigated this disease for 20 years, including 'the most terrible outbreak of cholera which ever occurred in this kingdom' – the epidemic of cholera in Broad Street, Soho.[4] He suspected contamination of the water in the Broad Street pump, a conclusion supported by his observations that the dead had lived or worked near the pump; a nearby workhouse and brewery had their own water supply and little cholera and people living far away but drinking Broad Street pump water were afflicted. The map (my commissioned revision of John Snow's original, Figure 1), shows the homes of people dying from cholera were clustered around the pump. He concluded that water, not miasma, was the source of cholera. He published in 1849 and 1855, and gave evidence to learned committees including one of the House of Commons. He was unable to convince those in power and died in 1858 before his ideas were accepted. John Snow's book cost him two hundred pounds to publish and he sold 56 copies in three years, making three pounds and 12 shillings. There may be a salutary lesson here for those who emphasise either peer review or indicators of popularity, such as the 'science citation index', in assessing the importance of research.

FIGURE 1
The Broad Street Epidemic.
Map showing places of residence of fatal cases and locations of pump wells. (Based on John Snow's original, drawn by the Department of Medical Illustration, Newcastle University, under R. Bhopal's supervision).

TABLE 1
Some key events in the early history of public health with particular reference to Edinburgh.*

Date	Events
1747	**James Lind (Edinburgh MD) conducts his experiment on scurvy on the ship *Salisbury*.**
1796	Edward Jenner vaccinates James Phipps.
1840	**William Pulteney Alison's *Observations on the Management of the poor in Scotland and its effects upon the health of the great towns* is published.**
1842	Edwin Chadwick's *Report on the sanitary conditions of the labouring population of Great Britain* is published.
1847	**First MoH in Britain, William Henry Duncan, is appointed in Liverpool – an Edinburgh graduate.**
1847	Rudolph Virchow investigates the typhus epidemic in Upper Silesia (Poland).
1847	Ignaz Semmelweis initiates the practice of washing hands with *chlorina liquida*.
1848	**Public Health Act creates General Board of Health. Thomas Southwood Smith, an Edinburgh student, appointed as medical advisor.**
1854	Board of Health discontinued.
	Cholera hits Soho.
1855	John Simon appointed as equivalent of the CMO.
1855	John Snow publishes *On the mode of communication of cholera*.
1862	**First MoH in Edinburgh is appointed: Henry Littlejohn.**
1871	First diploma in state medicine, Dublin.
1875	**First Postgraduate BSc in Public Health in UK led by Sir Henry Littlejohn.**
1878	Louis Pasteur argued the case for the germ theory of specific disease before the French Academy of Medicine.
1879	**Pioneering introduction of the notification of infectious diseases in Edinburgh by Henry Littlejohn.**
1882	Koch identifies the *tubercle bacillus*.
1884	**Pasteur attends 300th anniversary of Edinburgh University and enthuses Alexander Low Bruce who, with Younger Brewers and John Usher, fund the first British chair in public health.**
1888	Institute Pasteur founded in Paris.
1891	British Institute for Preventive Medicine is established (later called Jenner Institute, then Lister Institute).
1898	**First appointment to the Chair of Public Health at Edinburgh University (Charles Stewart).**
1902	**Usher Institute of Public Health opens.**

***Events in bold relate to Edinburgh.**

OTHER ADVANCES

Epidemiology and public health has advanced, together with other medical sciences, with triumphant insights into the causes and control of diseases including puerperal fever, pellagra, typhus, beriberi, congenital rubella, adenocarcinoma of the vagina, lung cancer, coronary heart disease, AIDS and sudden infant death syndrome. The list goes on. These landmarks showed how society could conquer disease by organised research and action.

ESTABLISHING PUBLIC HEALTH AND THE ROLE OF EDINBURGH

British public health took root in the nineteenth century, and Edinburgh was at the forefront in academic innovation and service leadership. Table 1 partially summarises the history of public health, with an Edinburgh bias. William Pulteney Alison, Professor of Medical Jurisprudence at Edinburgh University, prepared a report that was far-sighted in grasping the social basis of disease.[5] Henry Littlejohn, Professor of Medical Jurisprudence at Edinburgh University, was a pioneer, for example in the compulsory notification of infectious diseases and in applying epidemiology to the control of disease and the delivery of health services.

In 1884 Louis Pasteur attended the 300th anniversary celebrations of Edinburgh University. He enthused Alexander Bruce (Figure 2) who generated the impetus to fund a new chair in public health. Sadly, Alexander Bruce died in 1893 before his ambition was realised. The Younger firm of brewers and John Usher of Norton (Figure 3) contributed to his legacy towards the chair and funded the Usher Institute which opened in 1902. Table 2 lists the six professors who held the Alexander Bruce and John Usher chair before me. Doctor Una Maclean has written a splendid history of the Usher Institute of Public Health,[5] from which I have gratefully drawn, but the record of achievement needs completing. The centenary in 2002 will provide the motivation and opportunity to fill the gaps.

TABLE 2
Holders of the Alexander Bruce and John Usher Chair of Public Health at the University of Edinburgh.

1898–1924	Charles H. Stewart
1925–44	Brevet Colonel Percy S. Lelean
1944–55	Brigadier Francis A.E. Crew
1955–64; 1977–80	Sir John H.H. Brotherston
1964–75	Stuart L. Morrison
1983–97	William M. Garraway
1999–present day	Raj S. Bhopal

MY RESEARCH ON THE PATTERNS OF DISEASE

My own studies, for example on legionnaires' disease, the impact of industrial air pollution on the health of people living nearby, and ethnic variations in cardiovascular disease, derive from, and contribute to, the tradition of my predecessors.

FIGURE 2
Alexander Low Bruce.
(Photograph, date and photographer unknown)

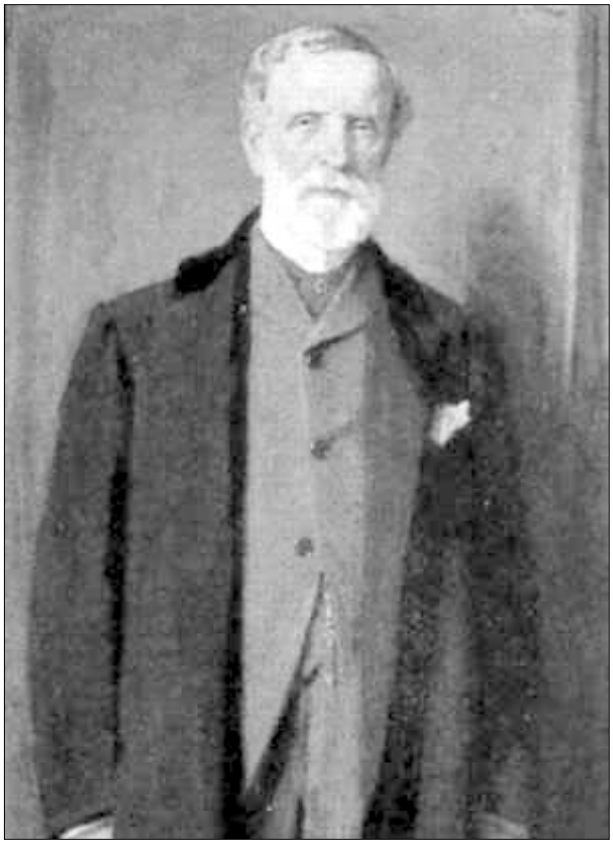

FIGURE 3
Sir John Usher of Norton and Wells (1828–1904).
(Oil painting by Sir George Reid PRSA, date unknown)

LEGIONNAIRES' DISEASE

Legionnaires' disease is a rare, environmentally acquired, pneumonia that may follow the inhalation of aerosols of water contaminated with legionnella bacteria. I participated in the investigation of the outbreak of 34 cases of legionnaires' disease in Glasgow in 1984, traced to a contaminated cooling tower located in Tennants brewery in Denniston.[6] One key, unanswered question in 1984 was: from which water sources do the aerosols that cause legionnaires' disease, especially sporadic infection, come from and what is their relative importance to each other? Is it aerosol from the home hot or cold water supply, spas and whirlpools, hot water systems in complex buildings, or industrial cooling towers that account for most infection? Without the answer we may be pouring millions, or billions, of pounds worth of destructive chemicals into the wrong water systems.

With colleagues I plotted the location of residence of cases of disease in Scotland, focusing on those considered to be sporadic. We found great variation in the incidence of apparently sporadic, non-outbreak disease.[7] I hypothesised that variation in the location of cooling towers was responsible for the clustering in sporadic cases. This hypothesis was tested on Glasgow data, by identifying cooling towers there. People who lived within 500 m of a cooling tower were three times more likely to develop legionnaires' disease than those who lived more than 1 km away.[8] Here, for the first time, was evidence that cooling towers were associated with apparently sporadic disease, offering a way of controlling the disease by focusing on the hygiene of cooling towers, rather than domestic water systems.

TEESSIDE STUDY OF ENVIRONMENT AND HEALTH

Teesside is the home of Britain's largest petro-chemical complex, and housing and industry there are side-by-side. Heavy industry is vital but there is a price to pay – pollution. People are becoming reluctant to pay this price, causing disruption to industries, through complaints, adverse publicity, litigation and public inquiries. Our research was carried out in the midst of heavy media publicity and pending litigation. General practitioners feared, as had successive medical officers of health for the last hundred years, that the pollution caused premature death, cancer, asthma and other chest problems. Routine statistics showed appallingly high mortality in the geographical areas, particularly Grangetown, close to industrial complex.

We hypothesised that the risk of mortality and disease, particularly for respiratory health, would be higher than expected close to industry. Death rates for lung cancer in women were exceptionally high and in line with the pattern predicted in our prior hypothesis.[9] For virtually every other cause of death and cancer, there was no such pattern; no evidence was found in favour of our prior hypothesis for birth weight, gender ratios and perinatal mortality in infants,[10] for self-reported health and for general practice consultation patterns.[9] We concluded that there was evidence that local industrial pollution had a causal role in the high rates of lung cancer in women, but that for a wide range of other health concerns alternative explanations were necessary.

Our work provided data to resolve a public health problem that had been simmering since the turn of the century.

ETHNIC VARIATIONS IN CARDIOVASCULAR DISEASE

The twentieth century was characterised by global migration, creating multi-ethnic societies. Britain's multi-ethnic populations provide new opportunities and challenges to epidemiology and public health. The ethnic mixing potentially creates a rich epidemiological source for describing interesting patterns of disease and for generating and testing epidemiological hypotheses. A word of warning is, however, essential. Study of racial and ethnic differences has beguiled science, trapping individuals and disciplines in the snare of racial prejudice.[11] Findings of racial differences, too readily interpreted as inferiority or superiority, provided a convenient justification for slavery, colonialism, unjust immigration policy, eugenics and the Nazis' 'final solution'. The first step in avoiding this perilous path is a valid definition of race and ethnicity. In science, race is the division of humankind that you belong to as a result of your genetically inherited characteristics – mainly skin colour, facial features and hair type. Genetics, biology and the study of variations in disease patterns have not supported any of the racial classifications created on the basis of these characteristics. Ethnicity, the population group that you belong to as a result of your shared ancestry, social customs, traditions, languages and other like factors that forge identity, is now superseding race. This concept is also challenging current classifications, but is proving useful, particularly in the inequalities in health arena.

One of the most important challenges for ethnicity and health research is cardiovascular disease. Adelstein and colleagues examined cardiovascular mortality in South African white (meaning of white European origin) and Asian (meaning people of Indian subcontinent origin) groups in 1963.[12] Asian groups had higher mortality than South African whites. This was surprising because cardiovascular diseases were at the time associated with northern European origins; Asian populations would be predicted to have fewer of the classical risk factors including smoking and meaty, high fat diets. Subsequent research demonstrated that people of Indian subcontinent origin living in urban settings in many countries have unexpectedly high cardiovascular mortality

The picture is complex,[13-15] but we clearly have a public health problem in South Asian populations. We also have a public health opportunity of maintaining the comparatively low rates of heart disease in other groups, e.g. the Chinese.[16] Our need is to understand the causes of these variations. South Asians are either more susceptible to cardiovascular diseases, or they are more exposed to the causal factors, or they get more cardiovascular disease because they are less likely to die of other causes, for example cancer. Presently, the first hypothesis is favoured, with the prediction that South Asians are more susceptible either for genetic reasons or because of early life metabolic programming. One major explanation is that the excess risk of heart disease in South Asians is mediated through their predisposition to diabetes and insulin resistance, in themselves coronary heart disease risk factors.[13] The explanation for the higher rate of insulin resistance is, in turn, usually assumed to be genetic. In my view the genetic explanation should not be accepted until the genes responsible have been pinpointed. The explanation that South Asians may have a higher level of risk factors has been too readily dismissed, with some dissent.[14,15]

I perceive a complex web of causes that needs disentangling carefully. The Newcastle multi-ethnic heart project was designed as a cross-sectional study with mortality follow-up and a multi-ethnic comparison.[15,16] We discovered vast differences between Indians, Pakistanis and Bangladeshis that question the value of previous studies of South Asians as a single population. For example, the smoking prevalence varied hugely, as did the lipid profiles.

The rapidity of the change in cardiovascular risk factors may itself be the cause of the enhanced susceptibility in South Asians. The underlying mechanism could be the mismatch between diet and metabolism *in utero,* in childhood and in adulthood. To test this and other key hypotheses my colleagues and I have proposed that Britain needs a new large-scale, multi-ethnic, cardiovascular cohort study. The proposed study would use new conceptual approaches, for example, comparing ethnic groups with each other and not simply against a single (usually) white reference population. Strong emphasis will be placed on examining variations within individual ethnic groups, thereby exploring heterogeneity and not suppressing it.

THE FUTURE OF PUBLIC HEALTH

Academic public health in Edinburgh has a proud history on which to build. Public health teaching will be increasingly important in both the undergraduate and postgraduate curricula, the latter continuing a 125-year tradition. As many important research questions derive from practical problems, we must ensure that in future research and practice intertwine better. The gap between theory and practice in research and education might be narrowed by practice-based undergraduate, masters and PhD level education.

In the twenty-first century technology and science will help to resolve current problems but will also accelerate change leading to new ones. In industrial countries the challenges will lie in the prevention and control of the diseases of older people whose lives will be supported by technological advances. Paradoxically, some of the solutions to these problems of older age lie in better maternal, fetal and infant health. Molecular science will deepen understanding of the interaction between the environment, lifestyle and the gene, for example demonstrating why some people have high serum cholesterol and how this causes atherosclerotic diseases. Yet, the public health dividend will come from altering the pattern of risk factors in the whole population. Reducing serum cholesterol from the currently pathological level of 6 mmol/l and more, to a physiologically normal value of 4 mmol/l or even less, without mass medication, requires an understanding of how people and societies change. It is not simply biochemistry that determines an individual and population's serum cholesterol level, but what and how food is grown, processed, purchased, cooked, eaten and metabolised. These factors are determined by more than personal taste: trade agreements, agricultural policy, marketing, economic subsidy and availability are crucial determinants of consumption.

Economic and health inequalities, particularly on a global scale, will hold centre stage in public health as they have done for two hundred years. Modern communication exposes the injustice of gross waste in some countries, and horrendous poverty in others – inequalities that would shock even Chadwick, Simon and Littlejohn. In many developing

countries there is a nightmarish combination of traditional public health problems of inadequate sanitation, inadequate nutrition and communicable diseases with those of the post-industrial era, i.e. cancer, heart disease, stroke and road traffic accidents. The academic challenge is the timely and low cost transfer of knowledge and technology to combat or prevent these problems. The social and political challenge is to tackle poverty, which is a potent and direct cause of ill health (and *vice versa*).

Public health must apply medical sciences including epidemiology in the social and political context, inevitably exposing the tensions between the responsibility of the individual and of the state. It is the role of universities to find, educate, and inspire the future leaders who will resolve these tensions. Epidemiology will help them to envision the health needs of their nations and articulate the coherent policies, laws and health care systems needed to nurture healthy populations. It is the privilege of academic public health and epidemiology to pass on the gift of generating health from the pattern of disease.

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