Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Available online at www.sciencedirect.com

ScienceDirect

Procedia Social and Behavioral Sciences 2 (2010) 7339–7346

Procedia
Social and Behavioral Sciences

Selected Papers of Beijing Forum 2005

Building the Capacity of Schools to Improve the Health of Nations

Lloyd J. Kolbe

Indiana University

INTRODUCTION

Humankind has entered a new era. In November, 2005, the U.S. Centers for Disease Control and Prevention counted 50 emerging and re-emerging communicable diseases, as listed in Table 1.[1] Certainly these, and other equally formidable, threats to individual and global health will require new approaches for the 21st Century.

Indeed, as part of rapidly evolving public health reform in the United States, the Association of Schools of Public Health has identified 12 core competencies by those who seek the Master of Public Health (MPH) degree.[2] These core competencies include five discipline-specific competency lists in: Biostatistics, Environmental Health Sciences, Epidemiology, Health Policy and Management, and Social and Behavioral Sciences. The core competencies also include a list of interdisciplinary/cross-cutting competencies across seven overarching domains in: Communication and Informatics, Diversity and Culture, Leadership, Professionalism, Program Planning, Public Health Biology, and Systems Thinking.

Systems Thinking.

To be brief, our traditional means of managing a given public health threat generally have focused more on separating, analyzing, and addressing individual components of the threat (including components of the health threat and strategies to resolve it). In contrast, systems approaches to managing a given public health threat generally focus on: critical inter-relationships among components of the health threat, other health threats, and strategies to address the given health threat and related threats. Leischow and Milstein[3] explain that:

> ... there is no single discipline for systems thinking, precisely because it is oriented to the linkage of disciplines. Equally important is an emphasis on relating different types of structures that shape our lives, including the biological systems of our bodies, the \[public health and other\] organizational systems in which we work, and the political systems with which we govern public affairs. Although there is no simple operational method for identifying and interpreting these relationships, there is, in fact, a common conceptual orientation recognizable as a systems approach: it is a paradigm or perspective that considers connections among different components, plans for the implications of their interaction, and requires transdisciplinary thinking as well as active engagement of those who have a stake in the outcome to govern the course of change.

Genetic, Non-Communicable, Environmental, & Communicable Health Threats

Illustrative of systems thinking, although it proves helpful to categorize health threats, we usefully are beginning to understand critical relationships among such categories.[4] Illustratively, we have learned that genetic factors, in addition to contributing to genetic diseases, also can determine significant susceptibility or significant resistance to non-communicable, environmental, and communicable health threats. Further, although classified as a non-communicable disease, asthma can be caused or exacerbated either by infectious agents, by environmental

doi:10.1016/j.sbspro.2010.05.090

contaminants in the air, or by both. We recently have learned several diseases previously thought to be non-communicable have been linked to infectious agents: peptic ulcer disease with Heliobacter pylori, cervical cancer with several human papillomaviruses, liver cancer and cirrhosis with hepatitis B and C viruses, to name a few. Evidence also is accumulating that microorganisms might cause or exacerbate atherosclerosis and cardiovascular disease, type 1 diabetes, inflammatory bowel disease, neurological diseases and neuropsychiatric disorders, and developmental problems among children. Environmental geographic agents (arsenic, iodine insufficiency) agricultural agents (pig, chicken, duck, and cattle production; herbicides and pesticides), and industrial agents (PCB, dioxin), might precipitate or aggravate diseases such as various cancers, goiter, mental retardation, Rift Valley Fever, bovine spongiform encephalopathy, pfiesteria, severe acute respiratory syndrome (SARS), H5N1 (Avian) influenza, neurological and developmental disorders, and compromised immunity.

Simultaneous Global Threats to the Public's Health in the 21st Century

As we enter the 21st Century, humankind faces at least the following 16 discrete, yet inter-related, global threats to the public's health. Addressing one or two of these threats alone will require resources and collaboration heretofore unknown in the history of our species. Addressing all 16 simultaneously will require new strategies; and, more important, a new global ethic. These global threats include: (1) populations increasing, urbanizing, poor, famined, and migrating; (2) increasingly toxic environments, deforestation, and insufficient or undrinkable water; (3) global air pollution, warming, dimming, climate changes, storms, floods, and droughts; (4) emerging and re-emerging diseases and global pandemics; (5) antimicrobial resistance; (6) rapid as well as slower transportation available to masses of people; (7) chronic diseases from environments and behavioral patterns; (8) unintentional and intentional physical and psychological injuries; (9) conventional, biological, chemical and radiological terrorism; (10) war undeclared, declared, intra-national, international, and nuclear; (11) natural disasters, including earthquakes, tsunamis, volcanoes, possible asteroids; (12) demographic extremes, aging and adolescent populations; (13) rapid changes in cultures; (14) stress, anxiety, depression, dementia, and consequently suppressed immune systems; (15) increasing economic globalization and economic disparities; and (16) increasing health disparities, underserved, and uninsured populations.

Public Health Reform

Public health workers, academicians, and leaders rapidly are applying systems thinking to reform our very conceptualization, organization, training, practice, and evaluation of efforts to protect and improve the public's health. In 1988, the Institute of Medicine Committee redefined public health as "what we as a society do collectively to assure the conditions in which people can be healthy."[5] In 2003, the Institute of Medicine Committee on The Future of the Public's Health in the 21st Century articulated the reasons for, and the nature of, public health reform as follows.[6]

> Health is a primary public good because many aspects of human potential... are contingent on it. In view of the value of health to employers, business, communities, and society in general, creating the conditions for people to be healthy should... be a shared social goal... The special role of government must be allied with the contributions of other sectors of society. The emphasis on an intersectoral public health system does not supersede the special duties of the governmental public health agencies, but, rather, complements it with a call for the contributions of other sectors of society that have enormous power to influence health...
>
> A public health system would include the governmental public health agencies, the health care delivery system, and the public health and health sciences academia, sectors that are heavily engaged and more clearly identified with health activities...\[But it also would include\] communities and their many entities (e.g. schools, organizations, and religious congregations), businesses and employers, and the media as potential actors in the public health system...

Such a public health system would comprise "...a complex network of individuals and organizations that, when working together, can represent 'what we as a society do collectively to assure the conditions in which people can be healthy.'"[6] A graphic depiction of A Public Health System for the 21st Century is represented by Chart 1.[7] Such a system increasingly will require collaboration and integration of efforts across public-sector, not-for-profit \[or civil\]

sector, and private-sector agencies. Importantly, it also will require substantially increasing collaboration among nations to improve the public's health in any one nation and among all nations.

Training Public Health Workers

Training not only health department workers, but workers in all other agencies of the public health system

Training not only about previous local health threats, but about global health threats in the 21st Century

Training not only about independent health threats and independent agency responses, but about simultaneous (syndemic) health threats and public health system responses

Health Literacy: Educating the Public and Decision-Makers

A New Ethic for a New Era

References

[4]Institute of Medicine, Board on Global Health, Forum on Microbial Threats. The infectious etiology of chronic diseases: Defining the relationship, enhancing the research, and mitigating the effects. Washington, DC: The National Academies Press, 2004.

Table 1

Emerging and Re-Emerging Communicable Diseasesd

1. rug-resistant infections (antimicrobial resistance)
2. bovine spongiform encephalopathy (Mad cow disease) and variant Creutzfeldt-Jakob disease (vCJD)
3. campylobacteriosis
4. Chagas disease
5. cholera
6. cryptococcosis
7. cryptosporidiosis (Crypto)
8. cyclosporiasis
9. cysticercosis
10. dengue fever
11. diphtheria
12. Ebola hemorrhagic fever
13. Escherichia coli infection
14. group B streptococcal infection
15. hantavirus pulmonary syndrome
16. hepatitis C
17. hendra virus infection
18. histoplasmosis
19. HIV/AIDS
20. influenza
21. Lassa fever
22. legionnaires' disease (legionellosis) and Pontiac fever
23. leptospirosis
24. Listeriosis
25. Lyme disease
26. malaria
27. Marburg hemorrhagic fever
28. measles
29. meningitis
30. monkeypox
31. MRSA (Methicillin Resistant Staphylococcus aureus)
32. Nipah virus infection
33. norovirus (formerly Norwalk virus) infection
34. pertussis
35. plague
36. polio (poliomyelitis)
37. rabies
38. Rift Valley fever

39. rotavirus infection
40. salmonellosis
41. SARS (Severe acute respiratory syndrome)
42. shigellosis
43. smallpox
44. sleeping Sickness (Trypanosomiasis)
45. tuberculosis
46. tularemia
47. valley fever (coccidioidomycosis)
48. VISA/VRSA-Vancomycin-Intermediate/Resistant Staphylococcus aureus
49. West Nile virus infection
50. yellow fever

A large body of epidemiological evidence suggests an association between ambient air pollution and cardiovascular mortality and morbidity, but the majority of these studies have been conducted in the developed Western world. The major risks have been linked to SO2 and PM. Sulfur dioxide is associated with increased daily mortality from both respiratory and cardiovascular disease even at the low levels now observed in the Western hemisphere.

Large cohort studies, using the same protocol, have been conducted in Western Europe based upon 29 cities (APHEA; Katsouyanni et al, 2001) and the US based upon 90 cities (NMMAPS; Samet et al, 2000), while a small number of studies have been conducted in Asia. The estimates of percent change per 10 ug/m3 increase in pollutant concentration on all-cause mortality showed slight variation, ranging form 0.41 in Asia and US to 0.6 in Europe for PM10, whereas it was 0.35 for Asia and 0.40 in Europe for SO2. PM2.5 appears to be more closely associated with mortality than PM10 or total suspended particulate matters (TSP). However, most of these studies have been conducted in the US (Pope et al, 2002). There are some concern using these data for estimation of risk in other populations due to difference in methods collecting and analyzing the concentrations of the particle and the chemical composition, e.g. different sources, different ratio between fine and coarse particles.

Long term exposure to ambient PM contributes to systemic inflammatory pathways, which are relevant aspects of atherogenesis resulting in atherosclerosis and ultimately cardiovascular death. Carotid artery intima-media thickness (CIMT) is normally considered a risk indicator of atherosclerosis. A recent epidemiological study has shown an association between PM2.5 and CIMT, and indirectly the risk of atherosclerosis. The risk was higher in women with a 15% increase in CIMT for a 10 ug/m3 increase in PM2.5 (K nzli et al, 2005).

Increased risk for infant and child mortality from respiratory diseases has been associated with exposure to air pollution both in developed and developing countries (Romieu et al, 2002).

Respiratory disorders

Exposure to traffic related air pollution has been implicated in impairment of respiratory health in children. Truck traffic and air pollutants associated with truck traffic were associated with chronic respiratory symptoms in Dutch school children living close to motorways, whereas there was no association with car traffic. Furthermore, there was no association with bronchial hyperresponsiveness in these children, however sensitization to outdoor allergen was increased in the group exposed to high level of air pollution (Janssen et al, 2003). The increased sensitivity to outdoor allergen may be a consequence of the fact that diesel exhaust particles modifies the allergen and enhances the immunologic response of the allergen and increase the inflammatory responses as demonstrated both in animal studies and human volunteer studies (Diaz-Sanchez et al, 2000).

Air pollution has not been found to induce asthmatic conditions in children, but will exuberate an already existing condition.

Cancer

Epidemiological studies have indicated that in Western countries the relative risk of developing lung cancer is 1.5-fold higher in urban that in rural areas after adjusting for cigarette smoking. Air pollution, e.g. vehicles, industry, power plants, has been reported to increase the risk of lung cancer (Vinies et al, 2004). The risk of lung cancer death has been suggested to increase by 8% for every 10 micrograms of fine particles/m3 of inhaled air (Nafstad et al, 2003). Epidemiological studies have also shown strong support for that the higher risk of lung cancer among non-smoking women in Xuanwei County, China is associated with the use of smoky coal for cooking and heating in homes without chimneys. The smoke of smoky coals is high in carcinogenic PAH (Keohavong et al, 2005).

Exposure assessment

Numerous epidemiological studies have reported association between outdoor concentrations of air pollutants and adverse health effects. Most studies have been using ambient monitoring data to estimate exposure. However, in case of PM, personal exposure is dominated by indoor sources especially taking into consideration that people spent up to 90% of their time indoor, thus the validity of using ambient concentrations as an exposure estimate may introduce misclassification. The risk of misclassification is less for gaseous air pollutants except in cases where a significant in-door source is present, e.g. nitrogen oxides. An alternative to ambient and personal measurement is the use of biomarkers to assess exposure, and a further advantage of these biomarkers is that they also reflect the time-activity pattern.

Biomarkers

The ultimate biomarker represents both an accurate exposure estimate and is also an indicator of health. Several biomarkers have been developed to assess exposure for genotoxic compounds in ambient air. Some of these biomarkers represent the exposure for specific compounds, e.g. benzene in blood and urine, and its metabolites, muconic acid and phenylmercapturic acid, in urine. However, the level of these products is not linked to the genotoxic effect. 1-hydroxypyrene (1-HOP) is another commonly used biomarker for PAH exposure although pyrene is not specific for ambient air pollution and can also be found in food. The carcinogenic PAH found to be associated with particles are metabolized to reactive molecules that can react with DNA to form bulky-DNA adducts. DNA adducts tend to be higher among subjects heavily exposed to urban and occupational pollutants (Autrup et al, 1999; Peluso et al, 2001). PAH-DNA adducts have also been detected in the blood from newborns, whose mothers were living in polluted areas of Poland and China. The adduct level was similar in mothers and in the child (Perera et al, 2005) suggesting that carcinogenic agents present in ambient can pass the placental barrier and initate damage in the unborn child that is relevant for carcinogenesis. A positive association has been established between the level of PAH in ambient air and the bulky adduct level at medium to high level of PAH, but not at the low level situation generally observed at ambient pollution. A weak but positive association has been established between the bulky adduct level and risk of development of lung cancer (Peluso et al, 2005; Bak et al, 2005) Particles generated by combustion are composed of a carbon core to which other compounds such as metals and PAH adhere. The particles do induce oxidative stress mediated by a particle-induced inflammation causing macrophages to release ROS. Oxidative stress to DNA is frequently measured as 8-oxodG. In a study among human volunteers we found a positive association between individual PM exposure and 8-oxodG, whereas no association was found between amount of PM and bulky adducts (S rensen et al, 2003). One of the consequences of DNA adducts, when un-repaired, is the induction of mutations that may ultimately induce cancer formation. An increased frequency of mutations in the p53 cancer suppressor genes has been observed both in the sputum of individuals exposed to smoky coal emission and in lung tumors from people in the region. The most frequently observed mutation seen in this group is a G to T transversion that is commonly associated with exposure to carcinogenic PAH (Keohavong et al, 2005).

Susceptible populations

Increased risk for adverse health effects due to air pollution could be related either to higher dose, acquired predisposing diseases and genetic susceptibility.

In the group exposed to higher dose are people living or working along highly trafficked roads, people with long commuting hours, or people who are occupationally exposed, e.g. bus drivers, traffic police officers. This group does also include children as their body burden with air pollutants is higher than adduct due to a relative high ventilation rate.

Elderly subjects, and especially subjects with pre-existing heart and lung disease are more susceptible to the effect of short-term ambient air pollution on mortality and morbidity. Asthmatics do also respond stronger than non-asthmatic on increase in air pollution, particularly PM. In case of long-term exposure, socially disadvantaged and poorly educated populations respond more strongly in term of mortality.

The toxic effects of air pollutants are modified by genetic variation in genes involved in, e.g. inflammatory processes, defense against reactive oxygen species formed by particulate matters, enzymes involved in the detoxification of PAH and other toxic compounds present in ambient air.

Prevention

Improving air quality will improve the quality of life and have important impact on economic development. Mobile sources are the major contributor to ambient air pollution in developed countries. Thus, stringent standards for new sources of air pollution (especially motor vehicles) will significantly improve air quality. However, the increasing economic welfare will result in an increasing number of vehicles and longer commuting distances, so the net benefits of these standards may be negligible. As the level of the pollution depends on the engine type and condition, fuel type, traffic congestions, driving habits and load of vehicles stricter control and introduction of cleaner technologies, e.g. hybrid cars. Furthermore, improved city planning to minimize traffic congestions and development of public transportation may help in minimize the air pollution in the megacities and improve the quality of life.

References

Ackermann-Liebrich U, Rapp R (1999) Epidemiological effects of oxides of nitrogen, especially NO2. In: Air pollution and Health, Academic Press, London England.

Arlt VM, Bieler CA, Mier W, Wiessler M, Schmeiser HH (2001) DNA adduct formation by the ubiquitous environmental contaminant 3-nitrobenzanthrone in rats determined by p32-postlabelling. Int J Cancer 93: 450-454.

Autrup H, Daneshvar B, Hansen M et al (1999) Biomarkers for exposure to ambient air pollution-comparison of carcinogen-adduct levels with other markers of genotoxic exposure and markers for oxidative stress. Environ Health Perspectives 107: 233-238.

Bak H, Autrup H, Thomsen BL et al. (2005) Bulky DNA adducts as risk indicators of lung cancer in a Danish case-cohort study. Int J Cancer, In press.

Bofetta P, Dosemeci M, Gridley G et al (2001) Occupational exposure to diesel engine emissions and risk of cancer in Swedish men and women. Cancer Cause Control 12: 365-374.

Cassoni F, Bocchi C, Martino A et al (2004) The salmonella mutagenicity of urban airborne particulate matter (PM2.5) from eight sites of the Emilia-Romagna regional network (Italy). Sci Total Environment 324: 79-90.

Diaz-Sanchez D, Panichet-Garcia M, Saxon A (2000) Diesel exhaust particles directly induce activated mast cells to degranulate and increase histamine levels and symptom severity. J Allergy Clin Immunol 106: 1140-46.

Ebelt ST, Wilson WE, Brauer M (2005) Exposure to ambient and nonambient components of particulate matter. Epidemiology 16: 396-405.

Fanou LA, Mobio TA, Autrup H et al (2005) Survey of air pollution in Cotonou, Benin air monitoring and biomarkers. Sci Total Environment In press.

Georgiadis P, Autrup H, Kyrtopoulos S et al (2001) Biomarkers of genotoxicity of air pollution (the AULIS project): bulky DNA adducts in subjects with moderate to low exposures to airborne polycyclic aromatic hydrocarbons and their relationship to environmental tobacco smoke and other parameters. Carcinogenesis 22: 1447-1457.

Harrod KS, Jaramillo RJ, Berger JA et al (2005) Inhaled diesel engine emissions reduce bacterial clearance and exacerbate lung disease to Pseudomonas Aeruginosa infectionIn vivo. Tox Sci 83: 155-166.

HEI (2004) Health effects of outdoor air pollution in developing countries of Asia: A literature review. Special Report 15, Health Effect Institute, Cambridge, MA.

Janssen NAH, Brunekreef B, van Vliet P et al (2003) The relationship between air pollution from heavy traffic and allergic sensitization, bronchial hyperresponsiveness, and respiratory symptoms in Dutch schoolchildren. Environ Health Perspec 111: 1512-1518.

Johansen K, Gabrielsson P. Autrup H et al (1997) Effect of upgraded diesel fuels and oxidation catalysts on emission properties, especially PAH and genotoxicity. SAE Technical paper series 973001: 1-5.

KEI (2004) Air pollution in megacities of Asia. Benchmarking report on urban air quality management and practice in major and megacities of Asia. Stage 2, Korean Environmental Institute, Seoul, Korea, In press.

Keohavong P, Lan Q, Gao W-M et al (2005) Detection of p53 and K-ras mutations in sputum of individuals exposed to smoky coal emissions in Xuan Wei country, China, Carcinogenesis 26: 303-308.

Katsouyanni K, Touloumi G, Samoli E et al (2001) Confounding and effect modification in the short term effects of ambient particles on total mortality. Epidemiology 12: 521-531.

Krewski D, Burnett RT, Goldberg MS et al (2000) Reanalysis of the Harvard six-cities study and the American Cancer Society study of particulate air pollution and cancer. Health Effect Institute, Cambridge MA.

K nzli N, Jerrett M, Mack WJ et al (2005). Ambient air pollution and atherosclerosis in Los Angeles. Environ Health Perspectives 113: 201-206.

Kwon HJ, Cho SH, Chun Y et al (2002) Effects of the Asian dust events on daily mortality in Seoul Korea. Environ Res 90: 1-5.

Lei Y-C, Chan C-C, Wang P-Y et al (2004) Effects of Asian dust event particles on inflammatory markers in peripheral blood and bronchoalveolar lavage in pulmonary hypertensive rats. Environ Res 95: 71-76.

Nafstad P, Haheim LL, Oftedal B et al (2003) Lung cancer and air pollution: a 27 year follow up of 16209 Norweigian men. Thorax 58: 1071-1076.

Norramit P, Cheevaporn V, Itoh N, Tanaka K (2005) Characterization and carcinogenic risk assessment of polycyclic aromatic hydrocarbons in the respirable fraction of airborne particles in the Bangkok Metropolitan Area. J Health Sci 51: 437-446.

Peluso M, Ceppi M, Munnia A, Puntoni R, Parodi S (2001). Metanalysis of thirteen p32-DNA postlabelling studies of occupational cohorts exposed to air pollution. Am J Epidemiol 153: 546-158.

Peluso M, Munnia A, Hoek G, Kryzanowski M, Autrup H et al.(2005) Bulky DNA adducts and lung cancer risk: a prospective study. Cancer Res.

Perera F, Tang D, Whyatt R et al (2005) DNA Damage from polycyclic aromatic hydrocarbons measured by benzo(a)pyrene-DNA adducts in mothers and newborns from Northern Manhattan, the World Trade Center area, Poland and China. Cancer Epi Biomarker Preven 14: 709-714.

Pope III, CA, Burnett RT, Thun MJ et al (2002) Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA 287: 1132-1141.

Romieu I, Samet JM, Smith KR, Bruce N (2002) Outdoor air pollution and acute respiratory infections in children in developing countries. J occup Environ Med 44:640-649.

Ruchirawat M, Mahidol C, Autrup H et al (2002) Exposure to genotoxins present in ambient air in Bangkok, Thailand particle associated polycyclic aromatic hydrocarbons and biomarkers. Sci Total Environ 287: 121-132.

Samet J, Zeger SL, Dominici F et al (2000) The national morbidity, mortality and air pollution study. Research report 94, Health Effect Institute, Cambridge, MA.

Schlesinger RB, Cassee F (2003) Atmospheric secondary inorganic particulate matter: the toxicological perspective as a basis for health effects risk assessment. Inhal Toxicol 15: 197-235.

Steerenberg PA, Withagen CE, van Dalen WJ et al (2005) Dose dependency of adjuvant activity of particulate matter from five European sites in three seasons in an ovalbumin-mouse model. Inhal Toxicol 17:133-145.

S rensen M, Autrup H, Hertel O et al (2003) Personal exposure to PM2.5 and biomarkers of DNA damage. Cancer Epi Biomarker Preven 12: 191-196.

Ueng T-H, Hung C-C, Kuo M-L et al (2005) Induction of fibroblast growth factor-9 and interleukin-1(gene expression by motorcycle exhaust particulate extracts aand benzo(a)pyrene in human lung adenocarcinoma cells. Tox Sci 87: 483-496.

Vinies P, Forastiere F, Hoek G, Lipsett M.(2004) Outdoor air pollution and lung cancer: Recent epidemiological evidence. Int J Cancer 111: 647-652.

WHO (2002) The world health report 2002: Reducing risks, promoting healthy life. WHO Geneva, Switzerland.