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JUNE 15, 2011

THE CLEAN AIR ACT AND PUBLIC HEALTH

UNITED STATES SENATE, COMMITTEE ON ENVIRONMENT & PUBLIC WORKS

ONE HUNDRED AND TWELFTH CONGRESS, FIRST SESSION

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Hearings

Statement of Barbara Boxer
Hearing: Full Committee hearing entitled, "The Clean Air Act and Public Health."
Wednesday, June 15, 2011
(Remarks as prepared for delivery)

I called this hearing to conduct oversight on one of the most successful and significant public health statutes in our nation's history, the Clean Air Act.

Before President Richard Nixon signed the Clean Air Act into law in 1970, the nation's air was heavily polluted in many places.

For example, a fog of pollution covered Donora, Pennsylvania, for five days in 1948. Records indicate that 20 people died, 6,000 people were sickened, and hundreds were evacuated as a result of the pollution.

In another tragic case, the eastern United States was blanketed by harmful smog in 1966. Scientists and researchers eventually concluded that the smog caused the deaths of 24 people per day over a period of six days.

The Clean Air Act, which has deep bipartisan roots, changed that. President Richard Nixon recognized the value of the Clean Air Act when he said: "I think that 1970 will be known as the year of the beginning, in which we really began to move on the problems of clean air...for the future generations of America."

When President George Bush signed the Clean Air Act Amendments in 1990, he said: "I take great pleasure in signing [the legislation] as a demonstration to the American people of my determination that each and every American shall breathe clean air...."

Now, 40 years after the Clean Air Act was created, many of the benefits to public health are clear and measurable. Let me show you how successful this landmark environmental law has been in protecting children and families in my State of California.

In 1976, there were 166 days when health advisories were issued in Southern California to urge people with asthma and other people with lung sensitivities to stay indoors. In 35 years, the number of smog-related health advisories issued in Southern California dropped from 166 days in 1976 to zero days in 2010.

While the Clean Air Act has dramatically improved health safeguards, more work remains to be done. A 2011 report by the American Lung Association shows that 154 million people live in areas with levels of toxic soot and smog pollution that current science demonstrates is dangerous.

Last year, the Pittsburgh Post-Gazette reported on an oily, black rain of pollution from an electric utility company that coated a local community in 2006. Because of the potential impact of the pollution on public health, local farmers were told that livestock should not graze in their fields, and families were told not eat fruits and vegetables from their own gardens.

In 2008, USA Today ran a series on toxic air pollution near our nation's schools. I asked EPA Administrator Jackson to help monitor for such threats, and now the Agency is focused on

addressing sources of toxic air pollution near schools.

The EPA is also helping my constituents in Mecca, CA, where an odor emanating from a soil recycling plant has made people sick, particularly students and teachers at two nearby schools.

Under the Clean Air Act, EPA is required to strengthen protections if scientific data indicates that pollution adversely impacts public health, including children's health. Recently, EPA proposed much-needed federal safeguards to reduce toxic air pollution from old power plants by requiring the use of modern pollution controls. These proposed safeguards would reduce mercury, lead, and chromium, which are known to cause cancer and birth defects.

When EPA reduces toxic air pollution, it helps families and children in communities across our country. EPA recently conducted a Congressionally-required, peer-reviewed analysis of the Clean Air Act that showed overwhelming health benefits now and into the future. The annual benefits by 2020 will include preventing:

• More than 230,000 premature deaths;
• 200,000 cases of heart attacks;
• 2.4 million cases of asthma attacks;
• 120,000 emergency room visits; and
• 5.4 million lost school days.

In contrast to the unsupported claims by some polluters who argue that health threats from mercury and other air pollutants are "exaggerated," we will hear today from EPA Administrator Jackson and representatives from the American Academy of Pediatrics, American Nurses Association, and the American Thoracic Society, who are experts on the issue. These witnesses will describe the critical steps that have been taken to reduce dangerous air pollution, and the important work that remains to be done.

Before I turn to Ranking Member Inhofe, I would like to personally thank EPA Administrator Jackson for the Agency's actions to help residents in Mecca, CA, who fell ill because of noxious odors from a waste recycling facility. Two weeks ago, I visited with children, teachers, and parents from Saul Martinez Elementary School who were adversely impacted by pollution affecting the community, and I am pleased that EPA is now working with the state and local governments to address the situation.

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Hearings

Statement of James M. Inhofe
Hearing: Full Committee hearing entitled, "The Clean Air Act and Public Health."
Wednesday, June 15, 2011

Madam Chairwoman, I appreciate your having today's hearing. This is the first time this session we've had Administrator Jackson here to discuss the Environmental Protection Agency's (EPA) air quality regulations. I'm glad you're here. I'd also like to thank the witnesses on our second panel. Cathy Woollums, I'm anxious to hear how EPA's regulations are affecting your rate payers. Dr. Brenner, I look forward to learning more about how energy price increases and unemployment affect public health. I think your testimony will be particularly insightful in light of the sweeping job losses we expect from EPA's rules.

Over the past two years, the Obama EPA has moved forward with an unprecedented number of rules that will have enormous consequences for families, businesses, and the nation's fiscal well-being. Take for example, EPA's new greenhouse gas (GHG) cap and trade regulations. Administrator Jackson, you have admitted that regulating GHGs in the U.S. will have no impact on global GHG concentrations, yet your rules will come at an estimated cost of $300 to $400 billion annually. The Agency's voluntary reconsideration of the national ambient air quality standards for ground-level ozone – a decision based on outdated data that could lead to significant economic constraints on the country – is an another Agency action of dubious merit. EPA projects the cost of this rule in the order of $90 billion. Meanwhile, the Agency is planning to tighten the standards again in just two years.

The Obama EPA is aggressively moving forward to regulate nearly all aspects of American life – it now has regulations covering dust on farms and puddles of water along the side of road. And it is businesses and working families who will pay the price.

Today we have a witness from the electric power industry with us, so let's focus on the regulations affecting her business for a minute. Just last week, in response to EPA's rules, American Electric Power (AEP) announced they would be forced to close nearly 6,000 Megawatts of low cost (coal) power generation. As a consequence, AEP estimates nearly 600 power plant workers will lose their jobs, totaling nearly $40 million in annual wages. These are good paying jobs in rural areas of Virginia, West Virginia,

Kentucky, Ohio, Indiana and Texas. These jobs won't easily be replaced.

Of course, the effects to the communities will be far greater than these direct job losses alone, as electricity prices increase and nearby businesses suffer in the wake of plant closures. A recent report by National Economic Research Associates (NERA) anticipates this will be replicated across the country, with an estimated 48 Gigawatts in plant closures. And this is from just two of EPA's rules. That's the AEP tragedy eight times over. And before this analysis is criticized, let me say that it is consistent with multiple projections, including that of Obama's Department of Energy, which estimates that plant closures could be as high as 70 Gigawatts. NERA goes on to predict that these two rules – the "Utility MACT" and the "Transport Rule" – will cause electricity prices to increase by as much as 23 percent. By 2020, 1.4 million jobs could be lost.

As I said at last week's hearing, we all have an interest in dealing with real pollution concerns and protecting public health. But we also know that President Obama has a cap and trade agenda that's specifically designed to raise energy prices by forcing coal and oil out of the market. He couldn't get it passed the Senate, so now he has the EPA doing it for him. This is something that no more than one-third of the U.S. Senate would vote for.

Today, the Clean Air Act is being implemented in a way that bears no resemblance to what Congress intended. Congress didn't give EPA the authority to set mandates that can't be achieved. Congress didn't give EPA the authority to pursue an agenda that hurts the very people it's supposedly trying to protect. And we all know that Congress didn't give EPA the authority to regulate greenhouse gasses. But here we are.

We hear a lot about the Clean Air Act these days. And I'll be the first to admit that industry and states have done a great job of cleaning up the air over the past 40 years. But the Clean Air Act is in dire need of modernization. It needs to be updated to undo years of bureaucratic overreach and messy court rulings; updated to meet the pollution challenges of today. And yes, updated to stop politicians from using it to pursue a reckless political agenda that hurts working families.

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TESTIMONY OF LISA P. JACKSON
ADMINISTRATOR
U.S. ENVIRONMENTAL PROTECTION AGENCY
BEFORE THE COMMITTEE ON ENVIRONMENT AND PUBLIC WORKS
UNITED STATES SENATE
June 15, 2011

"The Clean Air Act and Public Health"

Madam Chairman, thank you for inviting me to testify about EPA's ongoing efforts to protect the health of Americans by reducing air pollution. Pollutants such as mercury, arsenic, and particulate matter shorten or reduce the quality of Americans' lives and put at risk the health and development of future generations.

All Americans should be very proud of the significant progress we have made cleaning up our air. However, we still have more to do. For example, about 25 million people now battle asthma. One of those 25 million is my youngest son. I am reminded on a regular basis about the importance of cleaning up our air.

The Clean Air Act is one of the most important tools that the EPA has to protect public health. This landmark legislation was passed in 1970, and signed into law by a Republican President. It was substantially amended in 1990 under another Republican Administration. Simply put, protecting public health and the environment should not be – and historically has not been – a partisan issue.

In the 40 years since its enactment, the Clean Air Act has made steady progress in reducing the threats posed by pollution and allowing us all to breathe easier. Last year alone, the Clean Air Act is estimated to have saved 160,000 lives and prevented more than 100,000 hospital visits.[1]

Some may find it surprising that the Clean Air Act also has been one of our country's best economic investments. In contrast to doomsday predictions, history has shown, again and again, that we can clean up pollution at the same time the economy is growing and jobs are created. Over the 40 years since the Act was passed, the Gross Domestic Product of the United States grew more than 200 percent.[2]

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[1] USEPA (2011). *The Benefits and Costs of the Clean Air Act from 1990 to 2020*. Final Report. Prepared by the USEPA Office of Air and Radiation. February 2011. Table 5-5. This study is the third in a series of studies originally mandated by Congress in the Clean Air Act Amendments of 1990. It received extensive peer review and input from the Advisory Council on Clean Air Compliance Analysis, an independent panel of distinguished economists, scientists and public health experts.
[2] Bureau of Economic Analysis, National Economic Accounts, "Table 1.1.5. Gross Domestic Product," http://bea.gov/national/index.htm#gdp.

The Clean Air Act saves lives and strengthens the American workforce, and, as a result, the economic value of clean air far exceeds the costs. Expressed in dollar terms, the benefits of the Clean Air Act Amendments of 1990 alone are projected to reach approximately $2 trillion in 2020 with an estimated cost of $65 billion in that same year – a benefit to cost ratio of more than 30 to 1.[3]

It is also important not to overlook the jobs that come from building and installing pollution control equipment. For example, the U.S. boilermaker work force grew by approximately 35 percent, or 6,700 boilermakers, between 1999 and 2001 during the installation of controls to comply with EPA's regional nitrogen oxide reduction program.[4] In an Op-Ed in the Wall Street Journal, 8 major utilities that will be affected by our greenhouse gas pollution standards said, "Contrary to claims that EPA's agenda will have negative economic consequences, our companies' experience complying with air quality regulations demonstrates that regulations can yield important economic benefits, including job creation, while maintaining reliability."[5]

The Clean Air Act is a comprehensive statute that encompasses many different programs and parts. Each of these plays an important role in meeting the overall goal of improving public health by reducing air pollution. I will focus my remarks today on two current proposals required by the Act that are of critical importance to that goal.

Mercury and Air Toxics Standards Proposed Rule

On March 16, EPA proposed standards for mercury and other toxic air pollution from power plants. Although it has been many years since Congress enacted the requirement for standards to reduce power plants' toxic air emissions, when finalized, these standards would be the first-ever national standards for reducing toxic air pollutant emissions from power plants. While many power plants already meet these standards, the standards will require additional power plants to install widely available, proven pollution control technologies.

In 2016, deployment of these technologies will have the co-benefit of reducing particulate matter and ozone exposures which are estimated to prevent:

- **17,000 premature deaths**
- **11,000 heart attacks**

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[3] Dale W. Jorgenson Associates (2002a). *An Economic Analysis of the Benefits and Costs of the Clean Air Act 1970-1990. Revised Report of Results and Findings*. Prepared for EPA.

[4] International Brotherhood of Boilermakers, *Boilermaker Labor Analysis and Installation Timing*, March 2005, EPA Docket OAR-2003-0053 (docket of the Clean Air Interstate Rule).

[5] Peter Darbee, chairman, president and CEO,PG&E Corp.; Jack Fusco, president and CEO, Calpine Corp.; Lewis Hay, chairman and CEO, NextEra Energy, Inc.; Ralph Izzo, chairman, president and CEO, Public Service Enterprise Group, Inc.; Thomas King, president, National Grid USA,; John Rowe, chairman and CEO, Exelon Corp.; Mayo Shattuck, chairman, president and CEO, Constellation Energy Group; Larry Weis, general manager, Austin Energy , "We're OK With the EPA's New Air-Quality Regulations," Letter to the Editor, Wall Street Journal, December, 8, 2010.

- **120,000 cases of childhood asthma symptoms**
- **11,000 cases of acute bronchitis among children**
- **12,000 emergency room visits and hospital admissions**
- **850,000 days of work missed due to illness.**

The Mercury and Air Toxics Standards will dramatically reduce the amount of mercury emitted by power plants. Mercury is a toxin that, depending on the form and dose, may cause neurological damage to adults, children, and fetuses developing in the womb. Mercury, depending on the form and dose, may cause neurological damage, including lost IQ points, in children who are exposed before birth and is also associated with impacts on children's cognitive thinking, memory, attention, language, and fine motor and visual spatial skills.

In addition, these standards will also significantly reduce emissions of:

- metals such as arsenic, chromium, and nickel, which cause cancer and other health risks;
- acid gases that cause lung damage and contribute to asthma, bronchitis and other chronic respiratory disease, especially in children and the elderly;
- and fine particle pollution, which causes a host of health problems including premature mortality and lung and heart problems.

Charles D. Connor, President and CEO of the American Lung Association said of this rule: "When it becomes final, the cleanup rule that the EPA is putting forward today will save lives, protect the health of millions of Americans and finally bring about an action that is 20 years overdue. This must happen."

This proposed rule, which is going through a public comment process, is the product of significant outreach to industry and other stakeholders. The Clean Energy Group, a coalition of electric power companies, said: "Since 2000, the electric industry has been anticipating that EPA would regulate hazardous air pollutant emissions, and as a result, many companies have already taken steps to install control technologies that will allow them to comply with requirements of the rule on time. The technologies to control emissions at coal-fired power plants, including mercury and hydrochloric acid, are available and cost-effective."

Transport Rule

On July 6 of last year, the Agency proposed the "Clean Air Transport Rule," which would significantly improve air quality in cities throughout the eastern half of the U.S. by requiring 31 states and the District of Columbia to reduce their emissions of sulfur dioxide (SO2) and oxides of nitrogen (NOx) which contribute to ozone and fine particle pollution across state lines.

The proposed Transport Rule replaces EPA's 2005 Clean Air Interstate Rule (CAIR). A December 2008 court decision kept the requirements of CAIR in place temporarily, but directed EPA to issue a new rule to implement the Clean Air Act requirements concerning the transport of air pollution across state boundaries. This action responds to the court's concerns.

The final version of this rule is currently under OMB review; however, at the proposed rule stage, we estimated that the rule would result in more than $120 billion annually in health benefits by avoiding:

- **14,000 to 36,000 premature deaths,**
- **21,000 cases of acute bronchitis,**
- **23,000 nonfatal heart attacks,**
- **26,000 hospital and emergency room visits,**
- **1.9 million days when people miss work or school,**
- **240,000 cases of aggravated asthma, and**
- **440,000 cases of upper and lower respiratory symptoms.**

These numbers represent a major improvement in the quality of life of literally millions of real people throughout the country – especially working families, children, and older Americans. And that improvement translates into substantial benefits for our economy. These two rules demonstrate the common sense actions that have been and can be taken under the Clean Air Act in order to improve public health by reducing harmful pollution through the application of available technologies.

I look forward to your questions.

Written Statement of

Sarah Bucic, MSN, RN

On behalf of the

American Nurses Association
and
the Delaware Nurses Association

"The Clean Air Act and Public Health"

Before the
Committee on Environment and Public Works
U.S. Senate
Washington, DC

June 15, 2011

It is a privilege to appear before you today on behalf of the American Nurses Association and the Delaware Nurses Association to discuss the importance of the Clean Air Act, the positive impact it has had on the health of our nation, and the fundamental importance of continuing to support the Act to further protect public health.

The ANA is the only full-service professional organization representing the interests of the nation's 2.9 million registered nurses through its constituent member nurses associations--including the Delaware State Nurses Association. ANA advances the nursing profession by fostering high standards of nursing practice, promoting the rights of nurses in the workplace, projecting a positive and realistic view of nursing, and by lobbying the Congress and regulatory agencies on health care issues affecting nurses and the public.

Since the early years of the nursing profession, nursing leaders such as Florence Nightingale and Lillian Wald have recognized the role of nurses in controlling the influence of environmental factors on health. This underpinning of nursing practice was expressed by Florence Nightingale in her First Rule of Nursing: “Keep the air within as pure as the air without” (Nightingale 1859). ANA clearly recognizes the fundamental tie between the quality of our environment and the health of the nation, and I am honored to have the opportunity to appear before you today to discuss that connection.

Since its initial enactment and subsequent amendments, the Clean Air act has had a proven track record of success, cost-effectively cutting dangerous pollution, and positively impacting our environment and our health. According to a March 2011 report by the Environmental Protection Agency's Office of Air and Radiation, under the 1990 Clean Air Act Amendment Programs, the economic value of the substantial air quality improvements that would be realized by the year 2020 is estimated at almost $2 trillion, an amount which vastly exceeds the cost of compliance with the law.

This same report estimates that by 2020 the 1990 Clean Air Act Amendment Programs will have resulted in the prevention of 230,000 deaths, 2,400,000 incidences of asthma exacerbation, 120,000 emergency room visits, 3,200,000 lost school days and 13,000,000 lost work days. These statistics represent not only the Act's impact on health and quality of life, but its value in ensuring economic productivity.

Still, as the findings of the American Lung Association's 2011 *State of the Air r*eport show, despite these successes, we still have a long way to go to ensure that we all have clean air to breathe. The *State of the Air* report looked at levels of ozone and particulate matter — types of pollution with the most significant known health impacts--at monitoring sites across the country from 2007-2009, and it revealed some startling facts. While the study identified clear improvement in both categories over past years, serious problems remain. More than 154 million people, just over half the nation, endure pollution levels that make the simple act of breathing hazardous to their health.

The negative health effects of exposure to these pollutants, including ties to premature death, increased mortality, onset and exacerbation of asthma, increased susceptibility to pulmonary and respiratory infection, and more, are well established. In addition, there is a growing body of evidence that even exposure to lower levels of ozone and particulate matter poses a greater health risk than once thought.

For vulnerable populations—children, the elderly, people with asthma, those with diabetes, cardiovascular disease, or chronic bronchitis and emphysema-- the dangers posed by exposure is even greater. Approximately 3.2 million children and nearly 9.5 million adults with asthma live in parts of the United States with high levels of ozone, 1.2 million children and 3.8 million adults with asthma live in areas with high levels of short-term particulate matter pollution.

For these populations, a bad air day isn't just an inconvenience: a day when they are told not to mow their lawn, or have to wait until dark to fill their gas tank. A bad air day can be life or death. A bad air day can keep them from school, from work, from the grocery store, in short from living life.

Ozone is formed by chemical reactions in the atmosphere between gases that are emitted primarily when fossil fuels are burned. Alarmingly, one of the factors in that reaction is temperature, and as a result, warmer temperatures associated with climate change have the real potential to increase ozone pollution. A June 2011 report from the Union of Concerned

Scientists analyzed the health and economic impact of this climate-related ozone increase, with striking results. The report cites that by 2020, the US could pay an average of $5.4 billion in health impact costs associated with increases in ozone due to climate, and that the resulting higher concentrations of ozone could lead to hospitalization of an average of 3,700 more seniors and 1,400 more infants for respiratory-related problems by that same year.

The American Lung Association gives all three counties in my home state of Delaware an "F" grade placing close to 28,000 pediatric asthma cases at risk. An "F" is given if 9 days are over the ozone standard. There were approximately 3,000 asthma-related hospital admissions involving children through age 9 from 1994-2000 statewide. 46,000 adults are estimated to have asthma; and as many as 72,000 have had asthma at some time during their lives, and a new report on asthma in Delaware estimates that total direct charges for asthma-related health care could be between $25 and $30 million a year.

For nurses, these aren't just numbers, they are our patients. We see them in the emergency room on bad air days, struggling to breathe. They receive their albuterol treatments and stay to rest--sometimes for several days – this is an avoidable consequence of pollution that has a direct impact on individual lives and on our country's health care costs.

To be clear, asthma isn't always a condition where you get your medication and move on. In 2000 and 2001, the most recent years for which data are available, there were 17 deaths per year from asthma in Delaware, a state with less than 1 million people.

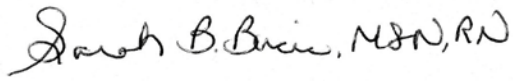
Ozone and particulate matter are by no means the only air pollutants that threaten our health. As a psychiatric nurse, I feel obligated to testify to the negative health effects of mercury. The harmful effects of mercury on children's developing brains is well known, as are its effects on memory, attention, language, fine motor and visual spatial skills. In addition, toxic metals such as arsenic, chromium, and nickel can cause cancer. According to the EPA, by 2016, cleaning up toxic emissions from power plants will save 17,000 lives each year, prevent 11,000 heart attacks each year, prevent 110,000 asthma attacks each year, eliminate 12,200 hospitalizations and emergency room visits each year and add 850,000 days when people don't miss work each year.

We encourage our patients to make responsible, healthy choices, but this personal responsibility alone only goes so far. What good is eating fruits and vegetables if they were grown in contaminated soil? What good is exercise if the air in their community is full of pollution? We must hold industry just as accountable as we hold our patients. We need to have "prescription/discharge instructions for industry" to be as clean as possible – investments in clean air benefit all of us and will pay dividends in lower health care costs.

The bottom line is pollution creates more patients. From a nursing perspective, our interventions remain limited if the environment remains polluted. We are fixed in a state of keeping patients with chronic conditions like asthma and other pulmonary and cardiovascular conditions stabilized, when we all know that prevention is the only real, effective and long-term treatment.

Our health is clearly and inextricably linked to the health of our environment, and we owe it to ourselves and our children to build on the success of the Clean Air Act by supporting the life-saving standards advanced under this landmark public health law. We cannot afford to roll back these vital protections, and we must ensure that the standards set for regulating ozone, particulate matter, mercury and other air toxics reflect the best science and truly protect the public.

Respectfully submitted,



Sarah Bucic, MSN, RN

American Academy of Pediatrics

DEDICATED TO THE HEALTH OF ALL CHILDREN™

Wednesday, June 15, 2011

Testimony of
Jerome A. Paulson, MD, FAAP

On behalf of the
American Academy of Pediatrics

Senate Environment and Public Works Committee hearing:
"The Clean Air Act and Public Health"

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Good morning. I appreciate this opportunity to testify today before the Committee on Environment and Public Works regarding the Clean Air Act and public health. My name is Jerome A. Paulson, MD, FAAP, and I am proud to represent the American Academy of Pediatrics (AAP), a non-profit professional organization of more than 60,000 primary care pediatricians, pediatric medical sub-specialists, and pediatric surgical specialists dedicated to the health, safety, and well-being of infants, children, adolescents, and young adults. I am the incoming chair of the AAP's Council on Environmental Health, and I direct the Mid-Atlantic Center for Children's Health & the Environment, one of ten Pediatric Environmental Health Specialty Units (PEHSU) in the United States, based at Children's National Medical Center here in Washington, D.C.

It has been more than 40 years since the Congress first passed the Clean Air Act, which, for the first time, gave the Environmental Protection Agency (EPA) the authority to enforce regulations to limit air pollution. Since the Clean Air Act was enacted, we have learned much about the relationship between air pollution and health through thousands of epidemiologic and controlled studies. The Clean Air Act has made incredible improvements in the environment, in the health of infants and children, and in the quality of life for all Americans. However, the impacts of the Clean Air Act have not been universally felt. Air quality in some areas of the United States has improved, but in some areas it has actually decreased, and millions of Americans still live in areas where monitored air fails to meet EPA standards for at least one of six criteria pollutants. In addition, in the last 40 years, we have learned that serious health effects of air pollutants are experienced at levels much lower than previously considered "safe" levels of exposure, particularly for vulnerable populations such as infants, children, the elderly, and individuals with respiratory diseases.

There is overwhelming evidence linking air pollution with a variety of adverse health outcomes. The AAP believes it is necessary for Congress to strengthen the Clean Air Act and the EPA's ability and authority to set, implement, and enforce Clean Air Act regulations throughout the country. Congress must not weaken or restrict these efforts. As a pediatrician who has cared for children suffering from the health impacts of air pollution, I am incredibly concerned about threats to clean air and the effect of air pollution on children's health.

Children are Disproportionately Impacted by Air Pollution

All aspects of the environment have especially profound effects on children's health. Children are disproportionately vulnerable to all environmental exposures; they breathe faster than adults, spend more time outside, and have proportionately greater skin surface exposed to the environment. A given dose of a pollutant will have a greater impact on a child than on an adult not only due to their smaller size, but because of the nature of their growing bodies and minds. At sensitive points in child development, environmental exposures can have especially harmful effects.

Infants and children are among the most susceptible to the adverse effects of ambient air pollution and are far more vulnerable compared to adults for a number of health and developmental reasons. First, children are more greatly impacted by air pollution due to their

extensive lung growth and development after birth. Eighty percent of alveoli (the part of the lungs where oxygen is absorbed and carbon dioxide is released from the blood) are formed postnatally, and the developing lung is highly susceptible to damage from environmental toxicant exposure during the early post-neonatal period.[1, 2, 3] Changes in the lungs continue through adolescence as respiratory cells actively proliferate and differentiate during this period of increased growth and development, creating increased susceptibility to the harmful effects of air pollution's chemicals and particulates.

Children also have increased exposure to many air pollutants compared with adults because of their higher minute ventilation (the amount of air breathed in or out of the lungs per minute), higher levels of physical activity, and because they spend more time outdoors. [4, 5, 6] Children in communities with higher levels of urban air pollution and children who spend more time outdoors are likely to have decreased lung function and growth. In addition to the increase in short-term respiratory symptoms, long-term exposure to air pollution may have lifelong consequences for children. In fact, air pollution is associated with impaired lung growth that may have permanent, lifelong impacts on an individual's ability to breathe. [7, 8] These impacts can have health consequences and impose increased health costs across the lifespan.

Ambient air pollution has been associated with several adverse birth outcomes. Air pollution has been linked to sudden infant death syndrome and mortality due to respiratory disease in normal birth weight infants,[9] with one study demonstrating that nearly one-quarter of deaths were attributable to elevated particulate matter.[10]

Because the lung is in direct contact with the air, children with underlying or chronic respiratory diseases are even more susceptible to the adverse effects of air pollution. In individuals with cystic fibrosis, elevated levels of particulate matter and ozone are associated with an increased risk of exacerbations and decline in lung function. For children with asthma, the most common chronic disease in childhood, ozone levels—even those below current EPA standards—are associated with increased respiratory symptoms and the need for rescue medication. School absences, emergency room visits, and hospital admissions are all directly associated with ambient air pollution. In a prospective cohort of children living in southern California, children with asthma living in communities with increased levels of air pollution (especially particulates, nitrogen dioxide, and acid vapor) were more likely to have bronchitis symptoms. The same mix of air pollutants was also associated with deficits in lung growth (as measured by lung function tests).

Impacts of Specific Air Pollutants on Children's Health

The scientific research on air pollution and its impacts on child health is comprehensive and has consistently proven over the past four decades that reducing exposure to toxicants and particulates in the air leads to healthier individuals. The following list of air pollutants have all been proven to have significant impacts on child health, and Congress and the Administration should take every effort to reduce their emissions and prevalence in the environment. It is also important to note that air pollutants never occur alone or in isolation from one another. Air pollutants occur in mixtures with different concentrations in different geographic areas

throughout the United States. Air pollutants interact with each other in the environment in different and sometimes exacerbating ways and it is less clear how pollutants interact once they enter the human body. In order to promote child health, it is necessary to address air pollutants as a whole and not take a piecemeal approach in addressing these environmental and health hazards.

Mercury: Coal fired power plants are the largest human-caused source of mercury emissions in the United States. Power plants that burn fossil fuels release mercury into the air, which then deposits in water, where living organisms convert it to methylmercury. Mercury emissions from power plants are of particular concern because mercury settles in our waterways and then accumulates in fish that are consumed by humans.

Methylmercury consumed through seafood is toxic to the developing brain of the fetus and young child. The damage it causes is permanent and irreversible. In studies of areas with high exposures to mercury outside of the United States, mothers gave birth to infants who initially appeared normal, but who went on to develop problems such as blindness, deafness, and seizures. In utero exposure to lower levels of mercury has been associated with more subtle effects on memory, attention, and language. The developing fetus and young children are disproportionately affected by methylmercury exposure, because many aspects of development, particularly brain maturation, can be disturbed by the presence of methylmercury. Minimizing mercury exposure is essential to optimal child health.

Ozone: Ozone is a powerful oxidant and respiratory tract irritant in adults and children, causing shortness of breath, chest pain when inhaling deeply, wheezing, and cough.[11] Children have decreases in lung function, increased respiratory tract symptoms, and asthma exacerbations on days with higher levels of ambient ozone. [12, 13, 14, 15] Increases in ambient ozone have been associated with respiratory or asthma hospitalizations, [16, 17]emergency department visits for asthma, and school absences for respiratory tract illness.[18] In Atlanta, Georgia, summertime children's emergency department visits for asthma increased 37% after six days when ozone levels exceeded 0.11 ppm.[19] In southern California, school absences for respiratory tract illness increased 63% in association with a 0.02-ppm increase in ozone.[20]

Ozone may be toxic at concentrations lower than 0.075 ppm, the current federal regulatory standard. Field studies suggest potential thresholds of as low as 0.04 ppm (one-hour average) for effects on lung function.[21, 22, 23] Studies of hospitalizations for respiratory tract illness in young children and emergency department visits for asthma suggest that the effects of ozone may occur at ambient concentrations below 0.09 ppm.[24, 25] In addition to studies on short-term effects, two studies of college freshmen suggest that increasing cumulative childhood exposure to ozone may affect lung function when exposed children reach young adulthood, particularly in measures of flow in small airways.[26, 27] Early childhood exposures may, therefore, be particularly important.

Particulate Matter: In children, particulate pollution affects lung function[28, 29, 30] and lung growth.[31] Recent studies in different countries have also found associations between ambient air pollution (especially particulates and/or carbon monoxide) and preterm birth,[32,33, 34, 35] low birth

weight, 50–53 and post-neonatal infant mortality (attributable to respiratory causes and possibly sudden infant death syndrome).[36,37]

Particle pollution contributes to excess mortality and hospitalizations for cardiac and respiratory tract disease. The mechanism for particulate matter–associated cardiac effects in adults may be related to disturbances in the cardiac autonomic nervous system, cardiac arrhythmias, or increased blood concentrations of markers of cardiovascular risk.[38,39] Daily changes in mortality rates and numbers of people hospitalized are linked to changes in particulate air pollution.[40,41,42,43] These studies and others have estimated that for every 10 microg/m^3 increase in PM10, there is an increase in the daily mortality rate between 0.5% and 1.6%.

Nitrogen Dioxide: Controlled-exposure studies of people with asthma have found that short-term exposures (30 minutes) to nitrogen dioxide at concentrations as low as 0.26 ppm can enhance the allergic response after subsequent challenge with allergens.[44, 45] These findings are of concern, because some urban communities that are in compliance with the federal standards for nitrogen dioxide (annual average) may experience substantial short-term peak concentrations (one-hour average) that exceed 0.25 ppm. Epidemiologic studies have reported relationships between increased ambient nitrogen dioxide and risks of respiratory tract symptoms[46, 47]and asthma exacerbations.[48]

Traffic-Related Pollution: Motor vehicles represent the principal source of air pollution in many communities, and concentrations of traffic pollutants are greater near major roads. Increased respiratory tract complications in children (e.g., wheezing, chronic productive cough, and asthma hospitalizations) have been associated with residence near areas of high traffic density, particularly truck traffic.[49, 50, 51, 52] Other investigators have linked various childhood cancers to proximity to traffic.[53, 54, 55]

Diesel exhaust, a known carcinogen and respiratory tract irritant as well as a source of fine particulate matter, is a particular concern for children. On the basis of extensive toxicologic and epidemiologic evidence, national and international health authorities, including the EPA and the International Agency for Research on Cancer, have concluded that there is considerable evidence of an association between exposure to diesel exhaust and an increased risk of lung cancer.[56, 57] Additionally, fine particles in diesel exhaust may enhance allergic and inflammatory responses to antigen challenge and may facilitate development of new allergies or worsen symptoms in individuals with allergic rhinitis or asthma.[58, 59]

School buses operate in close proximity to children, and most of the nation's school bus fleets run on diesel fuel. The EPA and some state agencies are establishing programs to eliminate unnecessary school bus idling and to promote use of cleaner buses to decrease children's exposures to diesel exhaust and the amount of air pollution created by diesel school buses (www.epa.gov/cleanschoolbus). One recent study found that a child riding inside a school bus may be exposed to as much as four times the level of diesel exhaust as someone riding in a car.[60] These findings underscore the need for increased regulation of diesel emissions, especially in areas where children congregate, such as school buses. The EPA should be encouraged to

continue to work with school districts to replace or retrofit diesel buses with pollution-reducing devices and limit school bus idling where children congregate.

Indoor Air Pollutants: Secondhand smoke is among the most harmful and common indoor dangers to children. According to the 2006 Report of the Surgeon General almost 60 percent of children aged 3-11 years are exposed to secondhand smoke. These children are at increased risk for multiple serious health effects like asthma, respiratory infections, decreased lung growth and exercise tolerance, and sudden infant death syndrome. This exposure is most dangerous for the youngest children because their lungs are not fully developed and they often spend time in close proximity to their parents who smoke. Other effects of secondhand smoking may include childhood cancer, childhood leukemia, childhood lymphomas, and childhood brain tumors. Smoking and exposure to secondhand smoke among pregnant women contributes to low birth-weight babies, preterm delivery, perinatal deaths, and sudden infant death syndrome. Well over 30,000 births per year in the U.S. are affected by one or more of these problems.

Other Air Pollutants: Airborne levels of lead, sulfur dioxide, and carbon monoxide have decreased dramatically over the past 40 years because of the implementation of the Clean Air Act. However, levels of these pollutants may still be high near major sources. For example, high lead levels may be found near metals-processing industries, high sulfur dioxide levels may occur near large industrial facilities (especially coal-fired power plants), and high levels of carbon monoxide may occur in areas with heavy traffic congestion.[61]

In addition to criteria air pollutants, there are numerous other air pollutants produced by motor vehicles, industrial facilities, residential wood combustion, agricultural burning, and other sources that are hazardous to children. More than 80,000 chemicals are used commercially, and many are released into the air. For most of these chemicals, data on toxicity are sparse.[62] Some pollutants remain airborne or react in the atmosphere to produce other harmful substances. Other air pollutants deposit into and contaminate land and water.

The Clean Air Act and Health Care Costs

As a pediatrician, I know that preventive health care is a fundamental investment in the health of all children and preventive health care at a young age can have lifelong impacts. Healthy children are far more likely to grow up into healthy adults. Conversely, children who experience poor health are more likely to suffer from ill health in adulthood. Inadequate attention to preventive health care mortgages the future health and welfare not only of children, but of society itself. Research across a broad range of interventions has shown that preventive health and wellness for children consistently produces a high return on investment. Ensuring that children breathe air that is free of chemicals and pollutants is an extremely effective and economical intervention for promoting lifelong health and reducing long term health costs.

According to the EPA's recent report "The Benefits and Costs of the Clean Air Act from 1990 to 2020," in 2010, the Clean Air Act prevented 160,000 cases of premature adult mortality, 230 cases of infant mortality, 130,000 heart attacks, 3.2 million lost school days, 86,000 emergency

department visits, and 1.7 million asthma attacks. These health quality measures and lives saved are expected to continue to improve significantly over the next decade.

According to the EPA's report, complying with the Clean Air Act will cost about $65 billion per year, but the benefits are projected at $2 trillion per year, most of which is saved through reduced morbidity and mortality. As a pediatrician, the Clean Air Act's tremendous cost savings represent not just economics, they represent children: fewer children suffering from asthma attacks, fewer hospitalizations, less respiratory tract illnesses, improved lung capacity and function for growing children, and healthier infants and newborns. Treating chronic conditions that are created or exacerbated by air pollution is currently expensive to our public and private sectors, and health care costs will continue to increase each year. At a time when lawmakers are intensely focused on reducing health care costs, expanding efforts to regulate and limit air pollutants could prove to be a successful and effective tool in accomplishing this goal.

AAP Recommendations

The AAP recommends in the strongest terms possible that the Clean Air Act should not be weakened in any way that decreases the protection of children's health. Weakening standards now will almost certainly result in increased emergency room visits and hospital admissions for children with respiratory issues, resulting in increased direct costs for medical care, and increased indirect costs from lost productivity due to missed school and work. Weakening standards now will almost certainly result in adults with increased chronic lung disease as they age.

Air quality standards should be drafted or revised to ensure that the most vulnerable groups are protected. Potential effects of air pollution on the fetus, infant, and child should be evaluated and all standards should include a margin of safety for protection of children. Congress and the Administration must keep these principles in mind when considering any changes or modifications to the Clean Air Act. If we fail to protect children against air pollution, we accept the cost of living with and treating preventable birth defects, chronic diseases, and disability among our nation's infants and children. If we fail to protect children against air pollution, we also accept the cost of permanently reduced lung capacity and productivity in adults.

In addition, the American Academy of Pediatrics submits the following recommendations to the Committee, which we believe will lead to cleaner air and better health for all American infants, children, and families:

Children's exposure to diesel exhaust particles should be decreased. Idling of diesel vehicles in places where children live and congregate should be minimized. Ongoing programs to fund conversion of diesel school bus fleets to cleaner alternative fuels and technologies should be pursued and supported.

Federal and state governments' policies should encourage reductions in mobile and stationary sources of air pollution, including increased support for mass transit, carpooling, retiring or

retrofitting old power plants that do not meet current pollution-control standards, and programs that support marked improvements in fuel emissions of gasoline- and diesel-powered vehicles. Additionally, the development of alternative fuel fleets, low-sulfur diesel, and other "low-emission" strategies should be promoted. Before promoting new alternative fuels, these alternative fuel sources should be critically evaluated and determined by governmental authorities to have a good safety profile.

EPA should increase funding for Pediatric Environmental Health Specialty Units. Pediatric Environmental Health Specialty Units serve a vital function in providing each of the ten EPA regions with direct access to pediatric environmental health experts. The PEHSUs could be directed to use a portion of this funding to increase the education of health and education professionals and others about air pollution and the impact of those pollutants on the health of children.

In conclusion, the American Academy of Pediatrics commends you, Madame Chairwoman, for holding this hearing today to call attention to the public health impacts of the Clean Air Act. We look forward to working with you to continue to improve air quality and children's health throughout the country. I appreciate this opportunity to testify, and I will be pleased to answer any questions you may have.

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Major Factors in the Prediction of National Life Expectancy: GDP and Unemployment

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Major Factors in the Prediction of National Life Expectancy: GDP and Unemployment

1. Income

At the individual level of analysis – i.e., in epidemiological studies – individual income is a standard and fundamental inverse predictor of illness and early mortality (Andersen, Gamborg, Osler, Prescott, Diderichsen' 2005; Ecob, Davey Smith, 1999; Ettner, 1996; Kahn, Wise, Kennedy, Kawachi, 2000; Kivimäki, Shipley, Ferrie et al., 2008; Lynch, Smith, Kaplan, House, 2000). In industrialized countries, the higher the level of income of individuals, the lower the illness and mortality rates attributed to the great majority of infections, chronic diseases and mental disturbances. In developing countries similar relations are found, but the impact of income is substantially stronger. Moving from the individual to the national level, however, real GDP per capita indicates the availability of basic goods and services: nutrition, potable water, sanitary engineering, housing and other means of climate control, transportation and primary health care. At the national level, real GDP per capita – especially for industrialized societies also conveys the capacity of the society to invest in the development of science and technology, improved working conditions at higher technological levels of safety and health, financing of education at all levels, stabilization of the income of individuals and small businesses, and the many types of social protection: unemployment insurance; active labor market policies; health insurance; disability insurance; social welfare payments to impoverished, frail populations and children; social security and retirement benefits.

Income, of course, inasmuch as it conveys the purchase of goods and services of different price and quality, also conveys the special 'social status' of the purchaser. Thus, certain aspects of purchases take on the meaning of 'status symbols' by which different levels of social prestige are attributed to the purchasers. Prestige, in turn, is a principal marker of the socioeconomic status of individuals and is understood to convey differential levels of psychological wellbeing (Marmot, 2002; Marmot, Wilkinson, 2001).

2. *Unemployment*

The unemployment rate is well established as a risk factor for elevated illness and mortality rates in epidemiological studies performed since the early 1980's. In addition to influences on mental disorder, suicide and alcohol abuse and alcoholism, unemployment is also an important risk factor in cardiovascular disease and overall decreases in life expectancy (Linn, Sandifer, Stein, 1985; Morris, Cook, Shaper, 1994; Jin, Shah, Svopoda, 1995; Martikainen, Ma, Ja, 2007; Virtanen, Vahtera, Kivimäki, et al., 2005; Bambra, Elkemo, 2008; Catalano, 1991; Catalano, Dooley, Novaco, Wilson, Hough, 1993; Dooley, Catalano, Wilson, 1994; Kasl, Jones, 2002; Tausky, Piedmont, 1967).

The first group of studies with a prospective epidemiological and demographic orientation were at the national level and used time-series analysis. These include the studies in the United States for the Joint Economic Committee of the U.S. Congress in 1976 and 1984 (Brenner, 1976; 1984). These studies demonstrated a temporal relationship between national levels of unemployment, on the one hand, and on the other age-adjusted mortality rates, cardiovascular

mortality rates and suicide over a six-year period following economic recessions in which unemployment was elevated. These long-term effects of unemployment at the national level were further supported by a study of England and Wales with elevated unemployment rates being related to increased age-adjusted mortality rates over the subsequent ten years (Brenner, 1979). These studies controlled for the impact of national income and wealth (GDP per capita), as well as inflation and the short-term impact of rapid economic growth. Subsequent studies of historical change in unemployment and mortality rates (especially cardiovascular), in other European and Western countries supported the earlier U.S. and British studies. These countries included: France, Germany, Italy, Belgium, Netherlands, Finland, Sweden, Japan, Australia and New Zealand (Brenner, 1997; 1987a; 1987b; 1982). For the entire 20th Century this finding was again confirmed, with respect to historical time in the United States in 2005 (Brenner, 2005). Most recently, the country-level relationship between unemployment and decreased life expectation has been confirmed for 31 industrialized countries for the year 2008 (Brenner, 2010) (see also table and graph below). In the European and industrialized country studies, adjustment was made for GDP per capita as well as alcohol, tobacco, and dietary risk factors. A study based on United States data not confirming the positive relation between unemployment and mortality rates, did not adjust for other economic factors (especially income inequality) and lifestyle factors was published in 2000 (Ruhm, 2000).

Rigorous epidemiological studies, at the individual level of analysis were conducted in England and Wales (Morris et al 1994, Moser et al 1987, Bethune 1997, Mitchell et al 2000) and in several Scandinavian countries, among others (Martikainen & Valkonen 1996, Iversen et al 1987, Pensola & Martikainen 2004, Voss et al 2004, Ahs & Westerling 2006, Gallo et al 2004),

documenting elevated risks of fatal or non-fatal cardiovascular or cerebrovascular events or of all-cause mortality among *unemployed* as compared to permanently employed people. Risks are particularly high among the long-term unemployed. Effect sizes of hazard ratios are usually quite substantial, i.e. in the range of 1.5 to 2.5.

Many less rigorously conducted studies support these results, demonstrating elevated risks of a range of other health outcomes. For instance, unemployment increases rates of depression, particularly in the young. Parasuicide rates in young men who are unemployed are substantially higher than for those in employment (Dorling 2009). In a longitudinal study in England, excess mortality from suicide was obvious among unemployed men (Moser et al 1984). In the British Household Panel Study unemployment among people in the most disadvantaged social group was related to elevated risk of incident limiting illness (Bartley et al 2004). Other studies point to impaired mental health, specifically depression, as a consequence of unemployment (Kasl & Jones 2000, Kaplan et al 1987), whereas becoming depressive in turn increases the probability of future unemployment and loss of income (Whooley et al 2002). Becoming reemployed is generally associated with a reduction in symptomatology (Kessler et al 1989, Kasl & Jones 2000), but not in the rate of mortality (Bethune 1997).

Several mechanisms are understood to operate in the relationship between unemployment and damage to health. One of these concerns the psychological stresses of loss and mental depression that separates the individual from his/her place in economic organizations – a position in which the content of work and social relations give meaning and direction to life. A second mechanism involves the loss of social relationships at work, which often constitute the major

social networks and friendship patterns for many individuals as well as a definition of their position in society. Thirdly, and over the longer term, there is the problem of loss of income – which could be transitory or relatively permanent. In the case of transitory income loss, the period of unemployment may be relatively short, and new employment will restore previous wage and salary levels. More common, as with lengthy recessions and structural unemployment, is the inability of the unemployed individual to find reemployment in the same industry and/or at the same level of seniority (Podgursky, Swaim, 1987a, 1987b).

In that case the unemployed person may find subsequent work in a different firm, or different industry altogether, in which the starting position will be at a very junior level. It may then take many years – if ever – f or the previously unemployed person to resume the level of income that prevailed prior to the unemployment. A far more serious situation occurs when the unemployed person becomes discouraged from seeking new work which would make use of the individual's skills and length of experience (Moore, Ranjan, 2005; Wilson, 1990; Blank, Blinde, 1985). In that case, the individual may leave the labor force entirely (or retire), and the likelihood of considerable loss of permanent income is relatively high (Uchitelle, Kleinfield, 1996). Finally, as we have seen in the case of loss of income – indicating permanent loss of socioeconomic status – there is then a significantly increased risk of illness and early mortality.

The Model: Influences of GDP and Unemployment on Life Expectancy

The most extensive and robust findings in epidemiology of mortality patterns in industrialized countries is that "socioeconomic status" is the single most dominant, pervasive (or powerful)

factor that routinely influences comparative length of life among industrialized country populations.

Thus it can be seen that the national income and wealth of a society, i.e., real GDP per capita, is the foundation of much of individual material welfare that is described in the concept of socioeconomic status. Beyond those sources of investment in socioeconomic status are societal investments in a variety of public health and safety measures to insure social protection. These include health care sciences and technology, health and safety regulations in the workplace, environmental regulations, investment in the epidemiological and clinical sciences that identify risks to population health in terms of the monitoring of food and drug safety, safety of water and sanitation systems, new knowledge of optimal diets and the effects of alcohol and tobacco on health, and transportation safety.

In addition, of course, growth in GDP per capita is almost by definition the antithesis of recession and, therefore, unemployment. It is indeed specified under the "law" proposed by the economist Okun that a specified quantitative increase in potential GDP will reduce the unemployment rate by a specified amount.

Finally, real GDP per capita is also the source of investment in new innovation and new employment, largely through the initiation of new small firms, frequently via self-employment. The strength of self employment, then, in a well developed industrialized society, represents much of the source of innovation that will maintain or advance the rate of economic growth. Equally important, however, self-employment serves as a source of opportunity to avoid

unemployment – whether that unemployment originates with economic cycles, technical and productivity development, globalization or other structural changes in the economy. Self-employment, thus, can provide a "safety catch" by which unemployment is reduced and through the development of small firms, can be an avenue through which active labor market policies can result in successful reemployment.

To exemplify this true set of factors which affect health and social wellbeing both directly and indirectly stemming from GDP growth, a model is presented which encompasses a number of the factors which are related to GDP-based investment and consumption patterns. This is a model which attempts to account for variation among industrialized countries in the age-standardized mortality rate.

The model includes a measure of the rate of productivity, namely GDP per employee. It includes as well, the rate of self-employment and both the unemployment rate for males in relation to population as well as the source of inflation, namely the consumer price index. This model also includes publicly-funded health expenditures which are derived from GDP investments. Finally it controls for a major risk factor that is well-known in the epidemiological literature for its ability to exert a short-term effect of a multiplicity of sources of illness and disability, including cardiovascular diseases, diabetes, work and home accidents and injuries, transportation accidents, suicide and homicide. This factor is alcohol consumption per capita.

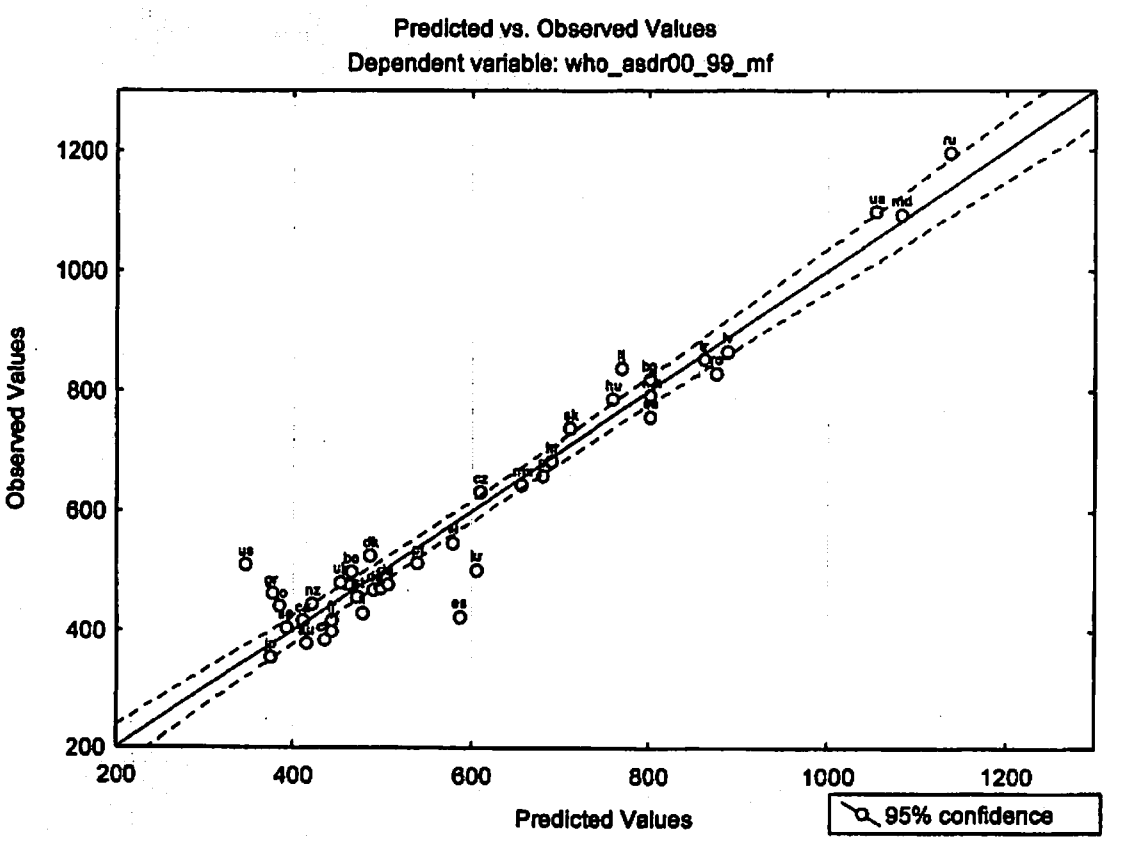
Figure 1

Relation of economic variables to age adjusted total mortality 40 European, CIS and OECD Countries, Year 2005

R= .97027845 R^2= .94144027 Adjusted R^2= .92863033

F(7,32)=73.493 p<.00000 Std.Error of estimate: 57.996

N=40	Beta	Std.Err.	B	Std.Err.	t(32)	p-level
Variables			422.1625	113.2121	3.72895	0.000745
GDP per employee (productivity)	-0.275164	0.084246	-0.0038	0.0012	-3.26619	0.002602
Shadow economy as proportion of GDP	0.442261	0.090652	7.8362	1.6062	4.87866	0.000028
Public health expenditure as proportion of GDP	-0.218179	0.062863	-29.1464	8.3978	-3.47072	0.001507
Self employed as proportion of total employment	-0.223634	0.045491	-4.5745	0.9305	-4.91595	0.000025
Unemployment rate, males	0.117708	0.050753	6.9823	3.0106	2.31922	0.026924
Alcohol consumption per capita	0.206031	0.046603	15.4188	3.4876	4.42102	0.000106
Consumer Price Index (Index Year 2000)	0.258445	0.050777	1.3576	0.2667	5.08979	0.000015



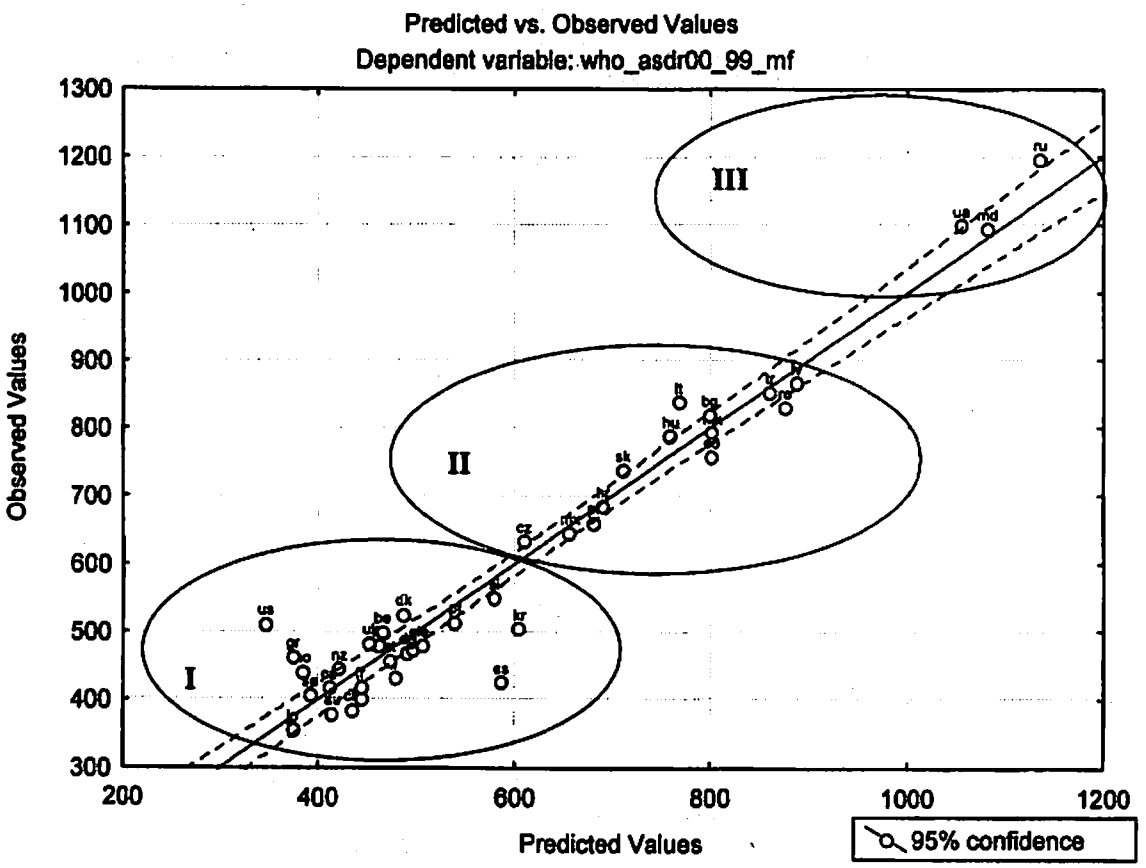
Importance of National Income to National Mortality Rates

The model above uses seven factors to account for the levels of age-standardized mortality rates among 40 industrialized countries. However a fundamental distinction among the 40 countries can be seen by distinguishing these countries by region and by average real GDP per employee. Figure 2 below repeats Figure 1 but groups the countries in terms of major regional classification and average GDP per employee. The first region enclosed in circle I, includes Western Europe, North America (except Mexico) and the highly industrialized Pacific countries of Australia, New Zealand, Japan and Korea with average GDP per employee of $ 45, 766. The second group of countries, in circle II, includes Eastern Europe and Mexico, with average GDP per employee of $20,292. The third group of countries, are within the Community of Independent States (CIS) and include Moldova, the Russia Federation and Ukraine, with average GDP per employee of $8,022.

Thus the basic differentiation among the group of countries is by geography and national income/wealth as well as productivity as measured by real GDP per employee. Within each of the three circles, one can see a relatively straight line representing the extent to which the entire set of seven predictors accounts for variation in age-standardized mortality rates among countries within the three regional groups. While the alignment of the three circles in a upward slope is essentially accounted for by GDP per employee, the variation of mortality among countries within the circles is largely accounted for by the remaining six variables in the model (shadow economy, self-employment, inflation rate, pubic health expenditures, male unemployment rate, and alcohol consumption per capita).

Figure 2

Relation of economic factors to age-standardized mortality rates: countries grouped according to average GDP per employee and geographic region.

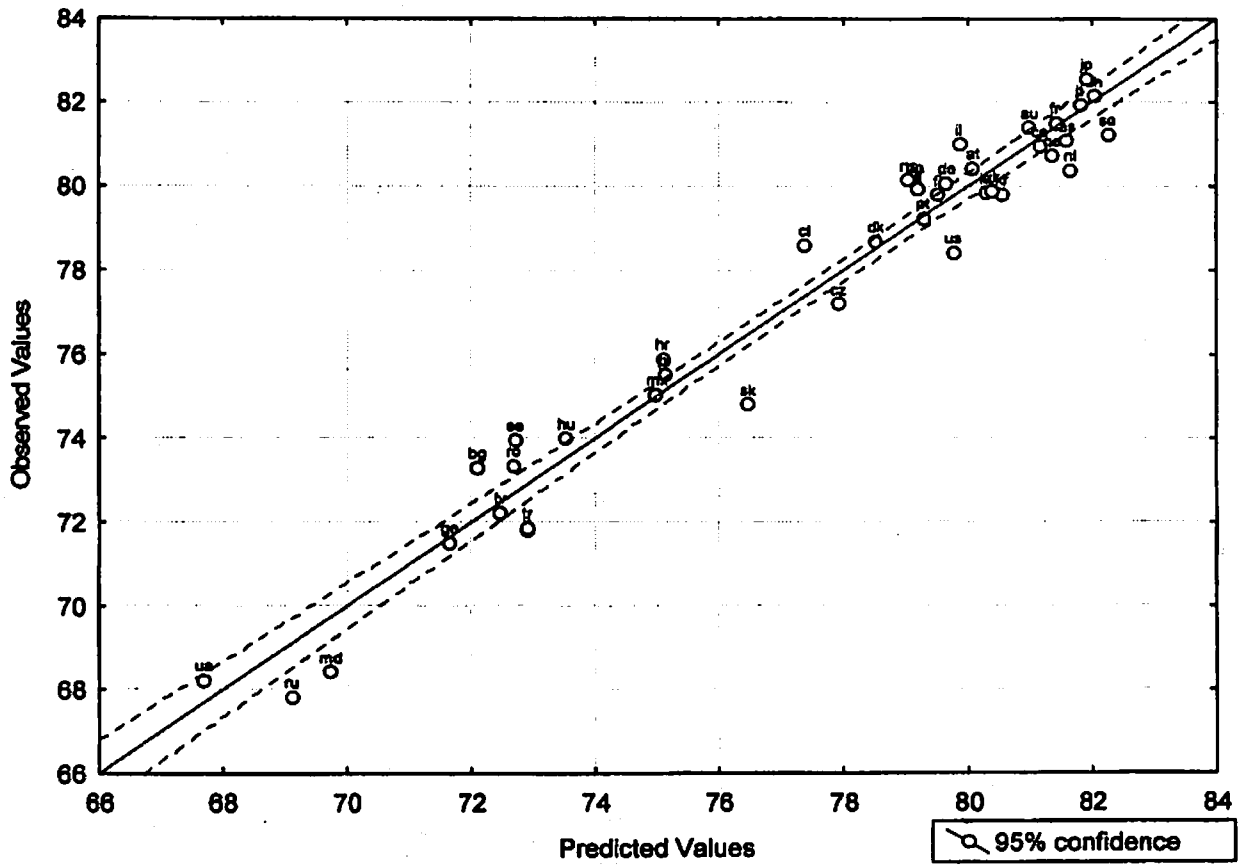


	Region	Real GDP per Employee (Average USD)
I.	Western Europe North America Pacific	45,766
II.	Eastern Europe Mexico	20,292
III.	CIS Moldova Russia Federation Ukraine	8,022

Figure 3
Regression Summary for total life expectancy at birth (in years)
for 40 European (West, Central and East) and OECD countries, years 2008

R= .97896360 R^2= .95836974 Adjusted R^2= .93986740
F(12,27)=51.797 p<.00000 Std.Error of estimate: 1.0379

	Beta	Std.Err.	B	Std.Err.	t(27)	p-level
Intercept			22.78837	5.798625	3.92996	0.000533
Logarithm of GDP per capita in constant 1990 international $	0.836529	0.068515	6.37221	0.521905	12.20951	0.000000
Social security expenditure as percentage of general government expenditure on health	0.158444	0.047908	0.01823	0.005513	3.30728	0.002671
Male self-employment as percentage of total male employment (15+ years old)	0.268693	0.055326	0.13455	0.027705	4.85654	0.000045
Male unemployment rate as percentage of male labor force	-0.134057	0.047406	-0.27670	0.097849	-2.82782	0.008724
Total food consumption in kilocalories per capita and day	-0.251139	0.070873	-0.00371	0.001046	-3.54349	0.001460
Protein consumption in grams per capita and day	0.234765	0.075766	0.07445	0.024027	3.09857	0.004506
Vegetable fat consumption in grams per capita and day	0.271082	0.061580	0.06312	0.014339	4.40213	0.000152
5 years average of total tobacco consumption in grams per adult and day (5 years lag)	-0.154843	0.055524	-0.06636	0.023796	-2.78876	0.009578
Current smoker prevalence in male adult population	-0.187336	0.056437	-0.06453	0.019441	-3.31938	0.002590
Crude birth rate (births per1,000 of total population)	-0.141387	0.045526	-0.21448	0.069061	-3.10566	0.004428
Carbon dioxide emission in metric tons per capita & year	-0.159418	0.052442	-0.16602	0.054614	-3.03989	0.005209
Dummy (Baltic countries=1, all others=0)	-0.218855	0.046422	-3.47263	0.736588	-4.71448	0.000066



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Commentary: Economic growth is the basis of mortality rate decline in the 20th century—experience of the United States 1901–2000

M Harvey Brenner[1,2]

Background The hypothesis that economic growth has been the principal source of mortality decline during the 20th century in the United States is investigated. This hypothesis is consistent with the large epidemiological literature showing socioeconomic status to be inversely related to health status and unemployment associated with elevated morbidity and mortality rates. Despite evidence over many years showing economic growth, over at least a decade, to be fundamental to mortality rate declines and unemployment rates showing lagged, cumulative effects on mortality rate increases, a recent paper argues that the impact of economic growth is to increase the mortality rate.

Methods This study utilizes age-adjusted mortality rates over 1901–2000 in the United States as the outcome measure, while independent variables include real GDP per capita in purchasing power parity, the unemployment rate, and the employment to population ratio. A basic interaction model is constructed whereby (i) real GDP per capita, (ii) the unemployment rate, and (iii) the multiplicative interaction between real GDP per capita and the unemployment rate are analysed in relation to age-adjusted mortality rates. The Shiller procedure is used to estimate the distributed lag relations over at least a decade for variables (i), (ii), and (iii). The error correction method is used to examine these relations for both levels and annual changes in independent and dependent variables.

Results While GDP per capita, over the medium- to long-term, is strongly inversely related to mortality rates during 1901–2000, in the very short term—i.e. within the first few months—rapid economic growth is occasionally associated with increased mortality rates estimated in annual changes. With respect to the unemployment rate, the first year (without lag) will frequently be associated with a decrease in mortality, but thereafter, and at least for the following decade, the effect is to increase the mortality rate. Thus, the net effect of increased unemployment is a substantial increase in mortality. This is also reflected in the entirely negative relation between the cumulative effects of the employment to population ratio and mortality rates over a decade.

Conclusions Economic growth, cumulatively over at least a decade, has been the central factor in mortality rate decline in the US over the 20th century. The volatility of rapid economic growth as it departs from its major trend, has a very short-term effect (within a year) to increase mortality—partly owing to adaptation to new technology and the adjustment of the formerly unemployed to new jobs, social status, and organizational structures.

Keywords Economic growth, per capita income, unemployment, business cycles, economic inequality, mortality

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Socioeconomic status, unemployment, and health

It is now among the firmest of epidemiological findings, across industrialized societies, that socioeconomic status is inversely related to health status. In particular, higher income has been routinely shown to be a significant inverse predictor of morbidity and mortality.[1-10] Similarly, the large and growing literature on unemployment and health is highly consistent in demonstrating elevated morbidity and mortality associated with unemployment and withdrawal from the labour force.[11-18] It follows that economic growth, the major source of socioeconomic status improvement, should lead to lower morbidity and mortality rates, whereas economic decline—especially in conjunction with high unemployment—should increase mortality rates.

The macro level

At the macro, i.e. national, level Thomas McKeown has demonstrated the fundamental importance of economic development to the decline—and near disappearance—of the classic infectious and childhood diseases as significant causes of death from the second half of the 19th century to the Second World War in England and Wales.[19,20] Over 1860–1950, the importance of economic growth to mortality decline is substantially owing to improvements in nutrition, sanitary engineering, and housing construction. Subsequently, it has been observed since at least the 1970s that the long-term effect of economic growth is a central source of mortality reduction, even after the Second World War in the US, the UK and the other 14 original EU countries, Canada, and Japan.[21-25] It has also been repeatedly shown at the national level that unemployment is a significant predictor of higher mortality rates over a period of at least a decade in many industrialized countries.[21-29]

How then are we to understand findings reported by Tapia Granados[30] that 'economic expansions' are related to higher mortality? Many epidemiologists may find this report incomprehensible on the grounds that the findings are incoherent—i.e. entirely inconsistent with the large epidemiological literature on low socioeconomic status and poor health, on the one hand, and on the relationship of unemployment and diminished health, on the other. The estimates by Tapia Granados are presented in simple correlations, without regard to lag estimation or the usual multivariable controls for confounding and interaction, or the standard econometric tests including those for residual autocorrelation and unit roots, and even without capitation of GDP itself.

Beyond these inconsistencies and methodological omissions, per capita economic growth over the 20th century cannot be positively related to increased mortality rates (age-adjusted), since these two trend-like variables are strongly inversely related to each other as is easily observed (Figure 1). In comparison, economic growth rates on an annual basis, without any lag, show a very weak, but positive relation to age-adjusted mortality rates (Figure 2). Furthermore, economic expansions cannot really be related to mortality increases over the 20th century because, during both expansion and recession years, age-adjusted mortality rates have, on average, fallen. During the 20th century, 72 years, or nearly three-quarters of the century, have been represented by economic growth ('expansions'). If economic growth literally caused mortality to rise, then age-adjusted mortality rates would have shown an increased trend over the 20th century rather than a rapid and massive decline.

Long-term impact of economic growth on mortality decline

What is the basis for the statement that economic growth is the 'central' precursor of mortality decline in the 20th century?

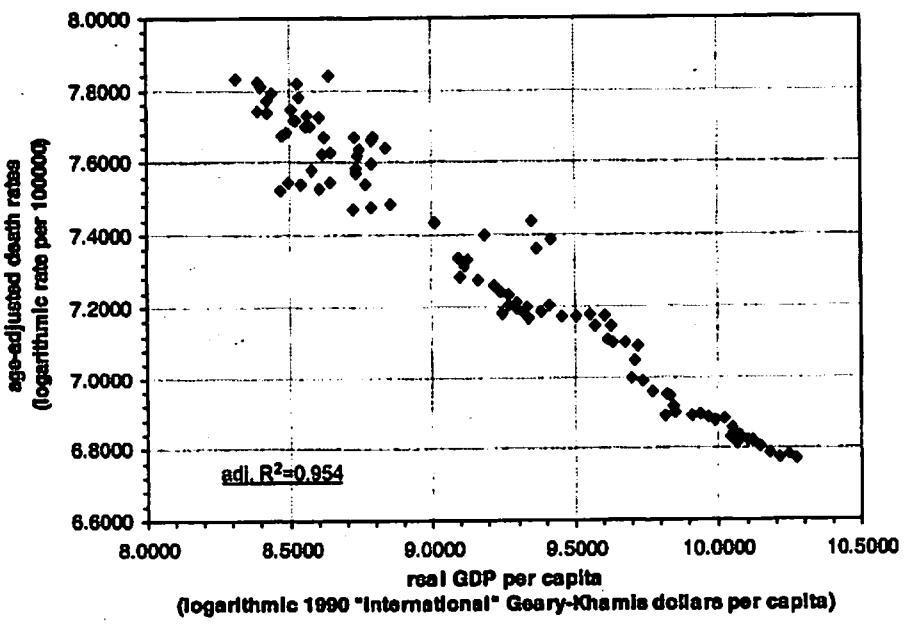

Figure 1 Relation of real GDP per capita to age-adjusted death rates, US 1900–2000 (natural logarithms)

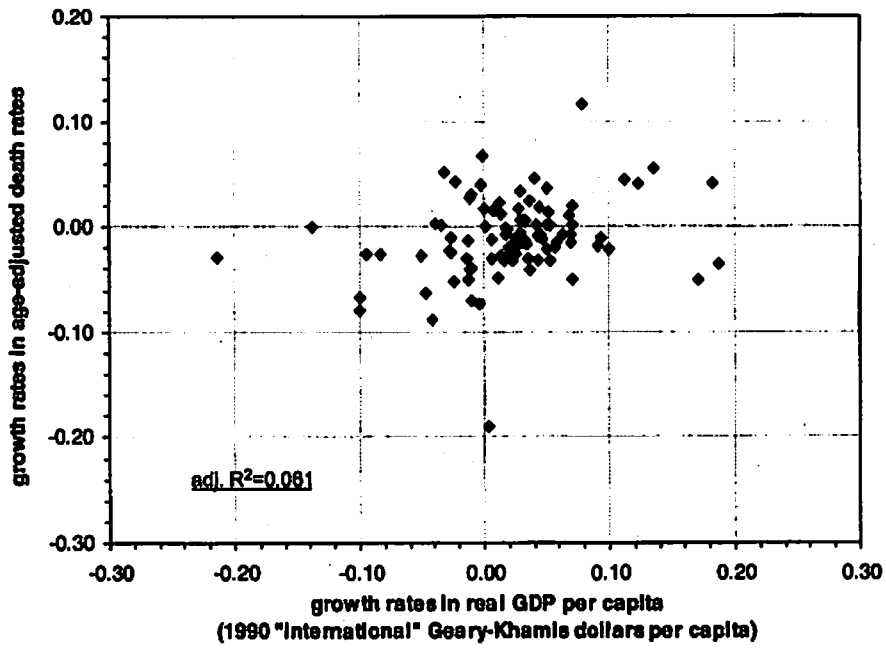

Figure 2 Relation of growth rates in real GDP per capita to growth rates in age-adjusted death rates, US 1901–2000

Many would immediately turn their attention to the fundamental contributions of economic growth to the reduction of poverty—through the elevation of real incomes via basic earnings and government and employer contributions to the 'social safety net'. For a broader view, it is important to keep in mind investment in the sciences and industrial technologies that directly minimize harm to health, including improved ergonomics, injury control, and reduction of toxic emissions. Of enormous importance is the considerable investment in new medicines, types of surgery and prosthetics, structure of care and hospital facilities and ambulance services.

Additionally, there is the considerable investment, during the last 30 years, in facilities catering to the requirements of the disabled and an older (and in the long-term more frail) population; these include housing, transportation access, and working conditions. More recently, we have had investment in the information produced by epidemiological researchers on the importance of 'lifestyle', including various aspects of consumption (calories, fats, vegetables, and fruits), as well as alcohol and tobacco use and exercise—all involving changes that have become staples of western industrialized culture. In summary, the capacity for investment in new technologies, health sciences, and education have made it possible to create a civilization in which health and life-expectancy outcomes become increasingly predictable.

Growth in GDP and mortality rates

Despite considerable evidence of the fundamental importance of economic growth to the increased health and life expectancy of industrialized country populations, is it likely that a positive relationship exists between GDP growth rates and mortality growth rates? When answering this question, it is important to bear in mind that the association reported by Tapia Granados refers to an extremely short-term relation—i.e. without lag and without any trends—that connotes a nearly simultaneous occurrence of higher GDP growth rates and higher mortality rates increasing during the same year, but without reference to long-term or even medium-term relations. The epidemiologist would have considerable difficulty interpreting such a relationship, because essential to the inference of causal relations is the time precedence criterion—i.e. the occurrence of the independent variable prior to that of the dependent variable.

Since the long-term relationship (i.e. over at least 10 years) between GDP per capita and declining mortality rates has been observed several times,[21–23,31] and is essential to the long-term trends in mortality reduction, it is clear that economic growth is inversely related to mortality. Under these conditions, is it likely that the zero-lag relation between GDP and mortality rates is actually positive? It is possible that these two observations are consistent with each other and this hypothesis has been published by the present author.[21,23,31] The underlying concept is that while medium- to long-term economic growth is the central source of improvement in life expectancy, the earliest phase of that growth—i.e. within the first year—can actually require increased adaptation.

This phenomenon has been referred to as the distinction between the trends in economic growth vs 'rapid' economic growth.[23,31] These were not the first citations of the positive relation between economic upturns at lag zero and increased mortality. The initial observation was by Dorothy Thomas,[31] utilizing the GDP national accounting method, which had recently been created by her colleague Simon Kuznets. Thomas reported the relationship without attempting an explanation. The original interpretation offered by the present author was based on the idea that while the long-term trend of GDP per capita was the fundamental source of increased life expectation, the volatility of changes in that growth were—in the very short term—a source of increased mortality.[23] In other words, it is

the extent of unpredictable change itself, as compared with sustained growth—or, in classical economic terms 'trend growth'—that contributes to mortality in the very short term. This formulation was built on Durkheim's concept of anomie as related to economic fluctuations, with both rapid economic growth and recession producing mental distress.[32]

Since 1979, the paradox of a positive relationship between GDP per capita and age-adjusted mortality rates in the US has undergone further exploration. Potentially, the most relevant and powerful explanation lies in Schumpeter's identification of the capacity for 'creative destruction' that is the source of economic growth—namely, innovations. Innovations are the basis of improvement in both productivity and the quality of goods and services,[33] but they are also the source of stress in social change. Schumpeter's destructive element lies in the elimination of jobs and termination of businesses that are unable to compete with more up-to-date innovations and procedures. Such innovations are introduced by way of investment, especially during periods of the most rapid economic growth—i.e. economic upturns. Thus, in the very short term, the introduction of new technologies within firms or the establishment of new firms that embody the newer technologies will impose on employees the learning curves or 'growing pains' of organizational adjustment. This will mean adaptation to new ways of working and the acquisition of new skills as well as the threat of loss-of-job status and income in a changing social hierarchy. At the same time, this period of rapid economic growth is the time at which the speed, intensity, and volume of production will be at it's highest since aggregate demand is at a maximum. The issue of work intensity is now prominent in the literature on work stress as pertains to injuries and cardiovascular illness.[34,35]

The intensity and overall workload (productivity per worker) also increases during the economic upturn for another important reason. The unemployment rate is known as a 'lagging' business cycle indicator. This means that it continues to increase or at least remain high after recession is over—i.e. in the upturn phase of the business cycle. Unemployment remains high at this time because employers are still reluctant to hire, or rehire former, employees since they do not yet have an indication that economic growth will be sustainable. The implication is that, despite rising orders for work, new employees will not be brought on very rapidly and the work staff will be assigned a greater volume of work despite potentially increasing stress levels.[36] Tapia Granados[30] also follows Ruhm[37] in proposing that economic upturns lead to elevated consumption of alcohol, tobacco, calories, etc. It is true that such consumption patterns have been shown to influence chronic disease mortality—but they do so over a minimal lag period of 5–10 years and are not specifically related to economic upturns.[38–43]

Long-term significance of unemployment

In considering the potential impact in the US of unemployment on ill health, attention should be directed to the duration of unemployment. In particular, since the Second World War in the US ~50% of the unemployed remain so for <5 weeks. This means that for a very large proportion of the unemployed, the return to employment—whether or not to the same firm—occurs in little more than a month. It would not seem that these short-term unemployed would be under major stress owing to loss of income, social status, or social relationships as to be at considerably greater risk for poor health or mortality. It is plausible that the stress of job change—i.e. adaptation to the requirements of a new position—could involve additional health risks.

Of substantially greater importance for health is unemployment lasting at least 6 months and certainly more than 1 or 2 years. The presumption is that these longer-term unemployed suffer considerable loss-of-job status, skills, income, social insurance and are at risk for long-term poverty. The evidence indicates that the longer-term unemployed are at major risk for downward social mobility, i.e. a substantial decline in socioeconomic status. The basic epidemiological literature informs us that a considerable reduction in the level of socioeconomic status is a significant risk to health and life expectancy. The literature on the long-term unemployed also shows increased exposure to alcoholism and use of other psychotropic substances such as tobacco and less expensive/nutritious foods. Equally important, the long-term unemployed are especially at risk for damaged family and friendship relations, quite apart from the social isolation induced by the loss of employment itself. Therefore, a time lag of at least a decade for increased health risk among the long-term unemployed is plausible and has been demonstrated for the UK.[23,44,45]

Interaction between unemployment and economic growth

The economist Okun has formulated a principle (Okun's 'law')[46] which specifies the potential reduction in the unemployment rate that would result from a prior specified increase in economic growth. This intrinsic relationship between economic growth and unemployment means that the experience of recession in a population is a joint function of income and employment loss.

If we are to represent the combined influence of economic growth and unemployment on subsequent mortality, we require a model that will include the (multiplicative) interaction of these two variables. In the unemployment–economic-growth situation, the population is most at risk for health damage when GDP per capita is low and unemployment is simultaneously high. This result is partly owing to the fact that two damaging health risks are interactively more harmful than one and, as indicated above, the first few months of a period of economic growth can carry health risks owing to the introduction of innovations, high work pressure on employees and the reintegration of the formerly unemployed.

Time-series model

This is the first century-long model of mortality rates based essentially on economic factors. It is nevertheless a preliminary model in that it consists of only three principal independent variables: (i) the long-term effects of economic growth over 0–11 years, (ii) the long-term effects of unemployment over 0–11 years, and (iii) the interactive effect of unemployment and GDP per capita over 0–11 years. For variables (i) and (iii) we

hypothesize a negative sign, and for variable (ii) a positive sign. This model, where all variables are logarithmically transformed, is examined in relation to age-adjusted mortality for the US over the period 1901–2000. Dummy variables are used to deal with potential external shocks to the mortality rate and to the economy owing to the First and Second World Wars, identifying the years 1918 and 1945 as those with the largest numbers of military personnel on active duty. (1918 is also the peak year of the influenza pandemic.) We utilize the error correction method[47] by which are estimated both a set of relationships between the levels of the independent variables and levels of mortality rates, and a first difference (annual change) model for the independent and dependent variables which includes the residual term from the initial level model. While the level model accounts for the fundamental non-linear, trend-like, and large oscillating movements in the age-adjusted mortality rate, the same variables explain nearly 60% of the annual changes in mortality rates (see Table 1 and Figures 3 and 4).

The weighted sums of the lagged variables—estimated by the Shiller procedure[48]—of the three variables distributed over 0–11 years (GDP per capita, unemployment rate, interaction of GDP per capita and the unemployment rate), are taken to represent their cumulated impact. It is found that the 0–11 year cumulative impact of GDP per capita is significantly inversely related to mortality; and the 0–11 year cumulative impact of the unemployment rate is significantly positively related to mortality. Similarly, as hypothesized, the multiplicative interaction of GDP per capita and unemployment cumulated over 0–11 years shows a significant negative sign (see Table 1).

Conclusions

Economic growth, cumulatively over at least a decade, is the central factor in mortality rate decline in the US over the 20th century. However, in the very short term—i.e. within the first few months of that decade—rapid economic growth is associated with increased mortality, probably owing to the initial stresses of adaptation to new technology in combination with greater work volume, speed, and duration. This very short-term effect disappears within a year and the medium- to long-term lagged effect of economic growth is entirely related to mortality decline.

Similarly, with respect to the unemployment rate in the US, the first year (without lag) will frequently show a negative sign in relation to mortality, but thereafter, and at least for the following decade, the effect is to increase the mortality rate. Thus, the net effect of increased unemployment is a substantial increase in mortality. But even the apparent short-term mortality rate increase related to reemployment is probably owing to the stresses of adaptation to new employment in the face of rapid economic growth. It can be argued that this need to adapt to new work by the formerly unemployed—owing to their having been made unemployed as a result of the previous recession—is an intrinsic part of the health damage attributable to unemployment. Thus, unemployment over a cumulative time period is strongly positively related to mortality increases.

To argue that economic expansions increase mortality is to emphasize peripheral effects to the exclusion of the main health effects of economic growth. It is analogous to pointing out the side-effects of antibiotics or surgery without mentioning their

Table 1 Multivariable relations between macroeconomic factors and age-adjusted mortality rates in the US, 1901–2000 (all variables in logarithmic form)

	Constant	Time trend	Age-adjusted mortality (lag 1 year)	Dummy 1918	Dummy 1945	Interaction GDP * UR (lags 0–11 years)	Unempl. rate (lags 0–11 years)	GDP per capita (lags 0–11 years)	Residual from level model (lag 1 year)	R bar square	Durbin-Watson
Model 1											
Level											
Coefficient	7.557	−0.007		0.098	−0.067	−0.517	0.468			0.994	2.240
(P-value)	(<0.001)***	(0.776)		(<0.001)***	(0.004)**	(<0.001)***	(<0.001)***				
First difference											
Coefficient	0.002			0.093	−0.057	−0.604	0.547		−0.575	0.548	2.276
(P-value)	(0.524)			(<0.001)***	(0.007)**	(<0.001)***	(<0.001)***		(<0.001)***		
Model 2											
Level											
Coefficient	8.44		−0.112	0.098	−0.080	−0.335	0.280	−0.247		0.994	2.262
(P-value)	(<0.001)***		(0.118)	(<0.001)***	(<0.001)***	(0.004)**	(0.016)*	(0.019)*			
First difference											
Coefficient	0.002			0.097	−0.068	−0.311	0.251	−0.277	−0.566	0.571	2.270
(P-value)	(0.601)			(<0.001)***	(0.001)**	(0.012)*	(0.038)*	(0.001)**	(<0.001)***		

* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$.

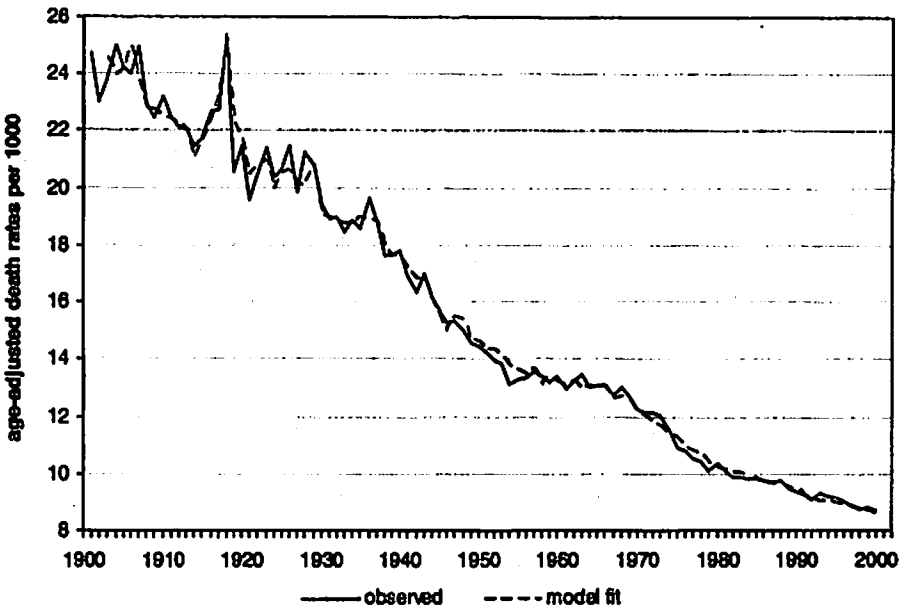

Figure 3 Multivariate relations between macroeconomic factors and age-adjusted mortality rates in the US, 1901–2000, level model

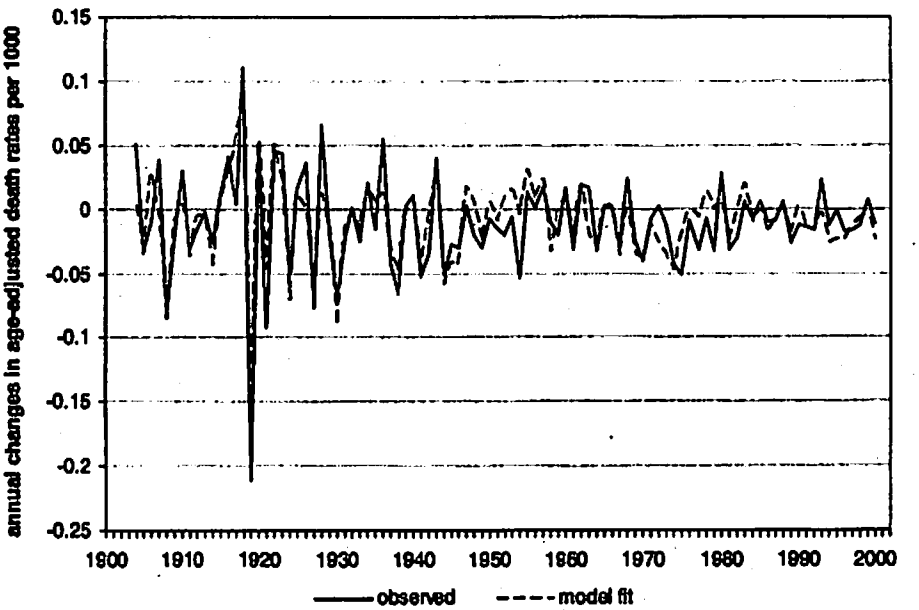

Figure 4 Multivariate relations between macroeconomic factors and age-adjusted mortality rates in the US, 1904–2000, first difference model

primary life-saving properties. An even closer analogy might be to claim that increased hospital, school, or home construction brings increased death without comprehending the life-sustaining effects of health care, education, or housing. Thus, it is crucial to place the health impact of the small oscillations represented by annual changes in economic growth into the broader context of the principal medium- and long-term trends of economic development.

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Commentary: The economic business cycle and mortality

Eric Neumayer

There is much evidence that economic recessions can have detrimental health effects for those losing jobs or in fear of losing their jobs. The unemployed in particular are vulnerable as in addition to material losses, they also potentially lose access to social networks, self-esteem, self-confidence, and a structured life schedule—all factors known to affect health.[1,2] Therefore, it is natural to presume that population health as measured by mortality moves counter-cyclically, i.e. one would expect mortality to be up in economic recessions. Yet, there is mounting evidence to the contrary: mortality is up in times of economic expansion and down in recession.[3–7] Importantly, the two pieces of evidence are not necessarily inconsistent with each other. This is because economic upturns can affect the health of many more people or affect health more strongly, for example, via higher working times, job-related stress, increased consumption of health-damaging consumer goods, and an increase in work-related accidents. Therefore, the overall effect of economic expansion on mortality can be negative despite the beneficial health effects of reduced fear of job loss and reduced number of unemployed people.

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Still, given its seemingly counter-intuitive results, it is important that the evidence showing mortality rates to move pro-cyclically is tested in different samples, different time periods, and with different estimation techniques to check its robustness. Tapia Granados[6] has provided a valuable addition to this literature. His time-series analysis of mortality rates in the United States over the period 1900–1996 complements nicely existing evidence based on panel data analysis across the states in the United States,[3] Germany,[7] or OECD countries[4] from shorter time spans as well as other time-series analysis by the same author for Spain and Sweden.[5] Time-series analysis evidence is perhaps particularly welcome since Brenner's contrary results suggesting that recessions raise rather than lower mortality rates are largely based on time-series analysis as well,[8] but note that his research on the issue had started >20 years earlier. I particularly like that Tapia Granados[6] provides estimates both for the entire time period and for selected sub-periods. The fact that the estimated coefficients for the total age-adjusted mortality rate do not differ much across time periods is a striking result, given that most other existent evidence is from much more recent time periods.

However, time-series analysis poses various econometric challenges that are either non-existent or much less prevalent in panel data analysis. Despite space constraints, I would have

Community Medicine

MORTALITY AND THE NATIONAL ECONOMY

A REVIEW, AND THE EXPERIENCE OF ENGLAND AND WALES, 1936–76

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Summary The long-term upward trend in real per-caput incomes is associated with better health, but the smoothed curve disguises cyclic fluctuations, recessions followed by periods of rapid economic growth, which may be stressful to certain groups in the workforce and, by extension, to their families. Especially vulnerable in recessions are those in industries whose goods or services are not essential, the less skilled who are the first to lose their jobs and the last to get their jobs back when the economy improves, and those who find that over one turn in the business cycle their skills are no longer needed. For these groups, most often to be found in the lower socioeconomic classes, this lack of economic security is stressful: social and family structures break down and habits that are harmful to health are adopted. Acutely, if the effect manifests as a psychopathological event (e.g., suicide), or after a time lag of a few years or even one or more turns of the business cycle for chronic diseases, economic recessions and subsequent periods of rapid economic growth are associated with a deceleration in the normally declining curve of mortality against time. This model, tested previously on U.S. data, has now been confirmed on data for England and Wales.

INTRODUCTION

SINCE at least the mid-19th century England and Wales has seen a secular decline in mortality-rates associated with long-term improvements in economic conditions. The standard of living has improved and economic instability and insecurity have been much reduced. Over the same time period there has emerged a consistent inverse relation between socioeconomic status and mortality: in general, the higher the income, occupational, and educational levels of a population, the lower its mortality-rate.[1–4] This mortality difference is not confined to infectious diseases, attendant upon poor nutrition and sanitation and poverty, but holds for many of the chronic diseases now afflicting modern industrialised nations.

In both the United States and Britain the socioeconomic differentials in mortality-rates may indeed have increased since the second world war[5]—a perplexing trend in Britain where a high-quality health service available to all was introduced in 1948. Even if different socioeconomic groups in Britain do not make equal use of the Health Service,[6] it is unlikely that differential utilisation is solely responsible for the inverse relation between socioeconomic status and mortality.

Factors other than health care which might account for socioeconomic differentials in mortality include intemperate life habits (immoderate use of alcohol, smoking, overeating, lack of exercise)[7] the disruption of basic social patterns involving family and community,[8–12] such as migration, urbanisation, and divorce or widowhood; and the influence on the probability of illness of both stress from specific deleterious events, such as unemployment, retirement or work under conditions of financial and job insecurity,[13–17] and of the cumulative influence of various stressful events over a considerable time span.[18–20] The hypothesis of this paper is that economic instability and insecurity increase the likelihood of immoderate and unstable life habits, disruption of basic social networks, and major life stresses—in other words, the relative lack of financial and employment security of lower socioeconomic groups is a major source of their higher mortality-rates. The hypothesis predicts that indicators of economic instability and insecurity, such as unemployment and unusually rapid economic growth-rates, should be associated, over time, with higher mortality-rates, while the smooth, long-term exponential trend in economic growth should be inversely correlated with mortality-rate trends.

ECONOMIC FLUCTUATIONS AND LIFE STRESS

Two classic sequences of economic loss associated with life stress and, subsequently, with morbidity and mortality can be identified. In the first the population is exposed to continuous economic instability and insecurity long term, which approach crisis levels during recessions. Particularly at risk are workers in industries whose fortunes depend on cyclic changes in the economy—e.g., in household equipment, cars, clothing, building, and recreation, these being goods and services the purchase of which can be deferred until a recession has passed. Also at risk here are the semi-skilled and unskilled workers. They are among the first to lose their jobs and among the last to be rehired. Populations subject to this "cyclic unemployment" show increased mortality two to three years after the recession which initiated a process of morbidity.

The second classic sequence is associated with changes in the structure of the national economy due to technological innovation within a particular industry through automation, for example, or the replacement of natural fibres by synthetics or because of relocation within or even outside a country where an industry has traditionally flourished. The risk of redundancy rises for workers who represent only a marginal gain to a firm; for them alternative employment in another industry will almost certainly be at a job status and income well below those previously held. Radical change in the individual's social environment can be seen historically too, in the migration of farmworkers to manufacturing centres during the lengthy process of urbanisation and in the post-war decline in manufacturing employment and the concomitant increase in employment in service and communications. In this sequence of "downward social mobility" the illness process begins with recession, and, within two to three years, the likelihood of mortality is greatly increased, particularly if another stress appears. For the downward mobile, the next major source of stress usually occurs during their reintegration into the economy, in the period of rapid economic growth

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which follows a recession; for these individuals the probability of mortality increases during rapid economic growth. Some will not be successfully reintegrated, ending up among the intermittently employed or forced into early retirement. In these cases, mortality may be associated with recessions long after the period of initial economic loss. Yet others may find that their work skills are no longer consistent with the pace of industrial development: the potential for job losses here is illustrated by the microprocessor.[21]

DEVELOPMENT OF BASIC MODEL

The basic model originated with the findings that the rate of first admissions to mental hospitals in New York State was inversely related to the rate of employment during 1914–67 and to an index of business cycles for 1841–1909.[22] Data for the entire United States showed that indices of acute pathological disturbances, including suicide and homicide, rose within a year of increased unemployment-rates.[23] From the psychopathological the research next focused on chronic physical illnesses with a stress element, especially cardiovascular diseases. The earlier study of psychopathology suggested that increases in cardiovascular morbidity would follow increased unemployment within a few months,[24] but that mortality would rise only after a considerable time lag. The lag was found to be two to three years for most age-groups and both sexes.[25] Higher correlations were found in lags of two or three years than at time zero or one year. The same relation, with a nearly identical lag structure, was found by Morris and Titmuss in 1944 for rheumatic heart-disease.[26]

The impact of recession on cardiovascular mortality, which represents 60–70% of total mortality in industrialised societies, raised the question of whether the secular decline in the total mortality-rate might be inversely related to the long-term economic growth trend. Since recession leads to increased mortality, long-term growth should lead to lower mortality. Evidence for this came from findings that the infant-mortality rate, a sensitive index of national economic development, also rose within one or two years of recessions in the U.S.[27]

Initial tests of equations incorporating indices of both long-term economic growth and recession began with mortality related to alcohol consumption, including cirrhosis of the liver.[28] These tests were followed by a large-scale study, prepared for the Joint Economic Committee of the U.S. Congress.[29] A multivariate model incorporated an indicator of economic growth, the rate of unemployment, and the inflation-rate. An additional innovation was the use of the polynomial distributed lag method, which permitted a test of the assumption that there probably was no single discrete lag between economic change and mortality-rates for the entire population. Instead, it seemed realistic to assume that, within a five-year period, the most vulnerable populations (e.g., the aged, previously ill, and lower socioeconomic groups) would respond relatively early to economic trauma, while the least vulnerable would show a longer lag. Thus, a distributed-lag estimate, beginning at year zero and ending at year five, replaced the single optimum discrete lag estimate.

Another significant addition to the model stemmed from the emphasis, in work on the impact of stress on morbidity, of the scale of the adaptation required to meet a group of quite ordinary life changes which accumulate over two or more years.[18,20] Of equal relevance were epidemiological studies which had identified adaptation to urban industrial life, an instance of critical change in the socioeconomic environment, as an important risk factor in cardiovascular disease.[8–10] The implication was that for a population which had endured losses of employment and income during recession, adaptation to new work and living environment during the following period of rapid economic growth would be additionally stressful. This view is strongly supported by evidence from the demographic history of major European cities.[30]

As a result rapid economic growth was included as a predictor of mortality. This model was successfully tested with U.S. data on suicide, homicide, cardiovascular mortality, and age-specific total mortality.[31,32] It then became possible to explain the anomaly, discovered by Thomas,[33] that the early phase of business-cycle upturns tended to be positively correlated with mortality-rates during the late 19th and early 20th centuries in Britain.

Using a graphic analysis, Eyer interpreted the Thomas anomaly differently: high unemployment, which is moderately inversely correlated with business-cycle indices at zero lag, causes lower mortality-rates at zero lag.[34] However, when this interpretation was tested on U.S. data by statistical methods, the unemployment-rate did not consistently show a negative simple correlation with the mortality-rate; usually the relation was positive even at zero lag, and, at lags of two to ten years the relation was consistently positive and significant. With controls for long-term economic growth, the lagged relations between unemployment and mortality (for all causes or for chronic diseases) became stronger, while the unlagged relations became weaker.

With increasingly larger proportions of total mortality being attributed to chronic disease, the lag pattern of heightened mortality behind higher unemployment should be lengthening. Tests based on U.S. data showed that the distributed lag both began at a later time (between three and four years) and extended over a longer period (ten to fifteen years) as the interval of analysis covered increasingly later time spans (e.g., 1950–76, as compared with 1940–76 or 1909–76).

The findings of increased mortality-rates for chronic diseases beginning with a lag of one year, and running as long as fifteen years subsequent to the initial periods of recession, led to the following conclusions:

(1) Recession causes severe economic loss and downward social mobility, which places many of those affected in a long-term state of vulnerability to subsequent recessions and periods of rapid economic growth.

(2) Severe economic loss and downward mobility can initiate long-term processes of chronic illness, which may endure for several years, prior to mortality.

(3) Therefore, in summary, severe economic loss and downward mobility initiate patterns of interaction over several years between chronic disease processes and vulnerability to economic stress.

ELEMENTS OF EXPLANATORY MODEL

The explanatory model, which permits the prediction of mortality patterns, consists of four main components: (1) the smooth exponential trend of long-term economic growth, measured by real per-caput income; (2) the rate of unemployment; (3) "rapid economic growth", measured by deviations from the long-term exponential trend in real per-caput income and by annual changes in the rate of growth of real per-caput income; and (4) government expenditure on welfare as a percentage of total government expenditure.

The *long-term trend in economic growth*, with major influences on nutrition, sanitation, and education, dominates the secular history of decline in the mortality-rate, up to the time when infectious diseases ceased to be important causes of death (1930–50). Since that time, its main influence has been to reduce the amplitudes of the cycles and fluctuations in national economic behaviour that have been intrinsic to the process of economic growth itself. These cycles and fluctuations ("economic instabilities") remain sources of stress, despite improved

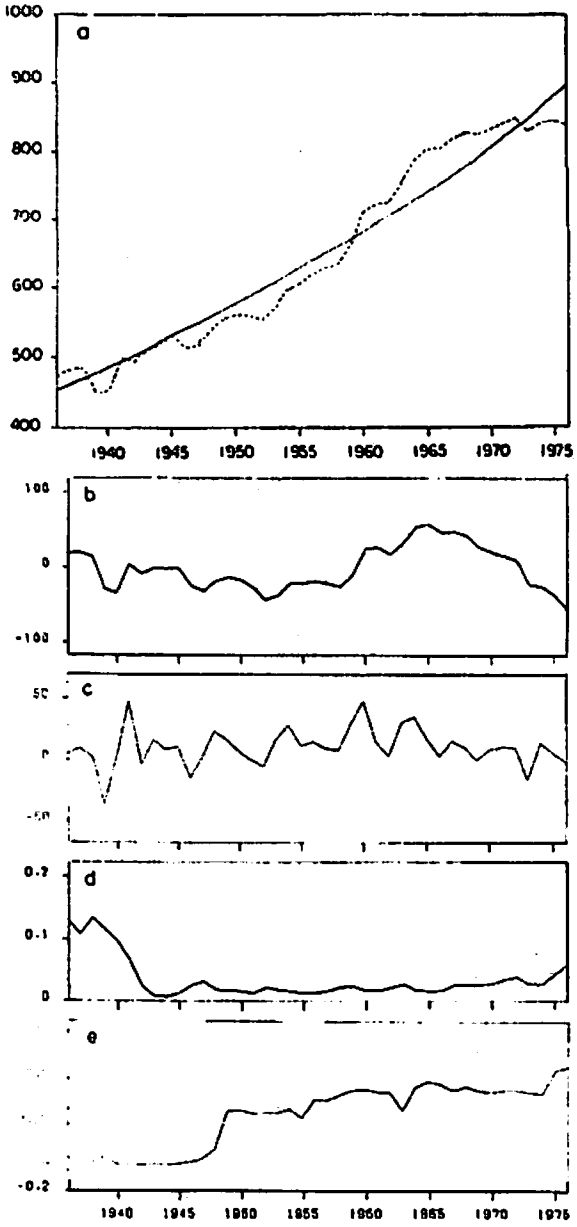

Fig. 1—Data for England and Wales 1936–76.

(a) Real per-caput income (broken line) and its smoothed exponential trend (in 1970 £ per person).

(b) Medium-term changes in growth-rate, representing departures from smoothed exponential trend of real per-caput income.

(c) Annual changes in real per-caput income.

(d) Proportion of civilian labour force unemployed.

(e) Proportion of welfare expenditures to total government expenditure, in £ million.

management of the national economy and of individual companies. Accompanying long-term economic growth have come technological developments in medicine. Their significance to the extension of the life span may be controversial, but, to the extent that disability and pain induce stress, health care in the long run may have contributed significantly to the reduction of chronic disease-related mortality.

The *unemployment-rate* reflects loss of income, social status, and close personal attachments for individuals and entire families, but as a national indicator of recession, it becomes an indirect global measure of income loss and work stress in employees of firms experiencing economic difficulty. Since loss of economic and social status is, theoretically, a major source of a series of disturbances, including dangerous life habits, breakdown of family and community social structure, stress-associated physical illnesses, and depression, we examine the association between the unemployment and mortality-rate over a lengthy lag period of zero to ten years.

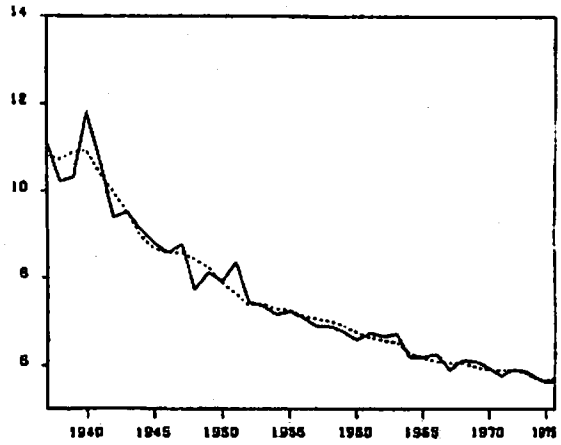

Fig. 2—Age-adjusted total mortality-rates per 1000 home population.

Actual (—) and expected (- - - -) values based on fit of composite economic change model including long-term and rapid economic growth variables, unemployment-rates over 0–10 years, and percent of Government welfare expenditures. England and Wales, 1936–76.

Rapid economic growth is harmful for specific minorities, particularly those who have suffered economic loss and are attempting to become integrated into the economy, especially during a time when other workers will be gaining significantly in income. It is also a period of rapid introduction of new technologies which produces a higher risk of accidents and the threat of job loss or demotion in the process of industrial reorganisation.

The *government allocation for public welfare* (social security) acts more as a control variable. The size of the allocation is determined by need, and the proportion of the population that requires help will in turn depend on the performance of the national economy. However, in most industrialised nations there is some impoverishment even under the most favourable national economic conditions, so we need to find out whether government transfer payments meant to alleviate economic distress do improve health as well.

EXPERIENCE IN ENGLAND AND WALES

An attempt was made to test the model by analysing the relations between trends in economic indices and mortality-rates in England and Wales over the years 1936–76. The raw data for this period are illustrated in fig. 1. Table I indicates the impact of each of the independent variables on the mortality-rate. The hypothesis that long-term economic growth is inversely related and unemployment (indicating recession) is positively related to mortality-rate trends is sustained for all age-groups. The rapid-economic-growth variables are weakly related to mortality; the residual from the long-term economic growth-rate shows no statistical significance, while the annual growth-rate shows significance for the population aged 10–44. Finally, Government expenditure on welfare is inversely related to mortality-rates for infants and for children aged 1–4.

TABLE I—COEFFICIE

Age	Intercep
Total[1]	12·57**
Infants	59·76**
1–4	4·31*
5–9	1·22
10–14	0·73**
15–19	0·64†
20–24	1·24*
25–34	1·86**
35–44	3·91**
45–54	7·59**
55–64	21·72**
65–74	57·63**
75–84	145·48**
85+	312·05**

For t (columns 2–7) an
For age-group 5–9 Coc
1. Age-adjusted mortal
2. Sum of coefficients
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strong prediction fo
2) as it does for age
(table I). The imp
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Fig. 3 suggests th
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TABLE II—COEFFICI

Lagged effects of unemployment in ye
Sum of years 0–5[2]: Annual changes
Annual changes and annual changes squ
Discrete years of lag: Annual changes
0
1
2
3
4
5

For t (columns 2–6) a
1. Cochrane-Orcutt
2. Distributed lag: thi

TABLE I—COEFFICIENTS FOR MULTIPLE REGRESSION EQUATIONS OF AGE-SPECIFIC MORTALITY-RATES ON NATIONAL ECONOMIC INDICES: ENGLAND AND WALES, 1936–76

Age	Intercept	Economic growth trend	Growth trend residuals	Annual change in growth-rate	Welfare expenditures	Unemployment over 0–10 year period[2]	R^2	Durbin-Watson statistic	F statistic
Total[1]	12·57**	−0·099**	−0·033	0·022	−0·33	20·68**	0·97	2·30	166·66**
Infants	59·76**	−0·05**	−0·004	0·033	−17·79*	156·57**	0·99	1·73	387·98**
1–4	4·31*	−0·002**	−0·033	0·0005	−6·57**	19·59**	0·95	1·69	97·50**
5–9	1·22	−0·001	0·003†	0·007	−0·54	9·97**	0·94	1·92	67·89**
10–14	0·73**	−0·0007†	0·0001	0·002†	−0·14	5·26**	0·98	2·00	232·37**
15–19	0·64†	−0·0003	0·0004	0·004*	−0·075	11·04**	0·98	2·05	197·97**
20–24	1·24*	−0·0009	−0·0005	0·004*	−0·18	13·00**	0·98	1·55	223·17**
25–34	1·86**	−0·001†	−0·001	0·005*	−0·26	10·79**	0·98	1·41	230·35**
35–44	3·91**	−0·003**	0·0001	0·003†	−0·16	12·84**	0·98	1·93	185·32**
45–54	7·59**	−0·003	−0·002	−0·002	−0·70	19·53**	0·95	2·09	95·84**
55–64	21·72**	−0·01	−0·002	−0·002	0·74	27·06**	0·89	2·08	38·16**
65–74	57·63**	−0·030**	−0·006	−0·011	3·82	47·23**	0·85	2·22	26·31**
75–84	145·48**	−0·079*	−0·055†	−0·025	14·09	100·28*	0·82	2·12	21·09**
85+	312·05**	−0·16†	−0·056	−0·12	41·65	300·86*	0·64	2·24	8·48*

For t (columns 2–7) and F †p<0·05, *p<0·01, **p<0·001.
For age-group 5–9 Cochrane-Orcutt transformation was used to minimise residual autoregression.
1. Age-adjusted mortality, based on 1940 population age structure.
2. Sum of coefficients for second degree polynomial fit to distributed lag. This degree of polynomial is the best fit for the total age-adjusted mortality-rate, but not necessarily for those of each of the thirteen age-groups. Using the identical lag structure for all of the age-groups serves to test the general applicability of the model to populations of different age.

The combined effect of all four independent variables yields strong prediction for the total age-adjusted mortality-rate (fig. 2) as it does for age-specific mortality rates at nearly all ages (table I). The importance of using the entire explanatory model, rather than a single component such as the unemployment-rate, is illustrated in fig. 3. Here we concentrate only on annual fluctuations in the total (age-adjusted) mortality-rate, in effect, withholding from consideration the long-term trend and all other smooth movements in mortality. In this case, short-term change (i.e., annual change summed over 0–5 years' lag) in the unemployment-rate alone is only a moderately good predictor (table II).

Fig. 3 suggests that short-term changes in unemployment as a measure of economic loss are the most important source of influence on annual fluctuations in mortality when both variables show at least moderate levels of variation. This may indicate a threshold below which the unemployment-rate is not a sufficient predictor of annual changes in total mortality, and other variables are also required. For example, adding annual changes in unemployment in exponential (i.e. squared) form, thus approximating a portion of the threshold effect, adds considerably to the explanation of annual changes in mortality (table II).

Finally, of the six years of lag of unemployment used as predictors, the years of strongest predictive value for total mortality are at lags 1, 2 (particularly), and 5 (table II); this coincides with the United States experience for total and especially chronic disease mortality.[25,29]

The lagged effect of unemployment varies according to the time span and specific causes of death under analysis. Suicide and homicide, for example, show increases within a year of unemployment increasing, while cardiovascular mortality begins to increase two to three years after increased unemployment, the effect persisting for ten to fifteen years. For the period starting in 1950, the lag structure for total and chronic disease mortality following heightened unemployment begins and ends later than when the analysis is started in 1936 or 1940. These findings, which Granada's *World in Action* programme

TABLE II—COEFFICIENTS[1] FOR MULTIPLE REGRESSION EQUATIONS OF ANNUAL CHANGES IN AGE-ADJUSTED TOTAL MORTALITY ON ANNUAL CHANGES IN UNEMPLOYMENT RATES, ENGLAND AND WALES, 1936–76

Lagged effects of unemployment in years	Constant	Sum of regression coefficients: annual changes	Sum of regression coefficients: annual changes squared	Regression coefficients	Rho	R^2	Durbin-Watson statistic	F statistic
Sum of years 0–5[2]:								
Annual changes	−0·82E-1	15·68†		..	−0·34†	0·35	2·25	4·69*
Annual changes and annual changes squared	−0·17†	70·77†	1572·13†	..	−0·41*	0·53	2·14	4·30*
Discrete years of lag:								
Annual changes								
0	−0·13†	..	..	4·80	−0·28†	0·09	2·26	3·55
1	−0·11†	..	..	8·76†	−0·35†	0·13	2·34	5·86†
2	−0·90E-1	..	..	12·69*	−0·24	0·21	2·24	9·99*
3	−0·14†	..	..	−1·39	−0·24	0·07	2·18	2·69
4	−0·14†	..	.	−0·33	−0·25	0·06	2·20	2·62
5	−0·11†	.	..	6·80	−0·23	0·11	2·16	5·92†

For t (columns 2–6) and F †p<0·05, *p<0·01, **p<0·001.
1. Cochrane-Orcutt transformation was used to minimise residual autoregression
2. Distributed lag: third degree polynomial fit

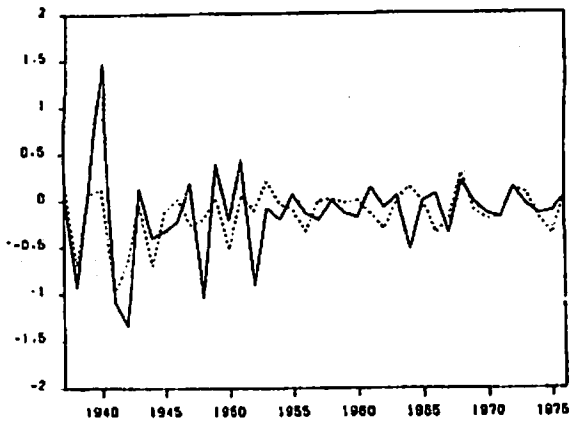

Fig. 3—Annual changes in age-adjusted total mortality-rates per 1000 home population.

Actual (—) and expected (- - - -) values based on lagged effect of annual changes in the unemployment-rate over 0–5 years. England and Wales, 1936–76.

reported graphically on British television on Feb. 5, 1979, will be presented in detail elsewhere.

Table III shows a cross-sectional analysis by county for 1971 indicating the association of unemployment and of high income, to mortality-rates, controlling for employment in manual occupations and employment in the chemical industry, a major source of environmental contamination. This analysis tends to confirm the time-series results. Indeed, three major factors account for over 90% of the variance: unemployment (positive), high income (inverse), and the proportion of the population over age 54 (positive). Table II also indicates that, when the cross-sectional relations are specified further by major cause of death, adjusted for age, sex, and the institutional population—i.e., total cardiovascular disease, ischæmic heart-disease, cerebrovascular disease, and malignancies—the model is most appropriate for the total cardiovascular disease and ischæmic heart-disease, largely effective in malignancy, and least appropriate for cerebrovascular disease. These cross-sectional findings based on counties are in general similar to those of Gardner et al.[35] for county boroughs.

DISCUSSION

A theoretical model of the implications of national economic changes for mortality-rates, developed from epidemiological, demographic, and stress research and incorporating the long-term trend in economic growth, the unemployment-rate, and periods of rapid economic growth, has been refined on the basis of U.S. data and has now been applied to England and Wales for 1936–76 with results similar to analyses of U.S. data for

TABLE III—COEFFICIENTS FOR MULTIPLE REGRESSION EQUATIONS ON COUNTY-SPECIFIC SOCIOECONOMIC INDICES OF COUNTY-SPECIFIC MORTALITY-RATES: ENGLAND AND WALES—1971

Mortality-rates	Unemployment-rate (1)	High income (2)	Nature of employment: Manual workers (3)	Self-employed (4)	Farm employment (5)	Chemical industry employment (6)	Population over 54 (7)	R (8)	R (9)	F statistic: equation
Crude total	0·20**	−0·21**	0·04	−0·05	0·02	0·10*	0·79***	0·96	0·95	103·9***
Crude total	0·35**	−0·34*	0·21	0·51**	0·19	0·19**	..	0·73	0·69	17·9***
Infant	0·43	−0·12	0·23	0·09	−0·12	0·01	..	0·30	0·20	2·8
Total minus infant	0·18**	−0·21**	0·07	−0·03	0·03	0·11**	0·79***	0·94	0·93	72·6***
Standardised Total	0·52***	−0·23	0·27	−0·27	0·20	0·28**	..	0·78	0·74	22·6***
Cardiovascular	0·25	−0·41*	0·34	−0·21	0·27	0·17	..	0·63	0·57	10·73**
Ischæmic heart	0·30*	−0·47*	0·11	−0·30	−0·09	0·17	..	0·53	0·45	7·07*
Cerebrovascular	0·19	−0·16	0·42	−0·13	0·67**	0·17	..	0·57	0·50	8·31**
Malignancies	0·45**	−0·23	−0·30	−0·26	−0·60*	0·26*	..	0·47	0·39	5·63*

(1) % unemployed of civilian labour force.
(2) % of employed with income greater than £1750.
(3) % of employed with occupation as skilled, semi-skilled, and unskilled labour.
(4) % of employed with occupation as self-employed professionals, and own-accounts workers.
(5) % of employed with occupation as farm owners, managers, and labourers.
(6) % of employed in chemical industry.
(7) Population over age 54.
(8) R=estimate of variance explained.
(9) R=adjusted estimate of variance explained.
Significance of F statistics: *p<0·05, **p<0·01, ***p<0·001.

Variable	*Source of data*
Unemployment for counties and county boroughs	1971 Census of England and Wales: economic activity, county leaflets. H.M.S.O. (old county designation).
Occupational groups (SEG categories) employment	1971 Census of England and Wales: economic activity, county leaflets. H.M.S.O. (old county designation).
Chemical industry employment	1971 Census of England and Wales: economic activity, county leaflets. H.M.S.O. (old county designation).
Income for counties	Survey of Personal Incomes: 1970–1971. Board of Inland Revenue. H.M.S.O.
Population for counties and county boroughs	1971 Census of England and Wales: county reports, H.M.S.O. (old county designation; 1966 population data replaced 1971 data for counties where data unavailable).
Mortality for counties	Registrar General's Statistical Review of England and Wales, 1971.
Mortality for county boroughs	O.P.C.S. Mortality Division. Direct communication.

the same timespan. In addition, a cross-sectional analysis of county-specific mortality-rates for 1971 shows a pattern that is consistent with the over-time predictive equation. These cross-sectional findings obtain when age, sex, the institutional population, and the presence of the chemical industry, for example, were controlled. The validity of the model for two populations (the U.S. and Britain) so different in ethnic groupings, geography, climate, industry, government, and health and welfare policies, is encouraging.

The secular decline in mortality-rate is accounted for by the long-term trend in economic growth, while fluctuations in mortality-rates are largely explained by recessional losses and rapid economic growth (i.e., economic instability). It has been argued that these instabilities in economic growth also account for the socioeconomic differential in mortality. Long-term economic growth will also moderate the problems associated with economic instability: management of the national economy improves, health care gets better in quality and availability, and more substantial income support can be provided for displaced workers and other non-participants in the labour force. Thus the long-term trend in economic growth (or "prosperity") clearly acts to reduce mortality, and certainly not to increase it as has at times been suggested. Rapid economic growth, on the other hand, does contribute to increased mortality within a year.

All three types of major economic change, additively and interactively, affect mortality trends, and the impact of any one of them cannot be estimated without statistical controls for the other two. When unemployment or rapid economic growth act to "increase" mortality, they do so by inhibiting the long-term decline in mortality-rates. Thus high unemployment in Britain in recent years is not expressed as an observable increase in mortality-rate trend, but as a slowing of the decline in mortality that would ordinarily have occurred. The mechanism by which unemployment and rapid economic growth act to slow the secular trend of mortality-rate decline is through a widening of the socioeconomic differentials in mortality. To the extent that one is able to generalise from the U.S. and British experience, the differential mortality-rate across countries depends on the interaction of real per-caput income, especially among the lower socioeconomic strata, and stable rates of economic growth. Countries with the highest real per-caput income in their poorest populations in conjunction with the most stable economic growth-rates should, and typically do, show the lowest mortality-rates at all ages.

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Explaining aggregate health status (mortality). Insights to the possible impact of the economic crisis

Technical Report of the European Commission
DG Employment, Social Affairs, Inclusion

September 28, 2009

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Introduction

The most extensive and robust findings in epidemiology of mortality patterns in industrialized countries is that "socioeconomic status" is the single most dominant, pervasive (or powerful) factor that routinely influences comparative length of life among industrialized country populations.

It can be seen that socioeconomic status of individuals, thus defined, is based on, firstly, investments in education (and other human capital including occupational training and skill). This investment in training will be a precondition to employment at different levels of occupational remuneration, including wages/salaries and benefits. Many of the benefits associated with specific wage levels will include elements of social protection that are based on both state and private sources of material welfare. This type of social protection will include various forms of social insurance i.e. unemployment insurance, involvement in active labor market policy initiatives, health and disability insurance, and payments and pensions.

Thus it can be seen that the national income and wealth of a society, i.e., real GDP per capita, is the foundation of much of individual material welfare that is described in the concept of socioeconomic status. Beyond those sources of investment in socioeconomic status are societal investments in a variety of public health and safety measures to insure social protection. These include health care sciences and technology, health and safety regulations in the workplace, environmental regulations, investment in the epidemiological and clinical sciences that identify risks to population health in terms of the monitoring of food and drug safety, safety of water and sanitation systems, new knowledge of optimal diets and the effects of alcohol and tobacco on health, and transportation safety.

In addition, of course, growth in GDP per capita is almost by definition the antithesis of recession and, therefore, unemployment. It is indeed specified under the "law" proposed by the economist Okun that a specified quantitative increase in potential GDP will reduce the unemployment rate by a specified amount.

Finally, real GDP per capita is also the source of investment in new innovation and new employment, largely through the initiation of new small firms, frequently via self-employment. The strength of self employment, then, in a well developed industrialized society, represents much of the source of innovation that will maintain or advance the rate of economic growth. Equally important, however, self-employment serves as a source of opportunity to avoid unemployment – whether that unemployment originates with economic cycles, technical and productivity development, globalization or other structural changes in the economy. Self-employment, thus, can provide a "safety catch" by which unemployment is reduced and through the development of small firms, can be an avenue through which active labor market policies can result in successful reemployment.

To exemplify this true set of factors which affect health and social wellbeing both directly and indirectly stemming from GDP growth, a model is presented which encompasses a number of the factors which are related to GDP-based investment and consumption patterns. This is a model which attempts to account for variation among industrialized countries in the age-standardized mortality rate.

The model includes a measure of the rate of productivity, namely GDP per employee. It includes as well, the rate of self-employment and both the unemployment rate for males in relation to population as well as the source of inflation, namely the consumer price index. This model also includes publicly-funded health expenditures which are derived from GDP investments. Finally it controls for a major risk factor that is well-known in the epidemiological literature for its ability to exert a short-term effect of a multiplicity of sources of illness and disability, including cardiovascular diseases, diabetes, work and home accidents and injuries, transportation accidents, suicide and homicide. This factor is alcohol consumption per capita.

Drawing on this logic, we present a second model, which examines the relation of economic change to the suicide rate in industrialized countries. Similar factors, such as GDP, self-employment, the unemployment rate, health services expenditure, as well as lifestyle factors and measures of social integration are able to produce a multi-variable model of coherence and predictability.

In a third approach, time series analyses are presented of some of the policy factors that influence age-standardized mortality in the United Kingdom and United States over the years 1970-2005. Attention is especially given to health care expenditures as a percent of GDP and the unemployment rate. Also examined is the potential effect of the dependency ratio on mortality. A time-series regression analysis is presented as well as a graphic display of the activity of the historical time-series during 1970-2005.

Finally, a concluding section is devoted to a numerical estimation of the impact, on mortality, of potential changes in the health care expenditures and the unemployment rate. Mortality figures are simulated and estimates are given under conditions where there are separate changes in health care expenditures and the unemployment rate. Estimates are also given for scenarios where both factors change simultaneously.

Major Factors in the Prediction of National Life Expectancy

1. Income

At the individual level of analysis – i.e., in epidemiological studies – individual income is a standard and fundamental inverse predictor of illness and early mortality (Andersen, Gamborg, Osler, Prescott, Diderichsen' 2005; Ecob, Davey Smith, 1999; Ettner, 1996; Kahn, Wise, Kennedy, Kawachi, 2000; Kivimäki, Shipley, Ferrie et al., 2008; Lynch, Smith, Kaplan, House, 2000). In industrialized countries, the higher the level of income of individuals, the lower the illness and mortality rates attributed to the great majority of infections, chronic diseases and mental disturbances. In developing countries similar relations are found, but the impact of income is substantially stronger. Moving from the individual to the national level, however, real GDP per capita indicates the availability of basic goods and services: nutrition, potable water, sanitary engineering, housing and other means of climate control, transportation and primary health care. At the national level, real GDP per capita – especially for industrialized societies also conveys the capacity of the society to invest in the development of science and technology, improved working conditions at higher technological levels of safety and health, financing of education at all levels, stabilization of the income of individuals and small businesses, and the many types of social protection: unemployment insurance; active labor market policies; health insurance; disability insurance; social welfare payments to impoverished, frail populations and children; social security and retirement benefits.

Income, of course, inasmuch as it conveys the purchase of goods and services of different price and quality, also conveys the special 'social status' of the purchaser. Thus, certain aspects of purchases take on the meaning of 'status symbols' by which different levels of social prestige are attributed to the purchasers. Prestige, in turn, is a principal marker of the socioeconomic status of individuals and is understood to convey differential levels of psychological wellbeing (Marmot, 2002; Marmot, Wilkinson, 2001).

2. Education

As in the case of income, educational level attained is strongly associated with lower illness and mortality rates in industrialized country populations. Educational level attained by individuals is almost universally associated with lower morbidity and mortality rates in epidemiological studies (Leigh, 1983; Ross, Mirowsky, 1999; Ross, Wu, 1995). This relationship is consistent with the more generally cited relationship – perhaps the single strongest empirical relationship in all of epidemiology – that overall socioeconomic status is a predictor of lower morbidity and mortality for nearly all diagnoses, at all ages, both sexes and populations of different ethnicity and immigration status (Smith, Hart, Hole, et al., 1998; Winkleby, Jatulis, Frank, Fortmann, 1992). The explanation of the positive relationship between educational status and health clearly involves multiple mechanisms. The most common understanding is that education itself, meaning the attainment of knowledge enables the comprehension of the most widely recognized health risks (dealing e.g., with matters of diet, environment, working conditions, stress and addictions) and the ability to secure the most timely and effective health care. Even more generally, education is the basis for adaptation to opportunities and challenges in economic and social areas of life.

Education is, of course, the most important prerequisite of higher skill and managerial levels of employment, and is virtually essential to employment in professional and administrative positions in larger economic and political organizations. Educational attainment level is, therefore, a strong factor in personal income and the maintenance and advancement of careers in technological societies. In this era of rapid technological change and globalization – leading to frequent restructuring of private firms and government organizations – relatively high education levels permit more rapid transition to new jobs and the resumption of careers. This often avoids

the substantial losses in permanent earnings that accrue to individuals who experience structural unemployment or downsizing of firms.

It should be kept in mind that educational level, a standard measure of 'human capital', has long been understood as a significant source of economic growth. Of course, the extent of education in a population is also financed from the wealth developed as a result of economic growth. This close linkage of education to the economic growth process often means that once GDP per capita is used as a variable to account for increases in life expectancy, no additional variables measuring the extent of education in a society is usually needed, even though the level of education in a population may well make a substantial contribution to a population's health. This will be especially true in comparative (i.e., cross-sectional) analyses of industrialized countries. Nevertheless, a variable that deals more directly with the extent to which the working population is highly educated can often make an additional contribution to the explanation of life expectation among industrialized societies. Such a variable, for example, is the proportion of the employed population who have achieved tertiary education.

3. *Unemployment*

The unemployment rate is well established as a risk factor for elevated illness and mortality rates in epidemiological studies performed since the early 1980's. In addition to influences on mental disorder, suicide and alcohol abuse and alcoholism, unemployment is also an important risk factor in cardiovascular disease and overall decreases in life expectancy (Linn, Sandifer, Stein, 1985; Morris, Cook, Shaper, 1994; Jin, Shah, Svopoda, 1995; Martikainen, Ma, Ja, 2007; Virtanen, Vahtera, Kivimäki, et al., 2005; Bambra, Elkemo, 2008; Catalano, 1991; Catalano, Dooley, Novaco, Wilson, Hough, 1993; Dooley, Catalano, Wilson, 1994; Kasl, Jones, 2002; Tausky, Piedmont, 1967).The relationship between increased unemployment and increases in mental disorder and cardiovascular illness was first observed at the macro (national) level using historical time-series analysis in United States, British and Scandinavian populations (Brenner, 1971, 1973, 1976, 1979a, 1979b, 1982, 1985, 1887). This work was further advanced under the sponsorship of the European Commission, where the interest was in the implications of unemployment and employment policies which could promote health (Brenner, 2000, 2002).

Several mechanisms are understood to operate in the relationship between unemployment and damage to health. One of these concerns the psychological stresses of loss and mental depression that separates the individual from his/her place in economic organizations – a position in which the content of work and social relations give meaning and direction to life. A second mechanism involves the loss of social relationships at work, which often constitute the major social networks and friendship patterns for many individuals as well as a definition of their position in society. Thirdly, and over the longer term, there is the problem of loss of income – which could be transitory or relatively permanent. In the case of transitory income loss, the period of unemployment may be relatively short, and new employment will restore previous wage and salary levels. More common, as with lengthy recessions and structural unemployment, is the inability of the unemployed individual to find reemployment in the same industry and/or at the same level of seniority (Podgursky, Swaim, 1987a, 1987b). In that case the unemployed person may find subsequent work in a different firm, or different industry altogether, in which the starting position will be at a very junior level. It may then take many years – if ever – f or the previously unemployed person to resume the level of income that prevailed prior to the unemployment. A far more serious situation occurs when the unemployed person becomes discouraged from seeking new work which would make use of the individual's skills and length of experience (Moore, Ranjan, 2005; Wilson, 1990; Blank, Blinde, 1985). In that case, the individual may leave the labor force entirely (or retire), and the likelihood of considerable loss of permanent income is relatively high (Uchitelle, Kleinfield, 1996). Finally, as

we have seen in the case of loss of income – indicating permanent loss of socioeconomic status – there is then a significantly increased risk of illness and early mortality.

4. *Public Expenditure on Health*

It is assumed by some health policy experts that health services expenditures, as a proportion of GDP, should bear some relation to the intensity, and the technological sophistication, of the health care utilized by a population (Musgrove, Zeramdini, Carrin, 2002; Comaish, Holdstock, 1983; Gerdtham, Jonsson, 1994; Gerdtham, Jonsson, 1992; Gerdtham, Jonsson, 1991; Gerdtham, Søgaard, Andersson, Jönsson, 1992; Gerdtham, 1993). The further assumption would be that the intensity and sophistication of health care should, in principle, be associated with decreased mortality rates (other things equal) (Getzen, 2000; Gerdtham, 1993; Murray, Govindaraj, Musgrove, 1994). There is an equally vocal group of health policy specialists who contend that health care expenditures need bear no relationship to improved health. This latter group feels that (1) much of the truly effective care occurs at the primary level which is not expensive and carries a minimum of expenditure of total health care in industrialized societies; (2) there has been little progress in improving mortality due to chronic common degenerative diseases – except perhaps in the case of cardiovascular disease; (3) health care expenditures heavily reflect administrative and management costs rather than actual performance of medicine and surgery; (4) the price of the same pharmaceuticals varies considerably among industrialized countries. Especially intense are critiques from United States health policy specialists who contend that, in the case of privately funded health care typically involving profit-making insurance companies, the cost of care bears little relation to its quality or intensity, since much of private health care expenditure is either medically unnecessary or occurs too late in life to significantly influence life expectancy (Bitton and Kahn, 2003; Woolhandler, Himmelstein, 1997; 2002a; 2002b).

There is some degree of consensus on the part of European and North American health economists that, for publicly funded care there are national criteria that roughly link personal health care procedures to their expenditures (Musgrove, Zeramdini, Karrin, 2002; Murray, Govindaraj, Musgrove, 1994). There is even greater consensus that publically funded health expenditure provides greater coverage to populations at higher morbidity levels – namely, the lowest socioeconomic groups (Ham, 1997; Hurst, 1991; Hurley, 2001) Publically financed expenditures also appear to provide some positive redistributive effects and less differential treatment (Doorslaer, Wagstaff, van der Burg, et al., 1999) There then seem to be some grounds for comparing health outcomes among industrialized countries, in relation to public expenditures on health care as a proportion of their real GDP (Clemente J, Marcuello C, Montañés A, Pueyo, F. 2004). Once this is done, controlling for other factors which measure economic growth and stability as well as lifestyle and environmental risk factors, we do observe a robust positive relationship between public health care expenditures and life expectancy. The more important observation, however, is that we can for the first time actually estimate the effect of health care expenditures on mortality reduction (Mackenbach, 1991). This conclusion takes on added plausibility when one recognizes that nearly all industrialized countries, apart from the United States, have the greatest proportion of their health care expenditure arising from public or not-for-profit sources.

5. *Quality of Work/Working Conditions*

Quality of working life or, more generally, "working conditions", have been traditionally known to influence health. In the oldest epidemiological literature, going back to Great Britain in the 1840's, one observes a powerful relationship between differential occupations, as identified by occupational skill level, and mortality rates at virtually all ages and for occupational, infectious and chronic diseases. Overall, the lower the occupational skill level of a worker, the higher the probability of premature mortality (Susser, Adelstein, 1975; Office of Population Censuses and Surveys Her Majesty's Stationary Office, 1986). Prior to the advent of

quantitative epidemiology, it was commonplace in clinical studies to demonstrate that the lowest-skilled workers (semi-skilled, unskilled) had the highest illness and mortality rates due to deficiencies of occupational safety. The principal problems in occupational safety and health are (1) physical, including toxic emissions, synthetic organic chemicals, carcinogens and ergonomic problems; and (2) psychological, especially including the lack of worker autonomy, stressful work (high demands), monotonous work and underemployment, insecure and unstable work and under-compensation in wages and benefits (Karasek, Theorell, 1990; Kuper, Singh-Manoux, Siegrist, Marmot, 2002; Lynch, Krause, Kaplan, Tuomilehto, Salonen, 1997; Lynch, Krause, Kaplan, Salonen, Salonen, 1997; Marmot, Bosma, Hemingway, Brunner, Stansfeld, 1997).

In cross-sectional analysis comparing countries, or historical time-series analysis, examining the experience of one country through time, it is difficult to identify precise markers of high or poor quality working conditions. However, in the industrialized country setting, it is well known that employment in the "shadow economy" generally offers working conditions of comparatively low quality. The shadow economy, in fact, involves illegal economic activity in that employers and employees do not pay government taxes (Frey, Weck, 1982; Eilat, Zinnes, 2002; Torgler, Schneider, 2009; Buhn, Karmann, Schneider, 2007; Hughes, 2000; Kyle, Warner, Dimitrov, et al., 2001; Chaudhuri, Schneider, Chattopadhyay, 2006; Boeri T, Garibaldi, 2005, 2007. This means, as well, that firms operating in the shadow economy are not subject to the payment of standard wages and do not offer the general benefits of insurance, including unemployment, health, disability, social welfare and pensions. Equally important, firms in the shadow economy often operate at long hours, with minimal health and safety rules and without time off (Centeno, Portes, 2006). In addition, workers in the shadow economy are subject to minimal autonomy and have higher stress levels due to the length of working hours, job instability and minimal wages and benefits.

Perhaps the single most important factor promoting the existence of firms in the shadow economy is either the lack of economic growth and development or the decline of the firm due to industrial restructuring, globalization or national recession (Boeri, Garibaldi, Salasco, Gobbi, 2002; Dell'Anno, Gomez-Antonio, Pardo, 2007). These phenomena reduce the capacity of the firm to pay taxes and to finance the modern technology needed to avoid problems of occupational safety and health, environmental toxins, ergonomically sophisticated work places, stable working conditions and minimally standard wages and benefits. Thus, for industrialized countries, we can conclude that firms in the shadow economy generally have considerably poorer working conditions, and that such conditions are risk factors for occupational safety and health problems, psychological stress and chronic degenerative diseases (Frey, Weck, Pommerehne, 1982; Chaudhuri, Schneider, Chattopadhyay, 2006; Boeri, Garibaldi, 2005, 2007; Bajada, Schneider, 2005).

It is important to bear in mind, however, that the shadow economy is most heavily represented in counties at comparatively low levels of economic development. Thus, one might argue that there is some redundancy in placing GDP per capita and the proportion of GDP that represents the shadow economy as independent variables in a single model by which one wishes to account for changes in life expectancy. In general, this would be true where one was comparing countries in a very large sample that included geographic areas dominated by relatively impoverished developing countries. However, this would not be the case where the comparison is among advanced industrialized societies at comparatively high per capita income levels. In the latter case, the presence of a substantial "shadow economy" tends to connote relatively underdeveloped sectors of the economy or an economy that is undergoing economic distress or extensive restructuring.

Self-employment is another important structural feature of working conditions. The self-employment rate is intended, in this analysis as a measure of entrepreneurship in small and medium-size enterprises (SME's). In traditional economic thinking entrepreneurship is the "fourth" factor of production and is essential to innovation and risk-taking in market economies. It is theoretically the source of continuous renewal of the technology and

structure of the economy and thus at least part of the basis for continuous long-term growth. The self-employment rate in SME's is also the source of a relatively high rate of job creation. And it is a traditional source of absorption into employment of persons unemployed as a result of recession, globalization and economic restructuring (including 'downsizing', 'offshoring', 'delocalization' and 'outsourcing'). Thus, self-employment is a traditional safety net for professional, managerial, skilled and unskilled workers who have lost employment.

There is also considerable theory that contrasts the SME with the large bureaucratic work organization, with its intensely hierarchical and impersonal employment structure. – i.e., the antithesis of "social capital" (Islam, Merlo, Kawachi, Lindstrom, Gerdtham, 2006; Kawachi, 1999; Lochner, Kawachi, Kennedy, 1999; Altschuler, Somkin, Adler, 2004). The hierarchical component itself is a source of enlarged socioeconomic (SES) inequalities within the firm's internal labor market. There is evidence that larger SES inequalities are an important factor in premature mortality (Kahn, Wise, Kennedy, Kawachi,. 2000; Lynch, Smith, Kaplan, House, 2000; Steptoe, Kunz-Ebrecht, Owen, et al., 2003). Scholars also point to the importance of the exceptional autonomy of the self-employed, obviously involving control (a risk factor in cardiovascular disease) over the extent of work demands as well as the pace, scheduling and intensity of work (Karasek and Theorell 1990). All of these factors lead to the inference that the rate of self-employment is important to economic growth and renewal, economic survival of the unemployed, and social capital – and for these reasons would provide important sources of societal coping with economic shocks.

6. *Lifestyles*

The primary variables we wish to investigate in relation to mortality concern factors intrinsic to the economy or to economic and social policy. However, in order to appropriately assess the impact of those factors on mortality, we also need to be sure that we have included in our model other risk factors to health that are well established influences on the mortality rate. Failure to include such variables, such as 'lifestyle' factors, may result in an over- or underestimate of the primary relation between the economic or social variables and health. One of these 'lifestyle' factors is high alcohol consumption per capita, which has a wide range of influences on illness including heart disease, stroke, kidney disease, specific malignancies, accidents, suicide, homicide and cirrhosis of the liver (Jernigan, Monteiro, Room, Saxena 2000; Room, Babor, Rehm, 2005; San Jose, Van De Mheen, Van Oers, Mackenbach, Garretsen, 1999). Alcohol consumption per capita can be seen to positively influence the age-adjusted mortality rates in our cross-sectional studies since we examine the effect of such a factor on mortality within the same year. Other high risk behaviors, such as tobacco (Bartecchi, MacKenzie, Schrier, 1994; Brownson, Eriksen, Davis, Warner, 1997), carbohydrate and animal fat consumption, are also known to influence mortality due to cardiovascular diseases and malignancies, but were not tested in the present model.

Economy, Social Policy and Mortality Among Industrialized Countries

Figure 1 shows age standardized mortality rates for 40 industrialized countries predicted by three types of variables: (1) economic factors, including GDP per employee, size of the "shadow" economy, the male unemployment rate and self-employment as a proportion of the labor force; (2) health expenditures funded by public sources as a proportion of the total GDP; and (3) alcohol consumption per capita, as representing a high-risk "lifestyle" factor harmful to health.

It can be seen that the mortality rate is distributed across the 40 countries largely in accordance with their income and wealth per capita as represented by the GDP per employee. As discussed above the GDP per capita is also strongly inversely related to the size of the shadow economy and the unemployment rate, while self-employment as a percentage of total employment is a measure of the economic health and density of entrepreneurship in the small and medium enterprises (SMEs).

The countries with the lowest mortality rates are among those with the highest GDP per capita, and the smallest shadow economy and unemployment rates. These relatively high income countries are those of Western Europe, North American and the Pacific. Western European countries include: Switzerland, Italy, Sweden, France, Spain, Norway, Austria, Greece, Germany, Finland, Netherlands, Ireland, United Kingdom, Belgium, Portugal and Denmark (in order of increasing mortality rates). The North American countries are Canada and the United States, while the Pacific countries are Japan, Australia, New Zealand and Korea.

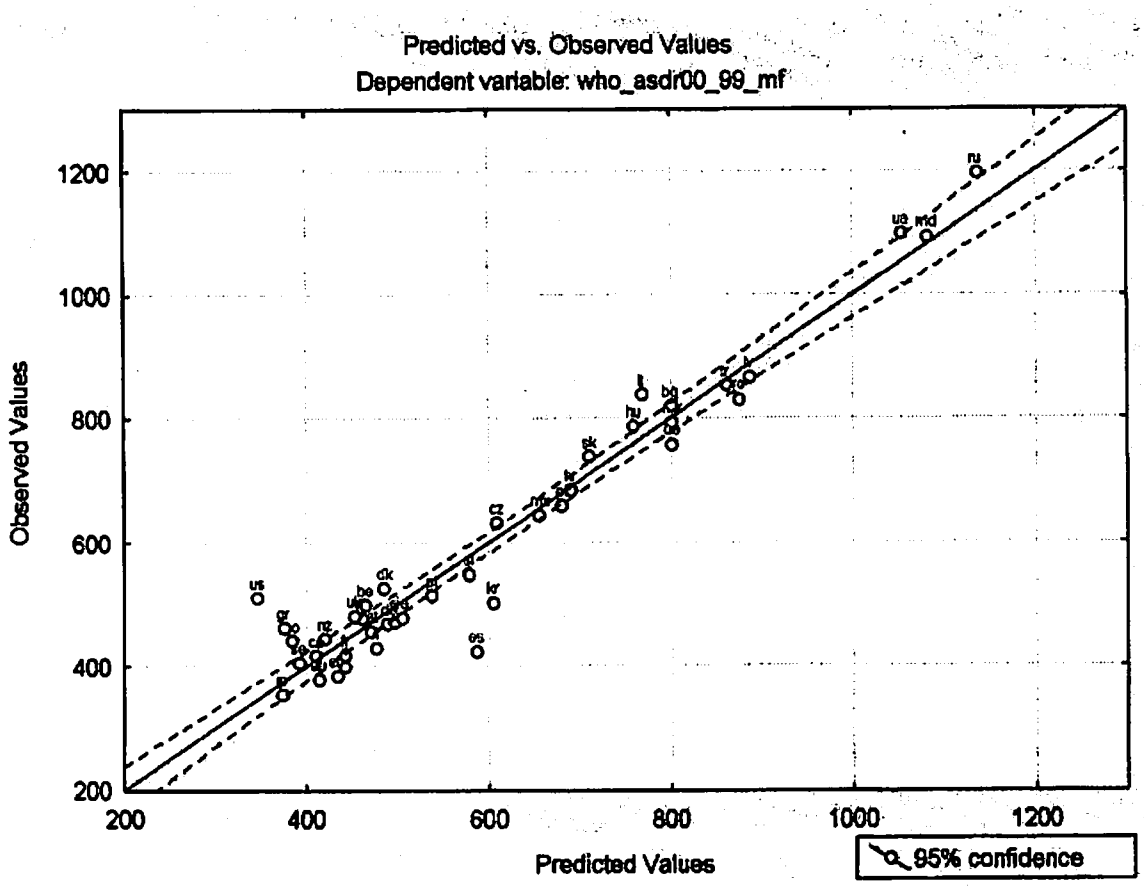
The middle income countries include those of Eastern Europe and, in the Western Hemisphere, Mexico. Eastern European countries in increasing order of mortality include: Slovenia, Czech Republic, Poland, Croatia, Slovakia, Estonia, Hungary, Macedonia, Bulgaria, Romania, Lithuania, Turkey and Latvia. Among the Eastern European countries it is interesting to observe relatively similar age-standardized mortality rates for societies that are geographically proximal and have similar GDP, income and wealth.

The third group of countries with very much higher mortality rates than those of Eastern Europe include: the countries of the Community of Independent States (CIS) including Moldova, Ukraine and the Russian Federation. As a group these countries have a lower GDP per employee than the average in Western or Eastern Europe and have the largest shadow economies among industrialized countries.

Figure 1
Relation of economic variables to age adjusted total mortality 40 European, CIS and OECD Countries, Year 2005

R= .97027845 R^2= .94144027 Adjusted R^2= .92863033
F(7,32)=73.493 p<.00000 Std.Error of estimate: 57.996

N=40	Beta	Std.Err.	B	Std.Err.	t(32)	p-level
Variables			422.1625	113.2121	3.72895	0.000745
GDP per employee (productivity)	-0.275164	0.084246	-0.0038	0.0012	-3.26819	0.002602
Shadow economy as proportion of GDP	0.442261	0.090652	7.8362	1.6062	4.87866	0.000028
Public health expenditure as proportion of GDP	-0.218179	0.062863	-29.1464	8.3978	-3.47072	0.001507
Self employed as proportion of total employment	-0.223634	0.045491	-4.5745	0.9305	-4.91595	0.000025
Unemployment rate, males	0.117708	0.050753	6.9823	3.0108	2.31922	0.026924
Alcohol consumption per capita	0.206031	0.046603	15.4188	3.4876	4.42102	0.000106
Consumer Price Index (Index Year 2000)	0.258445	0.050777	1.3576	0.2667	5.08979	0.000015

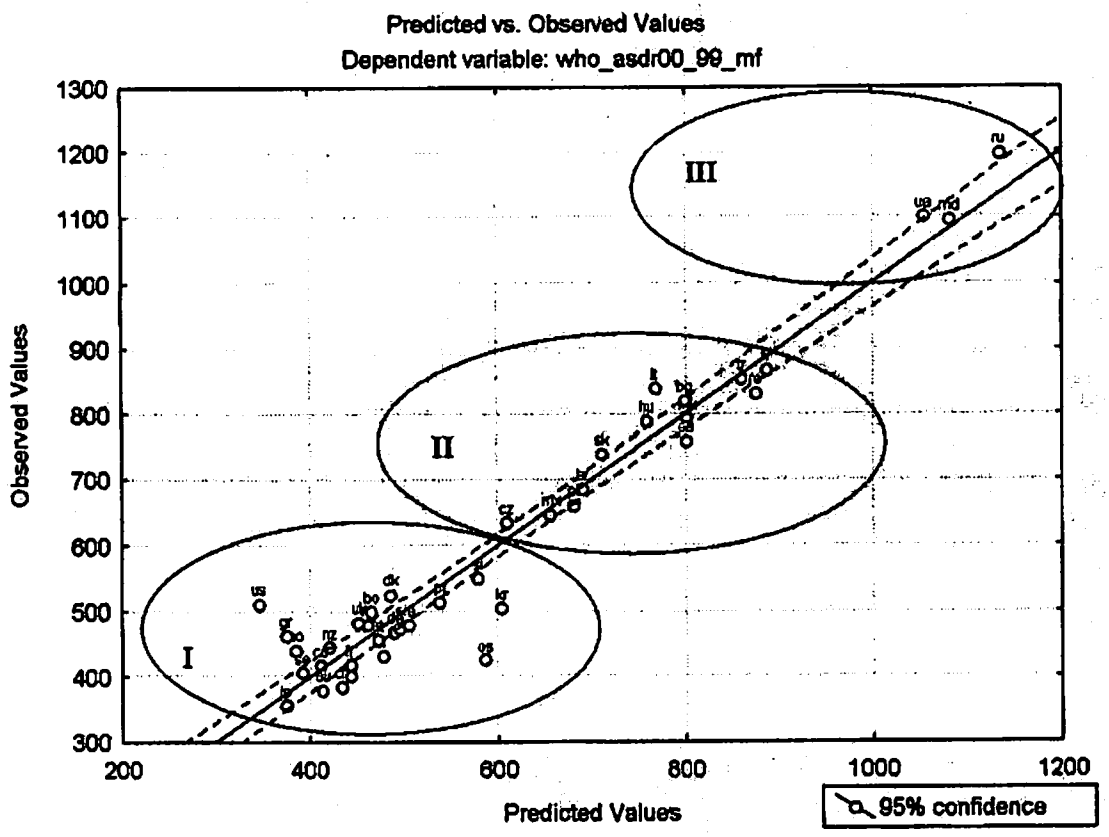


Importance of National Income to National Mortality Rates

The model above uses seven factors to account for the levels of age-standardized mortality rates among 40 industrialized countries. However a fundamental distinction among the 40 countries can be seen by distinguishing these countries by region and by average real GDP per employee. Figure 2 below repeats Figure 1 but groups the countries in terms of major regional classification and average GDP per employee. The first region enclosed in circle I, includes Western Europe, North America (except Mexico) and the highly industrialized Pacific countries of Australia, New Zealand, Japan and Korea with average GDP per employee of $ 45, 766. The second group of countries, in circle II, includes Eastern Europe and Mexico, with average GDP per employee of $20,292. The third group of countries, are within the Community of Independent States (CIS) and include Moldova, the Russia Federation and Ukraine, with average GDP per employee of $8,022.

Thus the basic differentiation among the group of countries is by geography and national income/wealth as well as productivity as measured by real GDP per employee. Within each of the three circles, one can see a relatively straight line representing the extent to which the entire set of seven predictors accounts for variation in age-standardized mortality rates among countries within the three regional groups. While the alignment of the three circles in a upward slope is essentially accounted for by GDP per employee, the variation of mortality among countries within the circles is largely accounted for by the remaining six variables in the model (shadow economy, self-employment, inflation rate, pubic health expenditures, male unemployment rate, and alcohol consumption per capita).

Figure 2
Relation of economic factors to age-standardized mortality rates: countries grouped according to average GDP per employee and geographic region.



	Region	Real GDP per Employee (Average USD)
I.	Western Europe North America Pacific	45,766
II.	Eastern Europe Mexico	20,292
III.	CIS Moldova Russia Federation Ukraine	8,022

Suicide, Economic Growth, Unemployment and Government Expenditure

Suicide is an "external" cause of death which is, at the same time, a principal measure of psychological depression, and is well known to respond to economic crises involving loss and insecurity.

In order to explain (statistically) variation in suicide rates by country, we can investigate several factors which deal with the issue of economic loss and unpredictability. At the individual level, perhaps the most common macroeconomic measure of loss is represented by the unemployment rate. It is also well known that the standard macroeconomic predictor of the unemployment rate is absolute decline and GDP per capita. We would therefore assume that countries with higher levels of GDP per capita, in addition to providing greater income and security, would also show lower suicide rates. Adding to GDP growth as protective against suicide, other major measures of economic development – such as the transformation of economies from domination by agriculture to that of manufacturing and, subsequently, to service industries – should likewise result in greater availability of material goods and services, and thus also reduce the tendency to suicide. However, a direct measure of consumption capacity, especially among the lower income population, is national consumption per capita of food and non-alcoholic beverages. We can compare this variable to expenditures on alcohol and tobacco, where a positive relation to suicide would be expected. Alcohol (and other psychoactive drugs) is known to influence psychological depression. Tobacco, in particular, influences cardiovascular disease and malignancies. The latter two chronic diseases themselves are risk factors to subsequent depression and suicide.

While the suicide rate is relatively strongly correlated (inversely) with real GDP per capita, it is subject to influences that go well beyond trends and fluctuations in national income and wealth (as measured by the GDP). Epidemiologically, suicide, as a marker for both psychological depression, and for poor mental health in general, is strongly associated in much of the literature over the last 40 years with unemployment experienced by individuals. The individual-level relationship between unemployment and psychological depression is so substantial that it is typically found in epidemiological studies to occur within a few months of the onset of unemployment. Nevertheless, the principal relationship between unemployment and the deterioration in mental health is typically observed over a period of several years. This considerable time lag is based on losses and damage to personal relationships (previously) at work, in the family and among friendship networks. Equally important are the longer term losses in income and economic position that frequently occur, especially among middle aged males, when they become reemployed – usually in a different firm, industry or occupation. In those situations reemployment will often involve starting at a considerably lower level of wage/ salary and job status; this will result in long-term losses in cumulative earnings, personnel benefits and retirement income.

In addition there is substantial evidence that the capacity to recover from crisis of cyclical or structural unemployment is, frequently, based on the extent of self-employment in small and medium enterprises (SMEs). Therefore one would assume that countries with a more viable pattern of self-employment would be (1) more highly productive of new employment, (2) able to generate a greater proportion of new cases of self-employment, and (3) able to absorb a larger number of the unemployed. Thus, the extent of self-employment like GDP per capita, should predict lower rates of suicide.

At the same time, one must consider the extent to which the society provides social protection to individuals in periods of economic crisis. One would expect that countries which traditionally offer stronger social safety nets and, in general, more extensive social welfare systems, would be able to create societies with more stable incomes and employment patterns, as well as higher levels of training for those who have lost jobs or need to restructure their careers, as well as unemployment, disability, health insurance and retirement income. A somewhat crude measure of the tendency of a society to provide such a social protection can be found in the magnitude of government consumption (as compared to private consumption) per capita. With such an

indicator, one would assume that greater levels of social protection and economic security provided by government would minimize economic loss due to economic shocks and structural changes; this would in turn predict lower suicide rates.

Social protection is often thought of, by non-specialists, as essentially referring to insurance systems (especially unemployment, disability and frailty, impoverishment and pensions). However, a major aspect of social protection, during working life and after it, involves expenditure on health care. This needs to be distinguished from government expenditure on overall collective consumption, which will also include education, transportation, national defense etc. We are thus especially interested in the proportion of total government expenditure that represents specific expenditure on health. One may say that this is a measure of the extent to which "governments", or the national polity, place a high priority on population health as a key element of social protection.

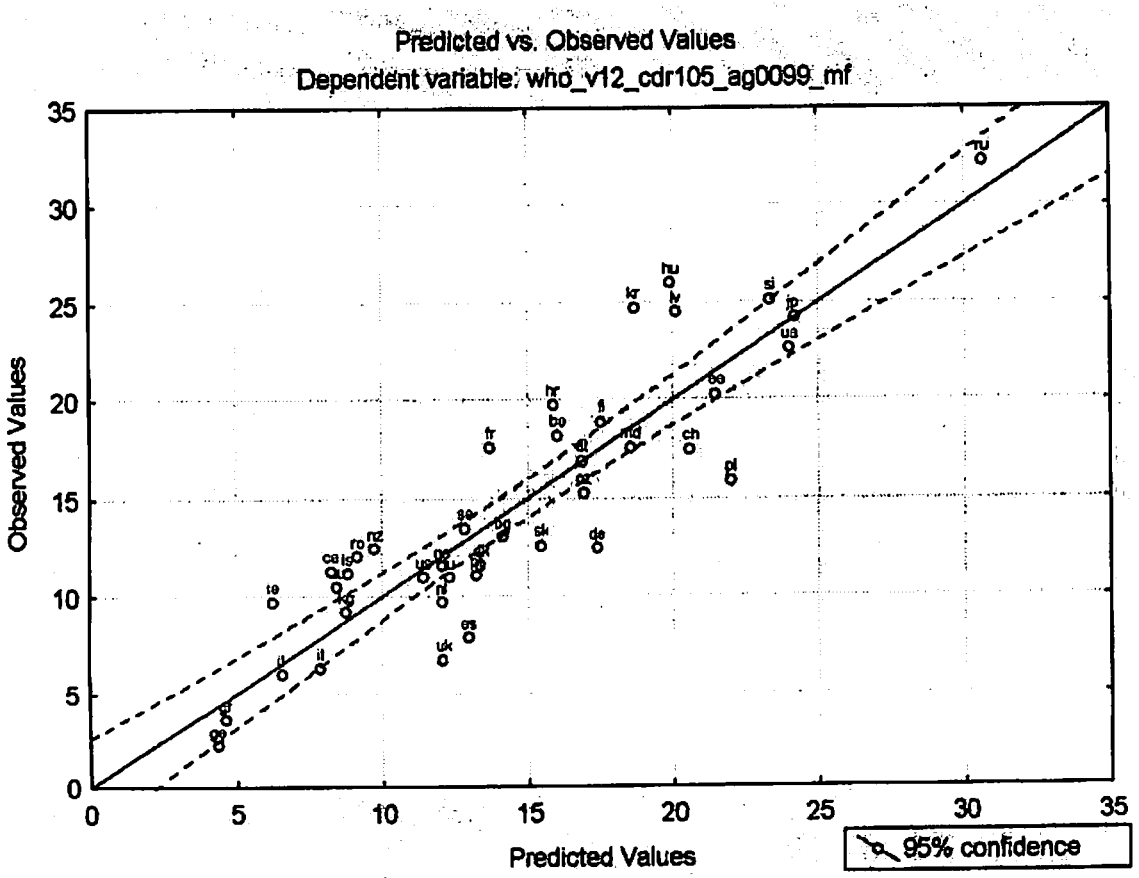
Another position entirely, based on the sociological writings on suicide of Emile Durkheim (1858-1917) puts forward the thesis of social integration as a principal factor militating against high suicide rates. One of Durkheim's key arguments is that a cosmopolitan lifestyle, especially related to urbanization, and incorporating many cultural, religious, political frames of reference and relatively high levels of education, make it difficult for the individual to adopt a coherent system of rational understanding and belief. Such a situation leads to what Durkheim referred to as "anomie" in which the individual becomes relatively "rudderless" and intellectually directionless. This situation, on Durkheim's view, is a primary casual factor in suicide linked to the relative absence of social integration. We could therefore include a measure of such urban cosmopolitanism which would refer to the proportion of the national population that lives in a situation of urban agglomeration. But since Europe is very extensively urbanized, a more penetrating measure of residential agglomeration is the extent of population density among the rural population. Along similar lines of argument, it is also assumed that the divorce rate will predict higher suicide rates.

The above potential causal factors lead us to a model of suicide rate variation among industrialized countries. The model consists (1) economic factors [GDP per capita (inverse), male self-employment (inverse), the unemployment rate (positive)], (2) government expenditures on health care as a proportion of total government expenditures (inverse), (3) social integration [rural population density (positive), divorce rate (positive)], (4) 'life-style' risk factors to suicide directly (e.g., alcohol consumption) and to chronic diseases which themselves are risks to suicide (e.g., tobacco). Figure 3 below presents this model. Nearly all variables are statistically significant and the model accounts for 74% of suicide rate variation among industrialized countries.

Figure 3
Regression summary for total suicide mortality rate (CDR per 100,000 of population)
40 European, OECD and CIS countries, year 2005

R= .89499048 R^2= .80100797 Adjusted R^2= .74131036
F(9,30)=13.418 p<.00000 Std.Error of estimate: 3.4128

	Beta	Std.Err.	B	Std.Err.	t(30)	p-level
Intercept			35.73286	8.755259	4.0813	0.000305
GDP per capita, in const. 1990 Geary Khamis $ (Groningen Growth and Development Centre, Total Economy Database, June 2009)	-0.381077	0.189844	-0.00031	0.000153	-2.00732	0.053798
Gen. gover. exp. on health - % of total gov. exp. (WHO Statistical Information System)	-0.382798	0.133949	-0.78077	0.273208	-2.85779	0.007682
Exp. on food and non-alc. bever. - % of total GDP (World Bank, Global PPP Indicators 2005)	-0.436559	0.197454	-0.4198	0.189873	-2.21094	0.034806
Exp. on alcohol. bever. and tob- % of total GDP (World Bank, Global PPP Indicators 2005)	0.239833	0.139155	1.04157	0.604341	1.72349	0.095094
Gover. exp. on collect. consump. - % of total GDP (World Bank, Global PPP Indicators 2005)	-0.349901	0.095794	-1.46623	0.401415	-3.65265	0.000982
Male self-employment - % of tot. male empl-t (ILO, Key Indicators of the Labor Market)	-0.381986	0.139958	-0.28356	0.103897	-2.72929	0.010514
Rural pop. density (per sq. km of arable land (World Bank – World Development Indicators)	0.475674	0.089228	0.01656	0.003106	5.33099	0.000009
Divorces per 1,000 population (United Nations, Statistic Division)	0.437951	0.110286	3.42576	0.862683	3.97105	0.000413
Unemployment rate - % of registered labor force (ILO, Key Indicators of the Labor Market)	0.319163	0.105784	0.48069	0.159319	3.01713	0.005162



Similar policy issues of national health expenditure as a proportion of GDP, and the unemployment rate can be examined over historical time to observe their implications for population health. In this case a model is required that will include these two variables as predictors of mortality in addition to other factors which help to predict the mortality rate. We begin with the observation that models can be developed which can account for mortality changes over time in industrialized countries, but tend to require at least the two variables of real GDP per capita and the unemployment rate. These models have been published for the United States, the United Kingdom and several other countries (Brenner, 1976, 1985, 1979, 1987). In addition there is cross-national and times series evidence for industrialized countries showing that increased health care expenditure as a proportion of the GDP greatly depends on GDP growth (Hitiris, Posnett, 1992). Therefore, on the grounds of substantial correlation among these predictors (i.e., multi-collinearity), it is unwise to include both GDP per capita and health care expenditures in the same predictive model.

At the same time it would be convenient for policy reasons to include in the model a variable that in some significant way dealt with the fact that the material wellbeing of the non-working segments of the population (youth and elderly) depend on the income and working capacity of the working population. One such variable is a purely demographic version of the 'dependency ratio' (Harwood, Sayer, Hirschfeld, 2004; Cohen, 2003; Crown, 1985), which measures the extent to which the non-working population depends for its income and social benefits on the working population. In this case, the economically dependent population involves the nonworking population under age 15 and (to a very large extent) over 65. The resulting variable of the ratio of the population 15-64 to the population under and over 65 portrays the burden of the working population to support itself as well as the nonworking population. In the micro view, one can see that a family with several offspring and elderly to care for would experience a considerable burden of stress in the need to earn a significant income. Similarly at the national level, a society with a substantial and increasing aging population (typical of wealthy industrialized societies), and/or with a substantial birth rate (characterizing less developed countries) would experience some constraints in investing heavily in health care, pensions, occupational health and safety, education and technological development (Lee, 2002; Masson & Tryon, 1990; Razin, Sadka, Swagel, 2002). Thus a relatively high dependency ratio might theoretically tend to decrease the beneficial health effects and sustainability of economic growth. However, the relation between population aging and economic growth appears to depend on economic and social policies (Fougere & Merette, 1999; Borsch-Supan, 1997. And it could sharply decrease the economic wellbeing of the older population under conditions of economic recession.

It would also be useful in a model like this to include, at least as a control, an important 'lifestyle' variable that represents an important risk to health – in this case, tobacco consumption per capita.

Finally, observation of the United States age-standardized mortally rate (ASDR) over 1970-2005 shows a sharp and continuous decline since 1975. This accelerated mortality decline has been attributed in the United States to the influence of a variety of new cardiovascular drugs which are understood to have been important in the sharp mortality declines in coronary heart disease (Ford, Aljani, Croft, 2007). Since we have limited ability to directly measure such an effect, we instead use a dummy variable to represent the downward shift in mortality since 1975. The same procedure is used to allow us a precisely comparable model for analysis of the United Kingdom ASDR over the same time span.

The models for the U.S. and the U.K. ASDR over 1970-2005 thus includes the following independent variables: (1) health expenditures as proportion of GDP in constant dollars; (2) the unemployment rate as a proportion of the labor force; (3) population 15-64 as a percent of the total population; (5) number of daily smokers in proportion of the population over 15; and (6) a dummy variable for 1975-2005.

The U.S. and U.K. provide acceptable times-series models, with nearly all variables significant (and minimal positive autocorrelation of residuals). For both the U.S. and the U.K. health care expenditures as a proportion of GDP is a highly significant inverse explanatory factor for ASDR. However, the coefficient for U.K. expenditures is higher (minus 30.75) than that for the U.S. (minus 21.70). This would indicate that U.K. expenditures "produce" a greater reduction in mortality than is true for U.S. expenditures. For the unemployment rate, the U.K. impact (8.265) is somewhat larger than that of the U.S. (7.213). This may reflect the higher and more rapidly changing unemployment rates in the U.K., compared to the U.S., during 1970-2005.

Figure 4
Regression Summary for total age adjusted mortality in total population (ASDR per 100,000)
Key predictors: health expenditures as percent of GDP, unemployment rate as percent of total labor force
UK, years 1970-2005

R= .99424896 R^2= .98853099 Adjusted R^2= .98661948
F(5,30)=517.15 p<0.0000 Durbin-Watson d=1.77

N=36	Beta	Std.Err.	B	Std.Err.	t(30)	p-level
Intercept			1588.874	455.8486	3.48553	0.001535
Total health expend as prop of GDP (OECD Database)	-0.285069	0.060696	-41.793	8.8985	-4.69664	0.000055
Total unemploy as prop of labor force (OECD Database)	0.150331	0.033483	7.158	1.5942	4.48978	0.000098
Pop 15-64 as proportion of total pop (World Bank Database)	-0.123770	0.053324	-15.134	6.5202	-2.32109	0.027263
Tobacco consumption in grams per adult (WHO Database)	0.677833	0.072545	77.493	8.2937	9.34360	0.000000
Dummy 1975-2005	0.014094	0.032453	5.603	12.9017	0.43429	0.667184

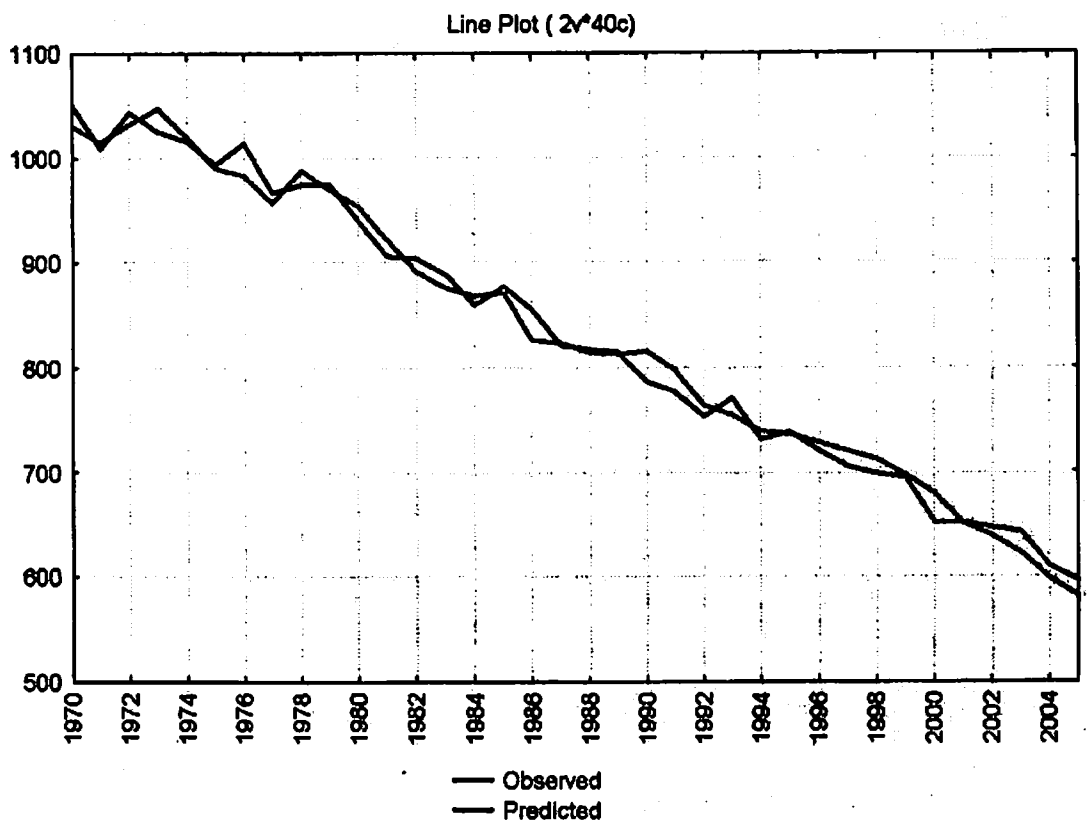


Figure 5
Regression Summary for total age adjusted mortality in total population (ASDR per 100,000)
Key predictors: health expenditures as percent of GDP, unemployment rate as percent of total labor force
US, years 1970-2005

R= .99673604 R^2= .99348273 Adjusted R^2= .99239652
F(5,30)=914.63 p<0.0000 Durbin-Watson d=1.88

N=36	Beta	Std.Err.	B	Std.Err.	t(30)	p-level
Intercept			1588.874	455.8486	3.48553	0.001535
Total health expend as prop of GDP (OECD Database)	-0.285069	0.060696	-41.793	8.8985	-4.69664	0.000055
Total unemploy as prop of labor force (OECD Database)	0.150331	0.033483	7.158	1.5942	4.48978	0.000098
Pop 15-64 as proportion of total pop (World Bank Database)	-0.123770	0.053324	-15.134	6.5202	-2.32109	0.027263
Tobacco consumption in grams per adult (WHO Database)	0.677833	0.072545	77.493	8.2937	9.34360	0.000000
Dummy 1975-2005	0.014094	0.032453	5.603	12.9017	0.43429	0.667184

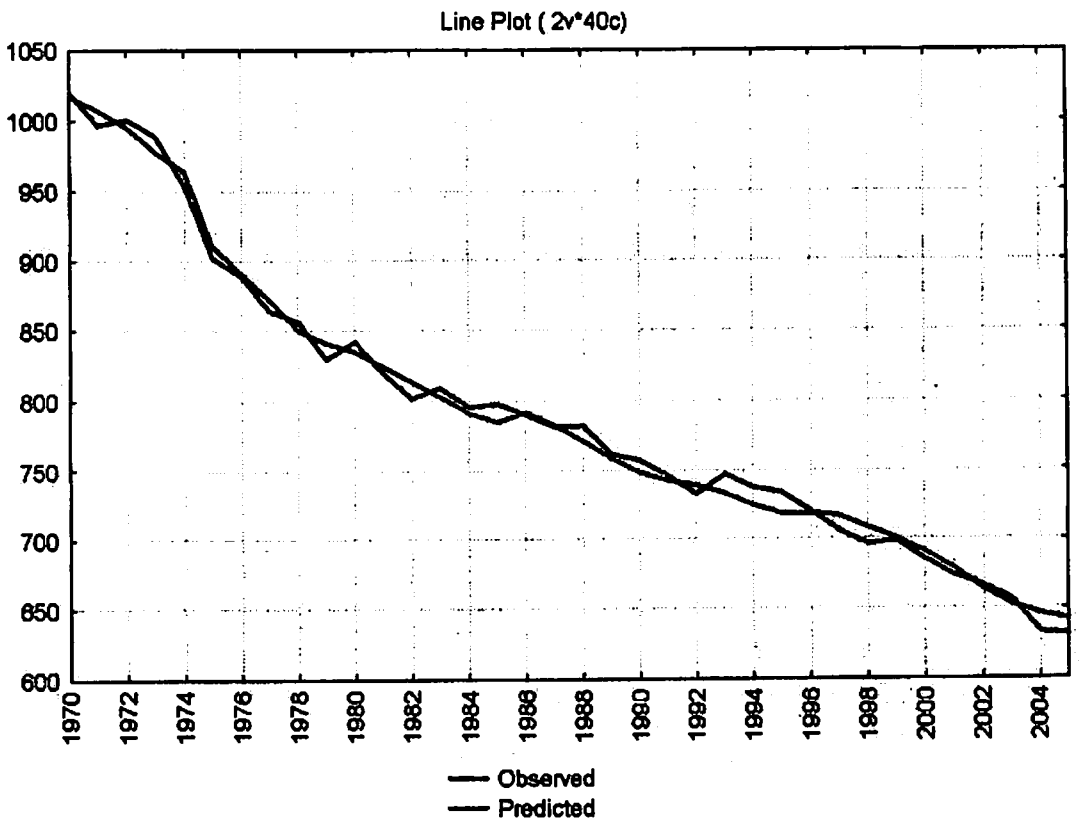


Figure 6

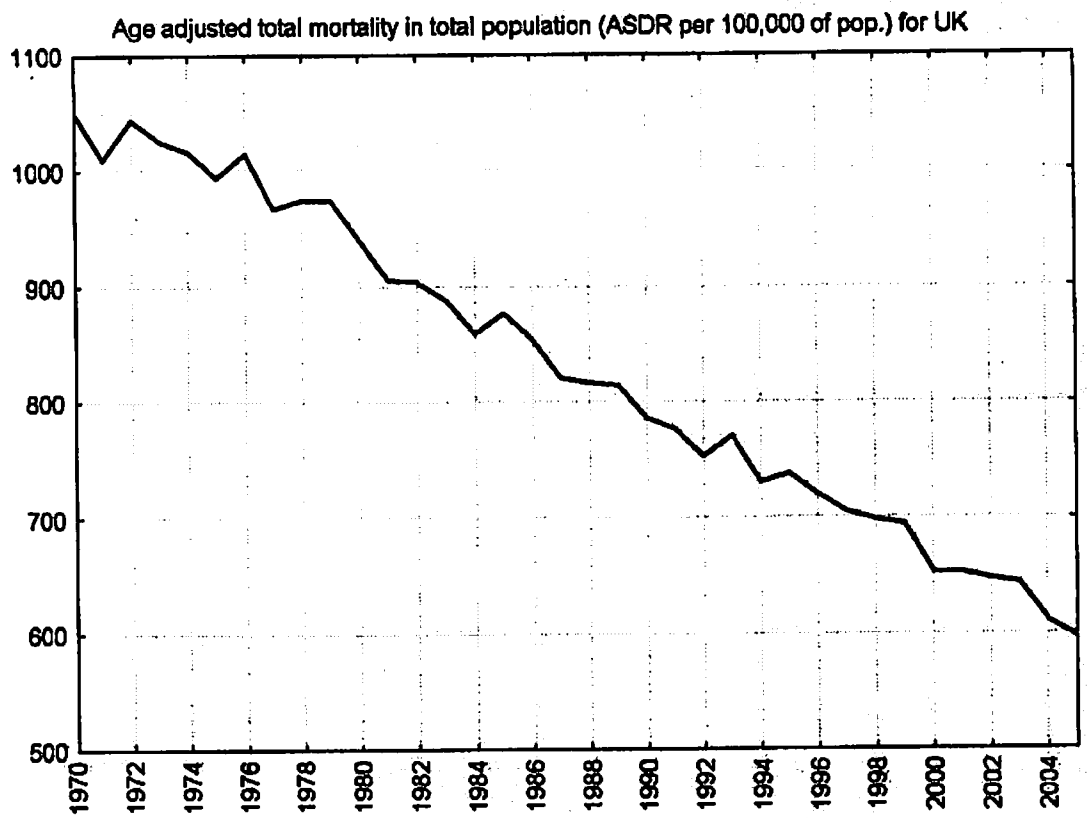

Age adjusted total mortality in total population (ASDR per 100,000 of pop.) for UK
1100
1000
900
800
700
600
500
1970
1972
1974
1976
1978
1980
1982
1984
1986
1988
1990
1992
1994
1996
1998
2000
2002
2004

Figure 7

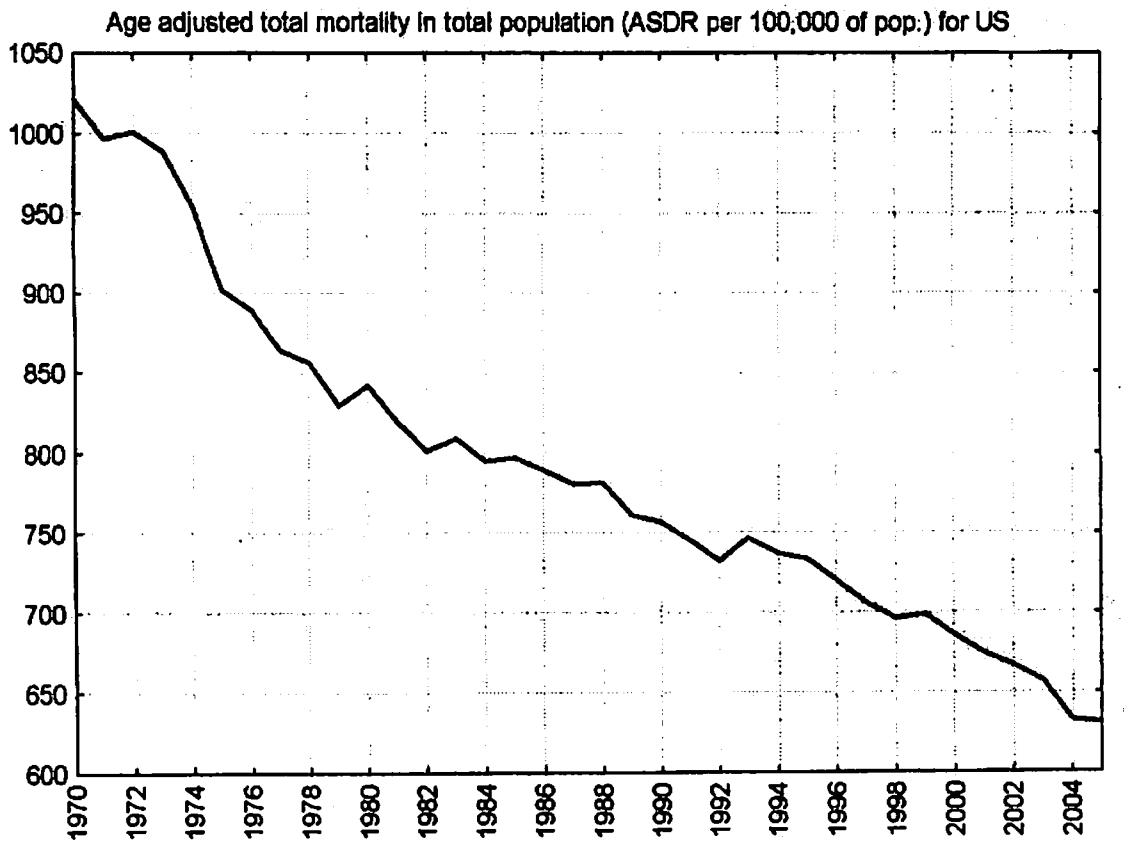

Age adjusted total mortality in total population (ASDR per 100,000 of pop.) for US
1050
1000
950
900
850
800
750
700
650
600
1970
1972
1974
1976
1978
1980
1982
1984
1986
1988
1990
1992
1994
1996
1998
2000
2002
2004

Figure 8

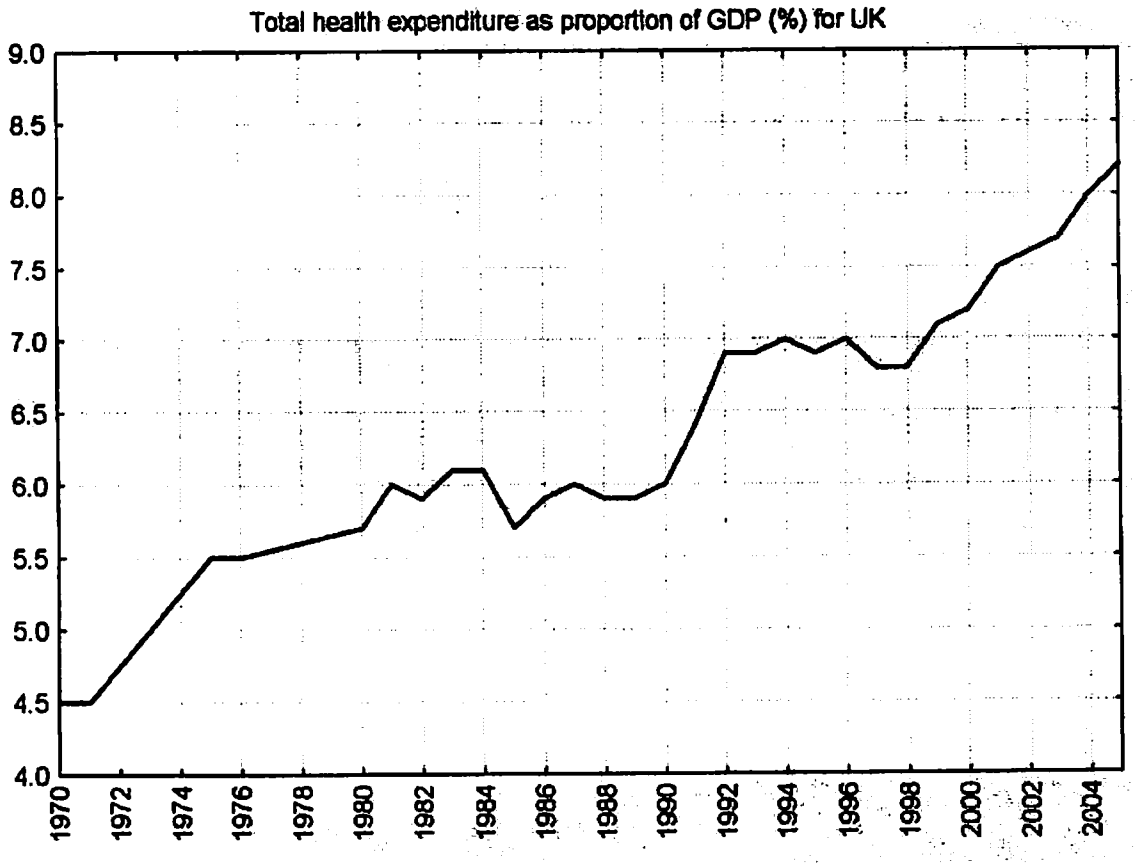

Total health expenditure as proportion of GDP (%) for UK
9.0
8.5
8.0
7.5
7.0
6.5
6.0
5.5
5.0
4.5
4.0
1970
1972
1974
1976
1978
1980
1982
1984
1986
1988
1990
1992
1994
1996
1998
2000
2002
2004

Figure 9

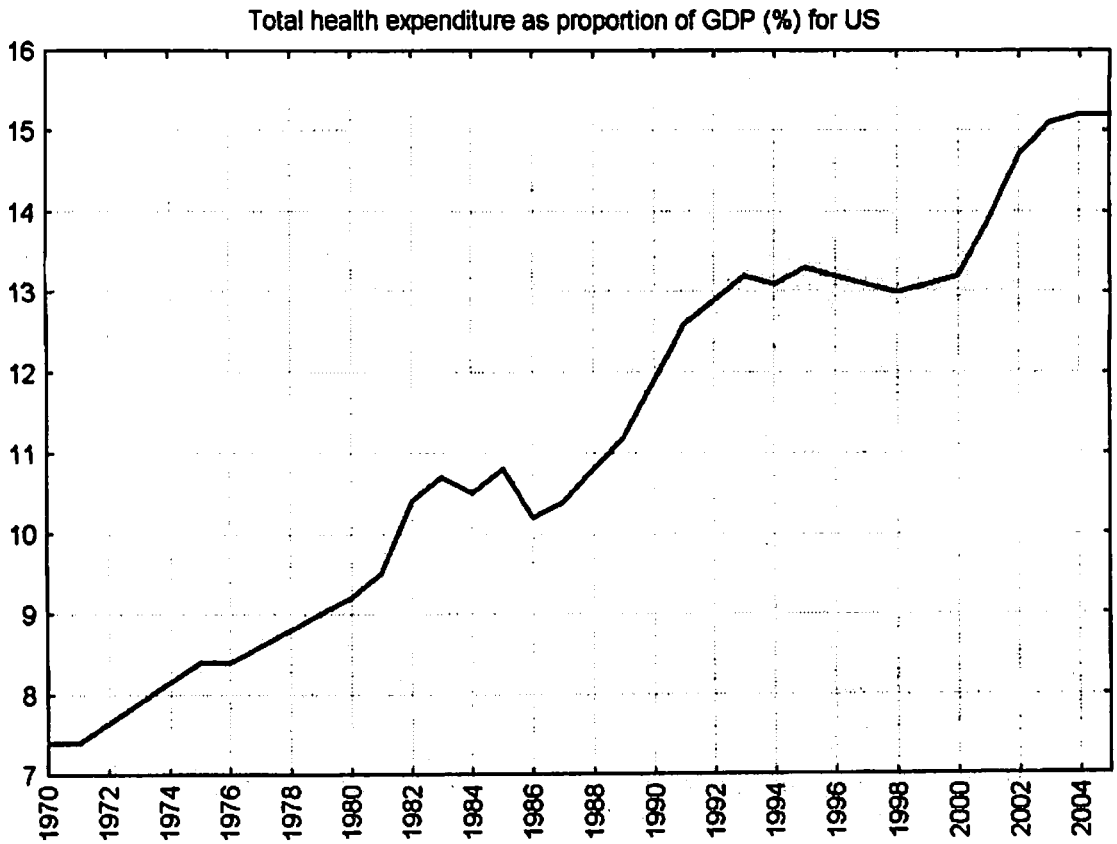

Total health expenditure as proportion of GDP (%) for US
16
15
14
13
12
11
10
9
8
7
1970
1972
1974
1976
1978
1980
1982
1984
1986
1988
1990
1992
1994
1996
1998
2000
2002
2004

Figure 10

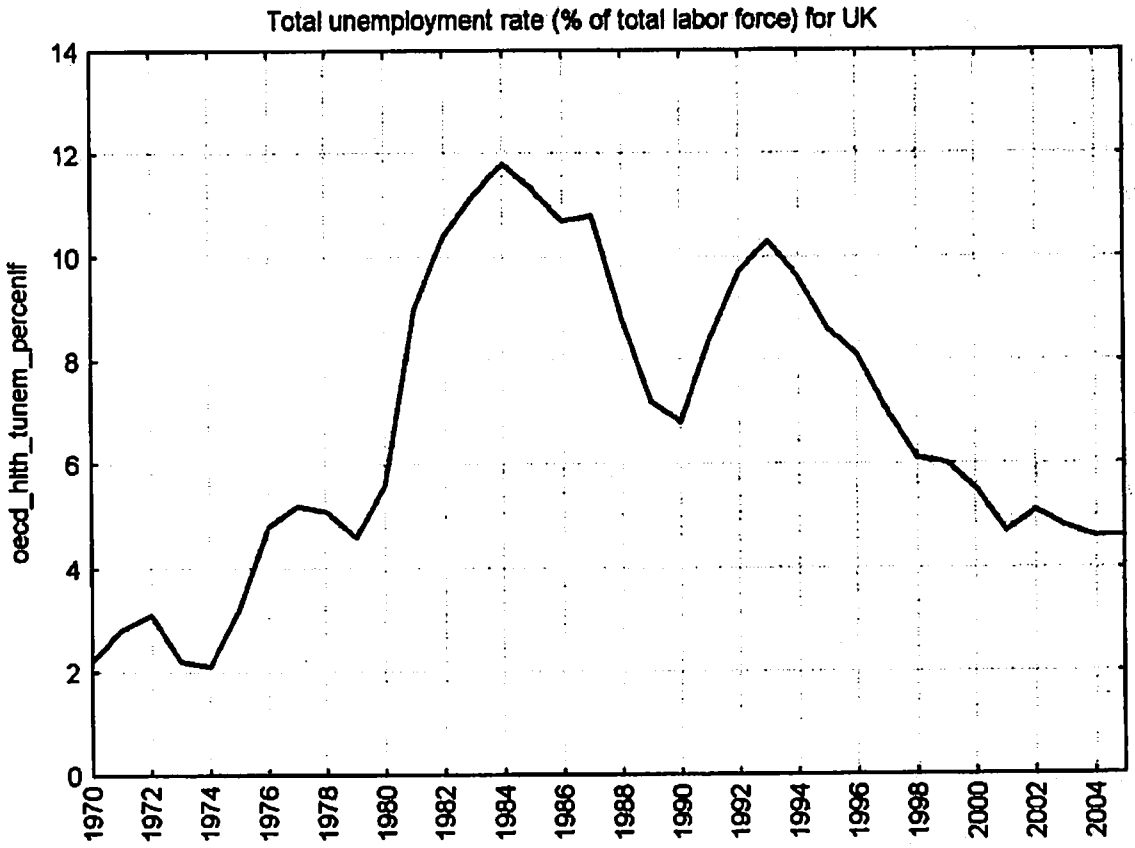

Total unemployment rate (% of total labor force) for UK
oecd_hlth_tunem_percenlf
0
2
4
6
8
10
12
14
1970
1972
1974
1976
1978
1980
1982
1984
1986
1988
1990
1992
1994
1996
1998
2000
2002
2004

Figure 11

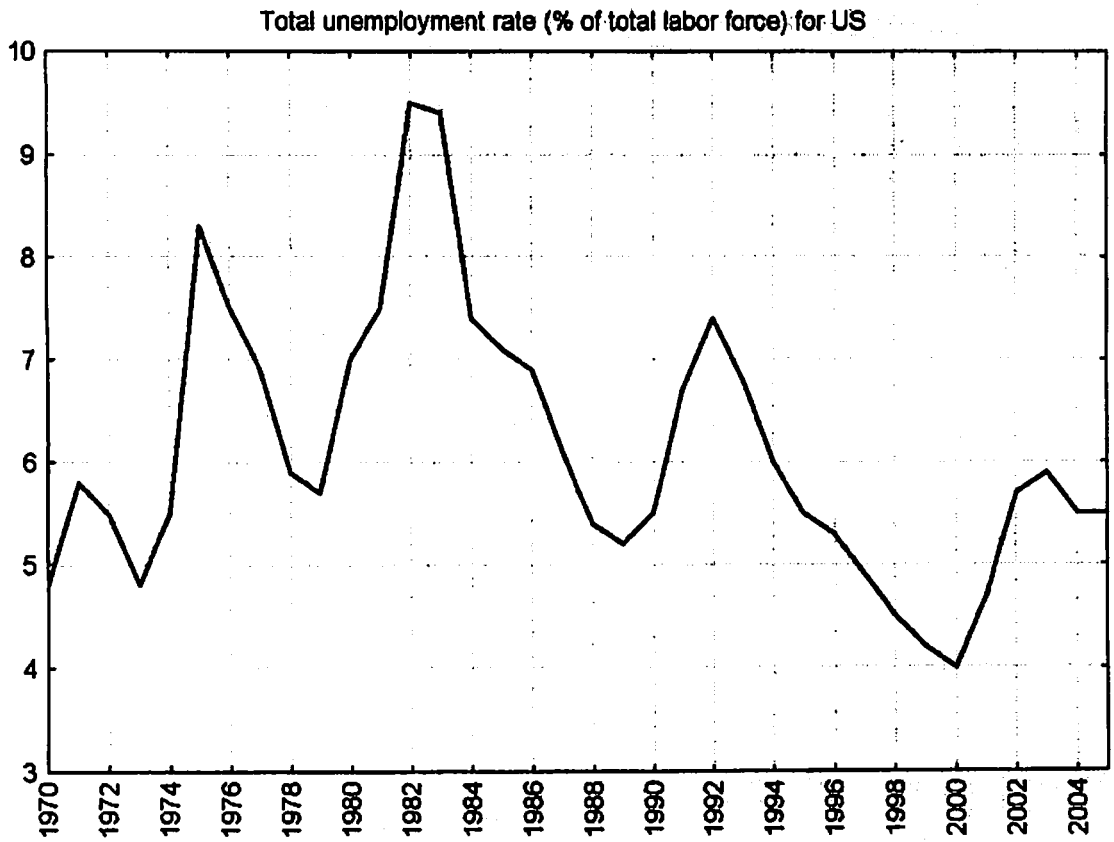

Total unemployment rate (% of total labor force) for US
10
9
8
7
6
5
4
3
1970
1972
1974
1976
1978
1980
1982
1984
1986
1988
1990
1992
1994
1996
1998
2000
2002
2004

Figure 12

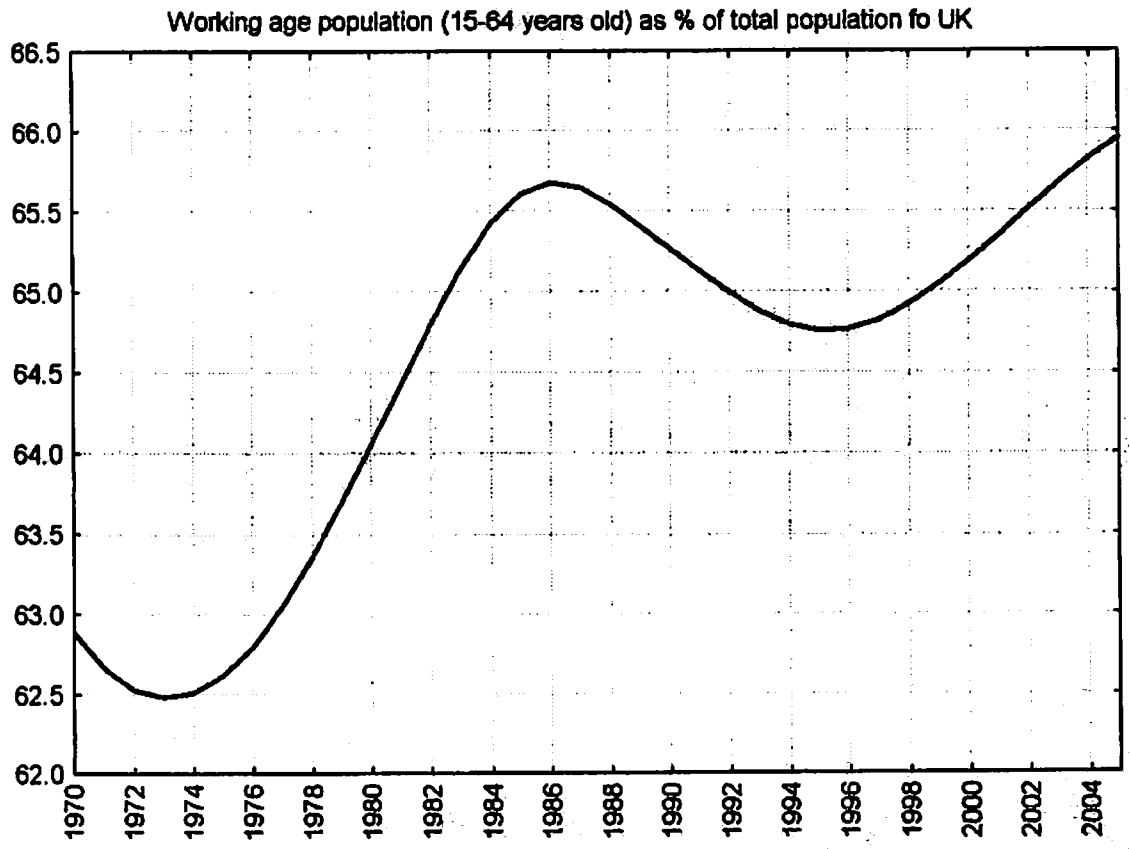

Working age population (15-64 years old) as % of total population fo UK
66.5
66.0
65.5
65.0
64.5
64.0
63.5
63.0
62.5
62.0
1970
1972
1974
1976
1978
1980
1982
1984
1986
1988
1990
1992
1994
1996
1998
2000
2002
2004

Figure 13

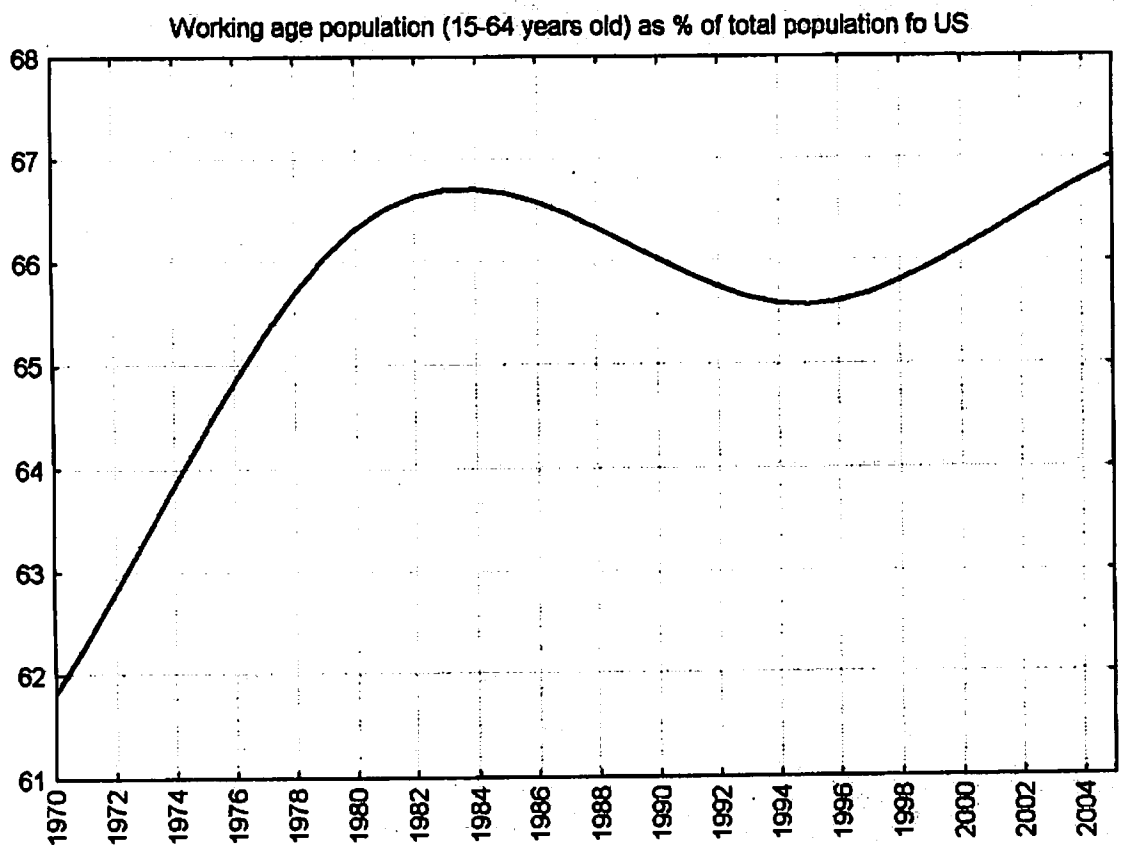

Working age population (15-64 years old) as % of total population fo US
68
67
66
65
64
63
62
61
1970
1972
1974
1976
1978
1980
1982
1984
1986
1988
1990
1992
1994
1996
1998
2000
2002
2004

Figure 14

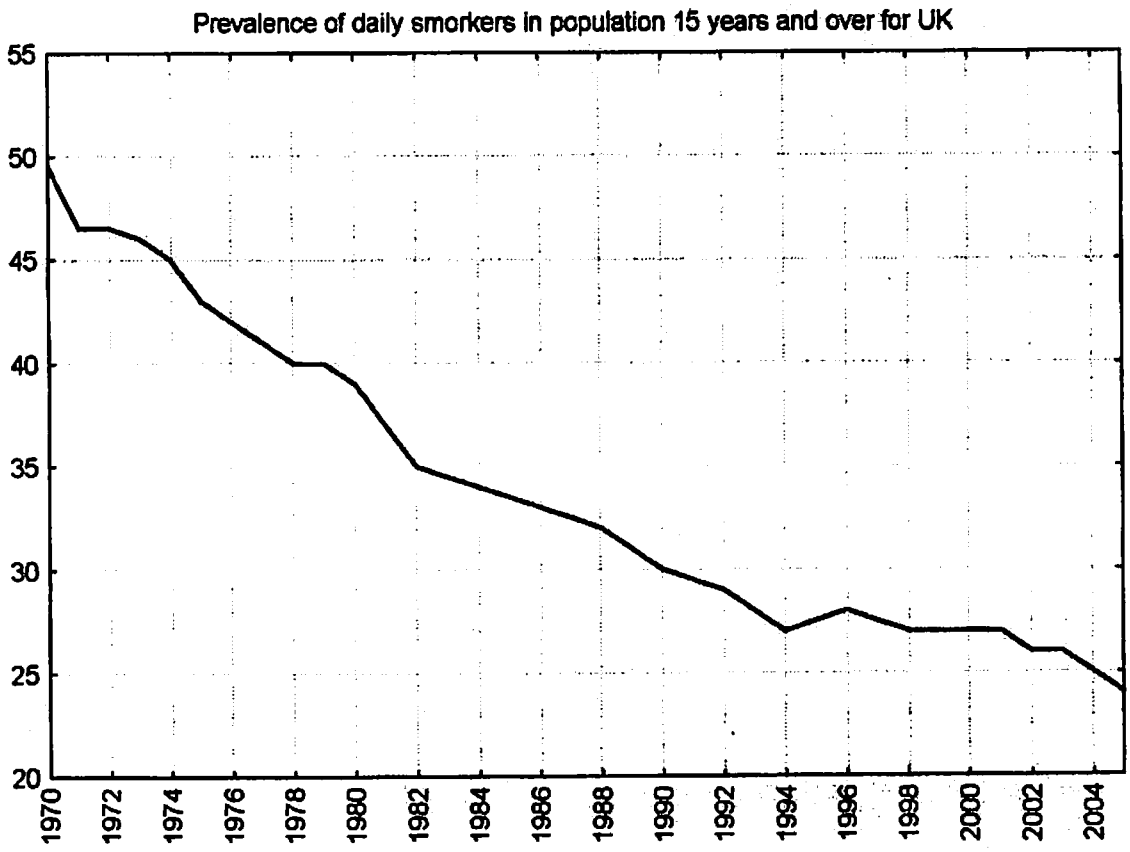
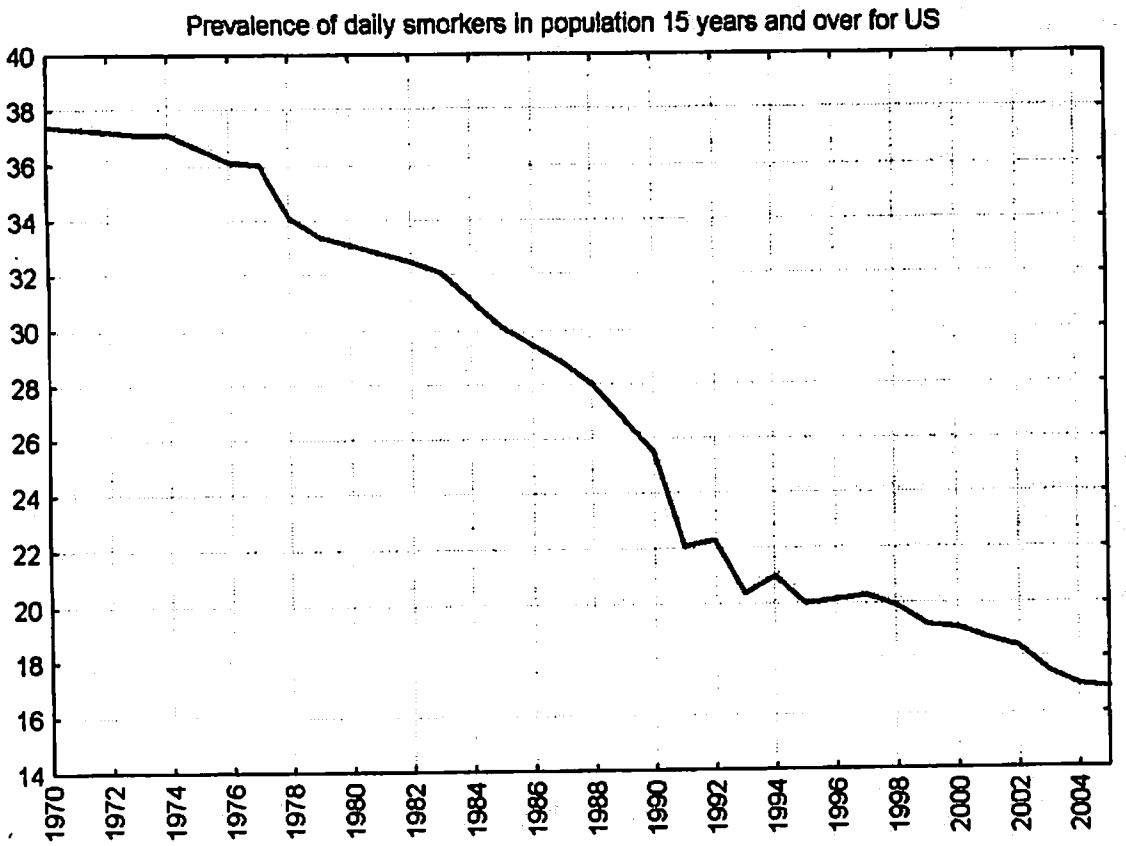

Prevalence of daily smorkers in population 15 years and over for UK
55
50
45
40
35
30
25
20
1970
1972
1974
1976
1978
1980
1982
1984
1986
1988
1990
1992
1994
1996
1998
2000
2002
2004

Figure 15


Prevalence of daily smorkers in population 15 years and over for US
40
38
36
34
32
30
28
26
24
22
20
18
16
14
1970
1972
1974
1976
1978
1980
1982
1984
1986
1988
1990
1992
1994
1996
1998
2000
2002
2004

Projected impact of health care expenditure, and unemployment, on mortality

In Table 1 below the impact of public health expenditure (as percentage of GDP) is estimated, in relation to changes in mortality under scenarios where the public health expenditure is reduced by 15%, and by 25%. Similarly the impact of decreases in unemployment (male) is increased by 50% and by 100%. A third set of situations is estimated whereby both public health expenditure is decreased and unemployment rates are also increased.

In the scenario where only public health expenditure declines by 15% and 25% the equivalent total change of deaths in the original 40-country sample is approximately 341,000 and 569,000. Using the same model for the 40 countries and making estimates only for 24 of the 27 E.U. countries (without Cyprus, Luxemburg, Malta) is approximately 143,000 and 238,000.

Under the scenario where only unemployment (male) increases by 50%, and by 100%, the equivalent number of deaths in the 40-country sample are approximately 257, 000 and 514,000. For the 24 E.U. countries (based on the 40-country model) the estimates of deaths are approximately 93,000 and 186,000.

Should both public health expenditures (percentage of GDP) decline by 15% and unemployment (male) increases by 50%, the estimated number of deaths in the 40 countries are approximately 599,000 and 236,000 for the 24 E.U. countries. If public health expenditures decrease by 15% and unemployment (male) increases by 100%, mortality increases in the 40 countries by 856,000 and in the 24 E.U. countries by 329,000. if public health expenditures decline by 25% and unemployment (male) increases by 50% total deaths in the 40 countries increase by approximate 826,000 and , in the 24 E.U. countries 331,000. If public health expenditures decline by 25% and unemployment (male) increases 100% then total increases in deaths for the 40 countries are approximately 1,083,600, and in the E.U. 24 countries by 424,000. (All of the above estimates assume that the changes in public health expenditures in unemployment, and the changes in mortality occur within a single year.)

Table 1

Impact of variations of public health expenditures and unemployment on mortality

Public Health Expenditure (as percentage of GDP)		Unemployment (Male)		Total Change of Deaths (40 countries)	Total Change of Deaths (only EU countries N=24)*
Factor	% decrease	Factor	% increase	(absolute numbers)	(absolute numbers)
1.00	0%	1.50	50%	257,208	92,885
1.00	0%	2.00	100%	514,416	185,770
0.85	15%	1.00	0%	341,514	142,852
0.75	25%	1.00	0%	569,190	238,086
0.85	15%	1.50	50%	598,722	235,737
0.85	15%	2.00	100%	855,930	328,621
0.75	25%	1.50	50%	826,398	330,971
0.75	25%	2.00	100%	1,083,606	423,856

***EU 27 without Cypress, Luxembourg, Malta**

Comment:

(1) A 25% decrease of public health expenditure as percentage of GDP means that e.g. when public health expenditure as percentage of GDP is 10% it will be reduced to 7.5%

(2) A 50% increase of the unemployment rate means that e.g. when the unemployment rate is at 10% it will go up to 15%

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Society and Health

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7

Political Economy and Health

M. HARVEY BRENNER

The political economy—the national economy, in interaction with government policies—influences a nation's health through the mechanisms of production, distribution, and consumption. The main links between the economy and health are economic growth and instability (especially recession), economic inequality (including inequality due to structural changes), production processes and consumption of goods that are harmful to health, high-risk social-interaction patterns disproportionately prevalent in the lower socioeconomic strata, and health-care utilization. This analysis will emphasize three elements of political economy crucial to health: (1) the national economic context of growth, business cycles, and social stratification, (2) the processes of production, distribution, and consumption, and (3) government policies.

Economic growth—the creation of wealth and elevation of the national living standard—has overwhelmingly beneficial effects on health, especially in reduction in acute infectious diseases and occupational injuries as causes of death. The main material benefits of economic growth are better nutrition; better housing; more advanced sanitary, industrial, and traffic engineering; and better health care. The psychological benefits include security from catastrophic economic loss and career damage; decline in the prevalence of physically exhausting and hazardous work; improved quality of life for the ill, disabled, and frail; and a sense of control, confidence, and hope for the future.

Also an outcome of economic growth, however, is the rise of chronic diseases in the industrialized world brought about by increases in consumption of harmful substances, shifts in dietary consumption patterns, sedentary lifestyles, and chemical production. Government policies—for example, regulating or discouraging high-risk consumption and production, providing support for the economically vulnerable, promoting productivity, and cushioning the adverse effects of structural change and recession—are the most effective mechanisms in industrialized societies for moderating the health-damaging effects of economic trends.

Thus the nearly exclusive emphasis in the field of health economics on health policy, and more particularly on health-care financing, ignores those policies that most profoundly influence a nation's health, and shape the overall political economy and its components, including real family income, per capita wage rates, unemployment rates, business-failure rates, consumption patterns (based partly on income availability), and specific sources of industrial toxicity. Thus, a society-and-health perspective should not only be concerned with the regulation of occupational and environmental exposures, food quality, and traffic safety, but it needs to view industrial, economic, and social welfare policies as the essential levers for improving health.

Political Economy and Health

If we can show that specific macroeconomic indicators and aggregate patterns of consumption and production are the main determinants of trends in population health, then those policies designed and implemented to influence these indicators must be acknowledged as the main policy sources of a nation's health. This chapter will argue that the backbone of a nation's health is its overall wealth production and the productivity of its technology and labor force.

A secondary determinant of population health is the relative stability of economic growth. The quasi-cyclical economic changes that accompany variations in the business cycle, especially as they affect unemployment and business failure, represent profound shocks to the population's economic, physical, and mental health. The damaging effects of these changes persist for at least a decade.

A third determining factor is the extent of economic inequality—that is, the degree of unequal distribution of wealth and of unequal consumption of the commodities and services that represent the products of economic growth. Economic inequality is heavily influenced by the cyclical nature of economic growth, especially the unemployment rate, although such factors as government-financed income supplementation and investment in "human capital," education, and health care play a substantial role. Economic inequality is mitigated to varying degrees by government-based programs, including education, and income supplementation for the elderly, unemployed, and impoverished.

Long-term economic growth has traditionally been assumed to promote increased economic equality (that is, income). In the last 15 years, however, this fundamental assumption has been undermined in the United States by the uncoupling of wage rates from real per capita income, so that economic growth has not lead to less income inequality. Long-term decline in high- to medium-wage manufacturing jobs and a substantial increase during the 1980s in new jobs characterized by substandard wages, benefits, and tenure appears to be largely responsible. This decline has resulted from increasing pressures for the replacement of labor by high-technology machinery, and by heightened investment in overseas plants where wage rates and benefits are substantially lower, unionization is weak, and occupational and environmental regulations are minimal.

It is the overall wealth of a society that enables it to invest in occupationally and environmentally safer production processes and products. Similarly, the extent to which a firm is willing to introduce safer—and more expensive—equipment and products depends on whether such changes will threaten its economic survival. And the extent to which industry and the general public can be persuaded to replace standard but high-risk commodities with safer products partly depends on the magnitude of the financial losses that would be incurred by industry. Such losses are minimized in periods of high economic growth. Finally, the extent of investment in basic research depends on the financial health of government and industry.

This chapter will test an empirical version of this formulation, the economic-change model of population health, using U.S. mortality data for the period 1950–1988.[1] The premises of a national-level, or macro, formulation of this type are considerably strengthened and validated if we can specify the individual-level or micro theoretical components of the model and secure corroborating evidence at the micro level. The fundamental micro-level hypothesis supporting this model views changes in socioeconomic status (SES) as a byproduct of market forces, economic policy, and social-welfare policy. Changes in SES in turn influence virtually all morbidity and mortality patterns, through several mechanisms: alterations in the material standard of living; changes in consumption patterns (which amount to changes in the person-specific biochemical environment); and changes in psychophysiology involving threats to self-esteem, personal security, autonomy or sense of control, achievement and creativity, strength of social relations, and identity with social groups.

It is crucial to demonstrate the key link between national economic change and mortality is SES because the most pervasive and historically important relationship in epidemiology is between SES and morbidity and mortality rates. This relation has traditionally been demonstrated using ranked occupational differences. More recently, education and, especially, income have been used as measures of SES, and both demonstrate an inverse relation to mortality rates. A vast epidemiologic literature has also accumulated on the significant and consistent relationship between unemployment and elevated morbidity and mortality rates.

In sum, the relations between SES and health at the national level show a high degree of explanatory power, statistical significance, and robustness. They also show little tendency to change from one 15-year period to another. This consistency over time makes for a high degree of predictability, and lends considerable support to the enactment of policy interventions, designed to improve economic well-being, that are the principal sources of improvement in the population's health.

After a discussion of the multiple links between SES and health, we will consider

1. This model, which has been under development for many years, has benefited from both methodological and substantive critiques of its earlier versions. A principal innovation has been the use of the Shiller procedure for the estimation of the cumulative relations of economic and epidemiologic risk factors that affect mortality patterns.

SES as a set of intervening variables that act as the link between changes in the national economy and population health. We will then consider the economic-change model of population health, along with empirical data on the capacity of the model to explain changes in mortality rates.

Mechanisms Linking Economic Position and Health

Socioeconomic Status as an Intervening Variable

It is widely recognized that the performance of the economy has a profound impact on a nation's health. As the standard of living increases over time, life expectancy normally increases and age-specific illness rates from most causes decline. We also have very clear evidence of the inverse relationship between socioeconomic status and illness and mortality. Indeed, SES is the most powerful and consistent epidemiological risk factor.

Changes in population SES in turn depend almost entirely on national economic changes, which are a product of market forces and public policy, that is, the political economy. Market forces consist of the following: (1) technological changes, dependent on investment in research and development and in new technology; (2) productivity changes, based largely on technological change and investments in "human capital" and infrastructure; (3) structural changes in industry and the labor force, lately involving large-scale decline in manufacturing employment and an upsurge in relatively low-wage service employment; and (4) international competition in manufacturing, finances, and exports. The most influential but routine policy changes are those undertaken to influence the national economy and the welfare of vulnerable or dependent populations: monetary policy; fiscal policy, including defense, education and infrastructural expenditure; employment retraining and compensation; taxation, minimum-wage policy, and social-security and welfare policy. Other policy changes are based on political movements dealing with the rights of specific social groups, such as women, the elderly, and ethnic and racial groups.

Health research has typically treated the role of SES in two ways. First, SES is treated as a proxy for other epidemiological risks, such as excessive consumption of alcohol, tobacco, and fats or exposure to environmental toxins, whose association with measures of SES is taken as incidental. Or, alternatively, SES confounds (or disturbs) estimation of the "pure" statistical impact of such risk factors on health. In other words, SES is not understood as a major variable, with its own specific but profound impact on health. Third, in a more sophisticated fashion, SES is viewed as a causal precursor of a set of epidemiological risks but rarely as a variable that has origins in economic, political, and cultural change.

This chapter proposes a fourth, more dynamic view of the role of socioeconomic status, placing it in an intermediate position between macroeconomics changes and health: that is, SES is a dependent as well as an independent variable. In a technical

sense, therefore, SES is an intervening variable between the macro economy and the major risks to health. It is important to note however, that SES is simultaneously an interacting variable. In this case, SES interacts with changes in culture, technology, and government policies in ways that modify, but have tended to reinforce, health inequalities.

Direct SES-Health Mechanisms

The mechanisms that link SES to health, can be classified as material, psychophysiological, and biochemical risk factors. These mechanisms are both direct and indirect (interactive) in nature. The most direct involve material factors dependent on income and wealth: nutrition, sanitation, housing, industrial engineering, transportation, primary education, health care. They are especially pertinent to infectious diseases, infant and child mortality, and injuries.

Among psychophysiological mechanisms, which influence the chronic diseases (cardiovascular, immune, metabolic) and those that involve mental health (depression, accidents, intentional injury), three broad types can be said to be direct in their effects. The most general involves the self-valuation of the person in terms of the individual's overall status and role (Linton, 1936; Parsons and Shils, 1951), that is, economic position (occupation) and social contribution (content of work and achievement). Individuals are judged against others and against the value standards of their society: Weber (1946) called this "status honor." A person's own judgment of intrinsic self-worth is not idiosyncratic but reflects that of the surrounding society (Cooley, 1902; Mead, 1934). As William James (1890) was the first to point out, public esteem is translated into self-esteem on the part of the individual. Negative self-esteem is closely related to the feelings of shame (Erikson, 1963), stigma (Goffman, 1959) and, most important, deprivation (of social importance). It is thus interpreted as a loss of "what might have been."

A second psychophysiological dimension of socioeconomic status is market position (Weber, 1947), or the ability to obtain income and wealth and thus to consume. Market position is manifested in the use of socially valued goods, services, and symbols that signify comfort, pleasure, intellectual stimulation, and participation in cultural life. From a psychological viewpoint, lack of access to desired consumption generates chronic stress, frustration, and tension, and ultimately profound deprivation.

A third psychophysiological dimension of socioeconomic status is the broad system of incentives and rewards that accompany status and role (occupation and performance) (cf. Davis and Moore, 1945). In addition to income, the rewards of assumed productivity include authority, skill and/or knowledge, access to social networks, and job and income security (cf. Weber 1947; Dahrendorf, 1959; Treiman, 1977), all of which represent basic resources with which the physical and social environment can be influenced. From a psychological standpoint, these resources contribute to a "sense of mastery" of potential life problems, or assurance of lack of vulnerability to stress or threat.

Interactive Mechanism I: Economy and SES

Let us now look at the interactions between various attributes of SES and changes in the social environment. The most important of these interactions links individual economic circumstances—especially change of job or income and work stress—to the fate of one's employer. It is the firm's capacity to survive and thrive in the competitive economic environment that determines its ability to offer employment, wages, and benefits, not to mention safe and pleasant working conditions.

In recessionary periods, strong firms with significant working capital, whose losses in sales are not substantial, usually retain their employees without adversely altering wages or working conditions. The reverse is true of the weak firm in a poor market position. According to the new ecological school of organizational demography, the environment of such a firm's employees depends largely on how well the firm manages its position in the competitive marketplace (Hannan, 1979; Nielson, 1980). When market competition creates a threat of layoffs or acceptance of adverse working conditions, the question that arises is who is to bear the brunt of those threats. In this situation, status within the firm is paramount. Socioeconomic status within the firm involves bureaucratic authority (Weber, 1947), that is, the authority attached to one's formal work position. It is this source of authority that has been hypothesized as the core of SES, or of social class (Dahrendorf, 1959; Blauner, 1972; Orum, 1978). Authority in the firm determines who is to steer it, who is sufficiently valuable to be retained in difficult times, who will be promoted in times of growth, and who will be harmed or benefited by investments in new technology.

The extent to which an individual will experience economic stress thus depends on the interaction of "demands" and "control" factors (Karasek and Theorell, 1990). Demands on the employer reflect the firm's ability to manage its economic environment, while control depends entirely on the firm's authority structure. Thus, the incidence of significant economic stresses depends largely on the economic conditions affecting one's industry and one's firm, and the responses of the firm's authority structure. Those stresses then filter down to the lower SES strata of the firm, where they stimulate individual coping strategies.

Interactive Mechanism II: "Lifestyle" and SES

Richard Doll (1987) explains patterns of change in the major causes of death since the early 1900s by applying classic "epidemic" models. According to Doll, the relevant health-risk behaviors have tended toward epidemic rises, peaks, and declines for two separate reasons. First, increased personal disposable income made items that had been relative luxuries (alcohol, tobacco, fat- and protein-rich foods) considerably more accessible to middle- and lower-income groups. Second, the diffusion of these new patterns of consumption throughout society (Hamblin et al., 1973; Burt, 1978) resembles an exponential function that approaches an asymptote. Subsequent decline in epidemic risk factors is promoted by health messages in the mass media, which report the latest findings on the health risks of various forms of consumption and exposure.

The evidence is strong that such "epidemic" changes in patterns of health-risk behaviors distribute themselves differentially among socioeconomic groups (Morris, 1979). Indeed, the rate of diffusion of common risk factors is so highly related to social class that epidemiologists are often content ot explain the entire SES health relationship by invoking this differential distribution (see Morris, 1979). Diffusion theory (Rogers and Shoemaker 1971) and social-marketing theory (Manoff, 1985) provide relatively clear-cut explanations of how the diffusion of risk factors interacts with socioeconomic level. Initially, the most highly educated individuals (those whose exposure to new information is greatest, and whose social status and wealth also tend to be high) are the first to procure goods newly available on the market. Gradually the rest of society becomes aware of their availability, and (as long as there are continual gains in real disposable income) "catches up" with the innovative leaders (Rogers and Shoemaker, 1971). The retrograde component of the diffusion curve is set in motion only after considerable investment in research on the health implications of the new patterns of consumption and exposure. The time lag between research investment and initial findings is lengthy: because 10–30 years of cumulative exposure is often required for a risk factor to take effect, a generation or more can pass before solid scientific evidence emerges of health-damaging effects.

Thus decades may pass before awareness of the risks to health of given consumption behaviors is diffused throughout the social-economic structure. The retrograde diffusion process may require another generation and may again proceed at different rates depending on social status, education, and income (McQuail, 1988). Income is especially relevant to the capacity to purchase substitutes for risk-laden consumer items. This argument has been used to explain the shift in coronary heart-disease mortality in Britain from an initially higher–SES based mortality rate to the reverse (Marmot et al., 1978, 1987). It can also be applied to any health-risk behavior in which individual choices of consumption items are relevant.

Interactive Mechanism III: Chemical Toxins and SES

Toxic residues of chemical production are a prime source of carcinogenicity and respiratory disease. Exposure to such residues is of two types: direct occupational exposure of employees in firms that produce or use such chemicals, and exposure of the general population to a contaminated environment.

Interaction with SES is substantial in both cases. Within a high-risk firm, lower-SES employees will experience the most exposure (Registrar General, 1976) because those in authority will leave the handling of dangerous materials and equipment to those who are not in a position to refuse. In general, high-risk firms, with relatively poor security measures and the least-sophisticated equipment, are found in the poorer subgroup of the segmented labor market (Doeringer and Piore, 1971; Edwards, 1975), and their

employees are likely to be those with the least skills and education; these individuals also earn the lowest wages and benefits, and (in the United States) have the least adequate health-care coverage.

The relative lack of authority and, thus, heightened vulnerability of lower-SES employees is compounded by their limited knowledge of occupational health risks and tendency to assume that, in any event, they cannot alter their work situation. These sources of occupational vulnerability are exacerbated by the relative lack of political influence of lower-SES groups on the development of occupational safety and health codes.

Place of residence is also a key risk factor in exposure to chemical carcinogens (Epstein and Swartz, 1981). Here again, the relative lack of political influence of lower-SES groups makes them especially vulnerable, as do the financial and discriminatory components in "choice" of place of residence. Obviously, the families with the fewest resources are not in a position to insist on environmentally healthy neighborhoods.

Alternative Explanations For Differences In Individual SES?

Origins of SES for Individuals

Economics and sociology offer four alternative explanations of differences in socioeconomic attainment: these are referred to as (1) institutional determinants, (2) human capital, (3) statistical discrimination and (4) status attainment.

The institutional determinants of individuals' SES are the differential rates of growth (and sometimes decline) of different industries, which therefore offer differential opportunities for advancement. Of particular importance in the literature is the small firm, in the poorly capitalized sector of the "segmented labor market," that offers inferior wages, benefits, and working conditions (Doeringer and Piore, 1971; Edwards, 1975). This segment of the labor market is subject to high failure rates and high unemployment rates, and is obviously highly sensitive to national, regional, and even international business conditions. The literature on institutional determinants makes clear that the basic structure of SES for a nation, and thus the parameters of socioeconomic attainment for the individual, are highly dependent on macroeconomic conditions.

Human capital signifies the skill level and productive capacity of the workforce, and thus its occupational abilities and earning capacity. The human-capital school of thought maintains that investment in education and training, by the individual and by society, is crucial to advances in SES (Becker, 1964; Thurow, 1969). Development of human capital requires both institutional structures (e.g., social investments in science and/or knowledge development, educational organizations, and manpower) and, on the part of the individual, sufficient income, leisure, and willingness to forego employment opportunities. Clearly, human-capital investments depend, foremost, on national economic growth and per capita disposable income.

The statistical-discrimination approach posits that, beyond skill and appropriate credentialing, "ascriptive" (Parsons, 1951) aspects of social status (sex, race/ethnicity, age, religion, national origin, region of origin) play a part in determining occupation, job position, and earnings (Coleman, 1974; Thurow, 1987). Over the long term, however, "universalistic" (i.e., objective) and formalized standards increasingly dominate hiring practices, work authority, and incentive and reward systems. This tendency is due to the increasing formalization ("bureaucratization") and professionalization of work, and to increasing requirements for formal training and certification (as suggested by the human-capital school).

The status-attainment approach to differences in SES (e.g., Blau and Duncan, 1967; Sewell and Hauser, 1972; Treiman and Terell, 1975; Featherman and Hauser, 1975) views socialization in childhood as a crucial determinant of future occupation and living standards. This outlook views the family subculture, particularly the occupation of the father (and, increasingly, the mother), as a powerful influence on future socioeconomic status. The SES of family of origin is thus a conservative force, tending to preserve the stability of the social-stratification system.

In sum, leaving aside issues of family and poor socialization, theoretical explanations of differential socioeconomic status focus on economic development and change, emphasizing the mechanisms of industrial development, human-capital formation, and formalization of occupational requirements. One might also add social-welfare payment mechanisms to support the incomes of lower socioeconomic groups, and government policies favoring progressive income taxation and labor-management wage-benefit negotiations.

Effects of Economic Change on Socioeconomic Status

Changes in the overall socioeconomic level, i.e., the standard of living, and structure (industrial and occupational) of a population are largely dependent on its rate of economic productivity, which in turn largely reflects technological developments (Krugman, 1991; Griliches, 1988; Kendrick, 1980, 1984). The degree of income inequality is related, over time, to several phenomena: the unemployment rate as an indicator of macroeconomic fluctuations (Dunlop, 1938; Menderhausen, 1946; Budd and Whiteman, 1978; Nolan, 1987; Podgursky and Swaim, 1987a, b); differential rates of development (and decline) of specific industries and regions (Kendrick, 1983; Harrison et al., 1986); union activity (Economic Policy Council, 1983; Weisskopf et al., 1983; Freeman and Medoff, 1984; Marshall, 1986); and policies on investments in human capital, infrastructure, and support of vulnerable and dependent groups (Kuttner, 1986; Anderson, 1986). Of lesser significance to income inequality are birth-cohort size (Dooley and Gottschalk, 1984) and such attributes of firms as size, market concentration, and capital to labor ratios (Hodson, 1983). Genetically based ability and aspects of family background are potentially important, but do not appear to influence the SES

structure over time. Income discrimination based on race, ethnicity, sex and age, though subject to political change, can alter the structure of economic inequality.

Feedback Mechanisms

While relations between measures of SES and health have proven consistent and powerful, causation has not been well delineated, often because its direction is incompletely specified. Reverse causation is logically possible: genetic and biological (that is, intrauterine) factors could account for differing intellectual abilities and functional capacities (thus affecting SES), and for differing morbidity and mortality rates. A substantial research literature stresses the impact of illness, disability, and addictions on reduced functional capacity, including work productivity (Rice, 1966; Luft, 1978; Manning et al., 1982). The possibility of reverse, or lack of, causation even applies to the impact of unemployment on health and mortality. It is plausible, for example, that even in a recession those most likely to be laid off are the least psychologically or physically healthy, who function most poorly. In the short term and certainly in the long term, this group might plausibly have higher mortality and morbidity rates than the employed, apart from any effects of unemployment itself. It is undoubtedly true that illness can lead to declines in economic functioning and thus in occupational level and income.

To the degree that either of these reverse-causation arguments is accurate, economic and social policy intended to improve health would clearly be ineffectual. When the SES-health relation is indirectly measured, however, and given a general ecological expression in which the subjects are unable to influence the economic environment, the relation persists. It thus becomes difficult to argue that the causal direction is reverse. At the macro level, several additional observations make reverse causation unlikely.

First, changes in the overall economy are unlikely to be influenced by the activities of seriously ill individuals. (One could even argue that the death of the very ill contributes to the economy by freeing up resources.) There is a mass of literature on the factors that normally influence economic growth and economic cycles, and mortality patterns (especially in industrialized countries) are not among them. Second, economic changes precede changes in mortality patterns: both short-term and long-term relationships are identifiable between changes in the economy and changes in mortality. Third, relations at the macro level are in accord with similar relations at the micro level, and with theoretical explanations of mortality patterns both in the social sciences and in psychophysiology.

A more plausible explanation is a straightforward feedback mechanism. Thus (1) low SES involves higher incidence of illness and/or disability due to inequalities in the distribution of material, biochemical, and psychosocial benefits and risks. In turn (2) serious illness and/or disability adversely affects employment possibilities, earnings, and productivity. This feedback effect may explain how recessions engender latent increases in mortality rates due to chronic disease: namely, downward social mobility is accelerated and reinforced by chronically compromised health.

Macroeconomic Determinants of Social Stratification

Long-Term Growth of the Economy

The distribution of jobs (i.e., the distribution of skilled and semi-skilled manufacturing versus customer-service and sales jobs), which depends on the level of technology prevailing in the economy, and the pay levels associated with the status of those jobs, are widely agreed to be the major determinants of income inequality. As Shorrocks (1987) puts it: "Job distribution, together with customary wage and salary differentials, . . . is the principal determinant of earnings inequality. Personal characteristics appear to be important only because they are used by firms to ration entry into the more attractive jobs."[2]

Long-term income inequality is accounted for by factors of economic growth, demography, and education. Kuznets (1955), whose work addressed the relation between economic growth and income dispersion within countries, found an increase in inequalities at the early stage of industrialization, in conjunction with large-scale movement out of agriculture into the modernizing sector. As the modernizing sector becomes dominant, inequalities decrease. Williamson and Lindert (1980) found evidence of this pattern in the United States: relative wage equality prior to 1816; striking increases in inequality in 1816–1856 and 1899–1916; and secular declines in inequality between 1929 and the Korean War.

The Impact of Recession and Unemployment

The business cycle, and specifically the unemployment rate, is the second macroeconomic influence on income inequalities. Inequalities widen as the business cycle contracts, specifically as unemployment rates grow. Kuznets (1953) found a tendency in the interwar period (1919–1939), for overall inequality to move inversely with the business cycle, the upper-income group gaining at the expense of the vast majority (probably no less than 85–90 percent) of wage-earners in periods of economic downturn (Kuznets, 1953, pp. 57–58). An earlier study (Menderhausen, 1946), found a similar pattern during the Great Depression (1929–1933).

2. On the issue of wage levels conventionally linked to social status of occupations, Phelps Brown (1987) states: "People insist on the maintenance of customary relations. They require that relative pay conform with status. That the laborer's rate stood at two-thirds of the craftsman's in the building industry of Southern England over more than six centuries . . . can be attributed only to convention. If women's rates had been simply proportioned to productivity, it is hard to account for their relative rise when the rule of 'equal pay for equal work' was enforced in the UK and the Netherlands . . . These observations are all consistent with differences of pay being set to match accepted gradations of status."

The pattern of increasing inequality in recessions and narrowing inequality in subsequent recoveries has persisted in a less extreme form in the postwar period, according to annual data from the U.S. Bureau of the Census.

Post-1970s Structural and Demographic Shifts

Income inequality in the United States, continued to shrink from the end of World War II until the late 1970s and early 1980s, when inequality began to increase again. This trend continues in the present (Levy, 1987; Maxwell, 1989; Winnick, 1989; U.S. Congress House of Representatives, 1989; Phillips, 1990; Ryscavage and Henle, 1990). For the first time in American history in fact, the economic expansion of the 1980s was accompanied by deterioration in the standard of living for a majority of households (Sweeney and Nussbaum, 1987; Harrison and Bluestone, 1990; Litan et al., 1990).

The existence of increasing inequality and real-wage stagnation in a period of economic expansion has several linked explanations (Maxwell, 1989). First, striking increases in foreign competition and dollar devaluation since the 1970s have forced fundamental changes in the manufacturing and export-oriented industries. These changes may reflect cyclic movements intrinsic to market economies (Thurow, 1980; Feldstein, 1980), as well as a permanent structural shift from manufacturing to service employment (Bluestone and Harrison, 1982). Because the resulting slowdowns in real wages and productivity vary among industries and occupations, changes in income distribution have resulted.

Dooley and Gottschalk (1985) have shown that low-wage workers experienced below-average wage growth. Meanwhile, workers in the rapidly growing high-technology industries, a high proportion of whom are highly paid (Rosenthal, 1985), experienced gains in real income (McMahon and Tschetter, 1986). Polarization of income distribution, and potentially a decline in middle-class income, follows in turn from simultaneous growth of low-wage and high-wage employment (Harrison et al., 1986).

Maxwell's (1989) multivariate statistical analysis of U.S. income inequality in the period 1947–1985 considers these factors as well as demographic shifts and government programs. The results show that income inequality is promoted by increases in the proportion of the nonworking (dependent) population, in service-sector employment, and in the proportion of blue-collar workers. Growth in the dependency ratio and in service-oriented industrial employment increase inequality by increasing the top quintile's income share and decreasing the bottom quintile's. Industrial and demographic forces, such as changes in labor market and immigration, exert the greatest impact on the income share of the lower quintiles, particularly that of the very lowest quintile.

While Maxwell (1989) found government spending on social welfare programs only affects the top and bottom quintiles, cross-national studies do indicate that social-welfare expenditures can have a marked effect on income equality. Of 10 intensively studied Western European and North American countries (Smeeding, 1991), the Scandinavian countries and Germany show the lowest income inequalities and the United States the highest.

The Cyclical Behavior of Income Inequality

The cyclical nature of earnings distribution is a major generator of income inequality. Even if workers who lost their jobs were drawn proportionately from all wage levels, according to Menderhausen (1946), they would be pushed into zero-income or very low income positions compared to those who retained their jobs; this enlarges the income gap between the unemployed and underemployed on the one hand and those with relatively full-time employment on the other. And, in reality, all workers do not face the same probability of becoming unemployed in a recession; the incidence of unemployment is greater among the less skilled and lower paid. Thus, wage differentials can be expected to widen, partly because unemployed workers are reemployed in lower-wage jobs.

During periods of declining product demand, Oi (1962) argues the employer has an incentive to retain workers with larger investments in human capital (the more skilled and highly paid) and to lay off those with little specific training. The result is larger increases in unemployment among the unskilled, and/or widening of wage differentials.

In the same vein, Reder (1955) argues that, instead of laying off both skilled and unskilled workers in a recession, employers choose to assign their underemployed skilled workers to less skilled jobs and to concentrate layoffs among the less skilled. Employers also tighten hiring standards in recessions and relax them in subsequent expansions.

Among the many studies of the impact of macroeconomic conditions on inequality, the most consistent finding has been a significant disequalizing effect of increases in unemployment, reducing the share of total income earned by lower-SES groups.[3]

Economic Change and Health: Macro and Micro Evidence

Stable Economic Growth and Health

Having explored the relationship between SES and health, we can now ask: What, theoretically, is the importance of the economy to the nation's health? To answer this question, let us look first at the health implications of stable economic growth.

The material contribution of economic growth is the basic differentiator among societies in terms of mortality rates. It separates the high-life-expectancy countries from

3. Schultz (1969) initiated the econometric analysis of the topic and estimated an equation relating the inequality in the distribution as measured by the Gini coefficient to variables such as inflation, unemployment, the growth rate, and time trend. Schultz failed to identify any significant influence of macroeconomic variables on the Gini coefficient for the U.S. distribution, while the remaining, disaggregated studies, did find such effects on the distribution time series. Simulation approaches based on micro data (Mirer 1973a,b; Budd and Whiteman, 1978; Nolan, 1987), panel analysis (Gramlich, 1974) and studies specifically of poverty (e.g., Hollister and Palmer, 1972) were also able to estimate the positive relation between recession, or unemployment, and income inequality.

low, and is the prime factor in infant and child survival and infectious diseases rates. Its key elements, as we have seen, are nutrition, housing, sanitary engineering, primary education (of the mother), and primary health care, all of which require societal investments and thus sufficient national wealth. Investments in industrial design and sophisticated equipment are also necessary to prevent disability and mortality due to unintentional injuries.

Stable economic growth is the *sine qua non* of investment in knowledge development, including systematic epidemiological investigation of health-risk factors. Such investment is crucial not only to engineering and the biological and physical sciences but also to the social and economic sciences instrumental in managing the overall economy and individual firms. Joint development of the material economy and of the scientific base provides for predictability, manageability, and comprehensibility in the human and physical environments. Stable economic growth is the prerequisite for fundamental political undertakings designed to promote equality of distribution (labor-management bargaining, taxation, and social-welfare expenditures). These fundamentals contribute in turn to a human psychology of hope and confidence (control, "self-efficacy," and security).

Stable growth is critical to social integration as well. Perhaps the most significant source of social integration is the process of urbanization. The transition of the bulk of the population from rural to urban life may well have caused a basic shift in human relations, as Toennies (1952) and Durkheim (1947) have asserted. In any event, the development and maintenance of urban life has generated the formation of numberless social organizations based on interests and values. It is inaccurate to say that the advent of such formal "secondary" institutions has caused the wellsprings of primary face-to-face relations to "dry up." On the contrary, formal organizations—from workplaces to government—require primary relations *within* the groups that constitute their working parts.

The Impact of Recession and Unemployment on Health: Descriptive Epidemiology

Studies conducted over the last 20 years on the relationship of economic change to morbidity and mortality in industrialized countries represent two distinct epidemiological traditions in the mental and physical disorders. The earlier work on mental-health problems tended to emphasize the role of economic loss in stress. More recent work on physical health has made it apparent that recession itself plays a causal role. It is now recognized that loss of resources and psychological stress are not easy to isolate causally: the implication is that, in the vast majority of pathologies, including mental and physical illness and criminal aggression, some combination of resource loss and psychological stress is probably involved.

Suicide

Suicide was the first indicator of mental pathology found to increase consistently with adverse changes in the economy (Durkheim, 1951; Henry and Short, 1954; Brenner 1971b, 1980a, 1984a; Vigderhous and Fishman, 1978; Boor, 1980; Marshall and Hodge, 1981). A number of researchers have replicated these findings, attesting to the severe stress brought about by economic recession (Brenner 1976b, 1984a, b; reviewed in Platt, 1986).

Mental Health

Research on the relationship between adverse economic changes and increases in mental-hospital admissions has demonstrated that the relationship can be measured consistently over time (Dayton, 1940; Pugh and MacMahon, 1962; Brenner, 1973a). The first of these studies dealt with Massachusetts during the 1930s, the second with the United States as a whole during the Depression, and the third with New York State in the period 1841–1967. During each recession since 1841, first admissions and readmissions to mental hospitals increased substantially. Cyclical change in the economy was the single most important factor affecting trends in admission. The New York State study has been replicated for the entire United States and for each state in the periods 1936–1970 and 1950–1980, with nearly identical results (Brenner, 1976b, 1984a).

Numerous epidemiological studies on the relation of unemployment to mental health have found symptoms of psychological disorder to be consistently associated with unemployment (Ferman and Gardner, 1979; Dooley and Catalano, 1984; Elder and Capri, 1988; Forcier, 1988; Kessler et al., 1988; Penkower et al., 1988; Warr et al., 1988; Hamilton et al., 1990).

The relation of criminal aggression—especially homicide, crimes known to the police, arrest rates, and imprisonment, particularly for violent crimes but also for all crimes against persons and property—to economic recession and especially unemployment has been observed for decades (Brenner 1976a, b, 1980a, b, 1984a).

Alcohol Abuse

The findings on alcohol abuse are consistent with a hypothesis of increased mental disorder precipitated by social-psychological stress during economic recession. Increases in mortality rates due to cirrhosis of the liver are positively related, over time, to consumption increases with distilled spirits (rather than wine or beer), and such consumption increases with cyclic declines in the national economy. Cirrhosis mortality itself increases substantially 1–2 years after national economic recession. Because it takes a long time to acquire chronic cirrhosis of the liver, it is clear that the short-term economic trauma had not initiated the cirrhotic condition, but that, once morbidity was present, economic recession tended to hasten mortality (Brenner, 1975).

Other findings underline the relationship between increased consumption of distilled spirits and declines in the national economy. Admissions to mental hospitals for psychosis related to alcoholism and other alcohol-related mental disorders showed substantial and stable increases during economic recessions during the period 1921–1968. Similarly, arrests for drunkenness in Massachusetts increased with adverse changes in the national economy over the period 1915–1968; the arrests lagged 2 years behind

fluctuations in the economy. Finally, arrest rates for driving while intoxicated were found to increase substantially during national economic recessions (Brenner, 1975).

Heart Disease

Early, empirical research on heart-disease incidence and mortality clearly demonstrated that fluctuations in heart-disease mortality rates were inversely related to the employment rate in New York State in 1915–1967. Mortality from coronary-artery disease was found to be related to fluctuations in the unemployment rate for the United States as a whole in the period 1930–1960. Both studies found that mortality peaked at least 2–3 years after economic recessions (Brenner, 1971a, b).

Subsequent studies of the relationship between national economic indicators and cardiovascular-renal disease hypothesized that these illnesses are characterized by significant psychophysiological factors involving hypertension and serum-cholesterol levels. Cardiovascular-renal diseases account for approximately 60 percent of all mortality in many industrialized nations. An examination of U.S. data for 1914–1968, by race, sex, and 10-year age groups, found a consistent relationship between national economic fluctuations, measured by per capita income or employment rates, and mortality rates due to cardiovascular renal disease. For cardiovascular-renal diseases in general, the peak lagged behind economic recessions by 3–6 years, depending on age; the peak for chronic nephritis lagged from 0 to 2 years; for cerebrovascular diseases, the lag ranged from 6 to 9 years (Brenner, 1971a, b).

Infant Mortality

The infant-mortality rate has long been recognized as one of the most sensitive indicators of the general socioeconomic level of a nation. The relationship between the economy, and infant, fetal, and maternal mortality has thus been extensively examined. In all industrialized countries for which data are available, industrial growth is inversely related to the long-term trend in infant-mortality rates (Susser et al., 1985). However, a society's adaptation to economic change is less a matter of the level of economic growth than of whether that growth is relatively smooth or unstable.

An investigation of the relationship between economic instability and mortality in infants under 1 year of age found fetal and infant mortality to increase sharply in response to economic recession, with a peak lag of 0–2 years (Brenner, 1973b). This relation has been demonstrated for the United States in 1936–1974 and 1950–1980 (Brenner 1976a, b, 1984a), the United Kingdom (Brenner, 1979, 1983), and Sweden (Brenner, 1987b). The relationship between economic fluctuations and trends in infant-mortality rates has historically been an important component of the relationship between low socioeconomic status and increased infant mortality; that is, the impact of economic shock, such as on infant mortality, is greater for lower-SES groups. The influence of economic instability, especially recession, on infant mortality trends has grown since 1950, perhaps because of a relative decline in the beneficial impact of secular economic growth in an economy that is already highly developed.

Overall Mortality

The influence of economic change on overall (non-cause) mortality rates has been studied for several decades. The principal long-term declines in overall mortality, as well as age-specific mortality for all age groups, are associated with long-term improvements in the national economy, especially real per capita incomes. The business cycle is heavily influential in the short- to medium-term fluctuations in mortality rates. Such variables as unemployment and business-failure rates are typically associated with increases in mortality two to three years following the lowest point in the business cycle and extending for at least the next 10 to 15 years. The relationships have been most fully explored with data for the United States, United Kingdom, and Sweden (Brenner, 1976a, 1979, 1980b, c, 1984a, 1987b, c, 1991, 1993). Middle-aged mortality rates have also been examined from this viewpoint in at least 10 additional countries (Brenner, 1989).

These types of relationships for overall mortality—with long-term trends inversely related to economic growth and short- to medium-term fluctuations significantly associated with economic cycles—have also been observed for the principal cardiovascular diseases, even when the models included controls for tobacco, fat, and alcohol consumption patterns (Brenner, 1976b, 1984a, 1987a, b, c; Brenner and Mooney, 1982).

Microepidemiological Research on Unemployment

Unexpected large-scale increases in unemployment rates in European countries that have occurred since the 1973 oil crisis and which continue to the present day have interested epidemiological researchers working at the individual level in prospective studies of morbidity and mortality. A classic study by Moser, Fox, and Jones (1984) examined a nationwide sample of employed and unemployed British men in 1971 to see whether an impact on mortality in 1982 could be discerned. They found not only that the unemployed men had substantially higher standardized mortality ratios (SMRs), after controlling for social-class differences in mortality, but that the wives of these men (who were not themselves unemployed) also showed significantly elevated SMRs. Similar studies in the United States (Sorlie and Rogot, 1990), Denmark (Iversen et al., 1987), Sweden (Stefansson, 1991), and Finland (Martkainen, 1990) found significantly elevated SMRs a decade after the unemployment event; those who were not seeking work or were otherwise outside the labor force tended to show the highest mortality rates.

Studies of psychiatric and somatic morbidity in Germany (reviewed by Schwefel, 1986), the United Kingdom (Jackson and Warr, 1987), Norway (Westin et al., 1989; Westin, 1990), the United States (Linn et al., 1985), and Denmark (Iversen et al., 1989) have repeatedly confirmed the importance of unemployment as a prospective risk factor in physical and mental illness. These studies have taken care to separate out the effects of social selection, which can be especially pernicious in unemployment-health studies because seriously ill and disabled individuals are highly vulnerable to job loss.

While there has been remarkable consistency in the findings of individual-level epidemiological studies, studies of the relation between unemployment and subsequent morbidity and mortality at the national level have not been as consistent. Macro-level suicide and mental-health studies have tended to confirm the generally adverse impact of unemployment, but the results of studies of overall and chronic-disease mortality have been more mixed (Forbes and McGregor, 1987). Whenever controls for traditional risk factors (alcohol, tobacco, fat consumption) have been used, however, the results have uniformly shown elevated mortality rates following recessions, peaking after approximately 2–3 years (Brenner, 1971a, b, 1980a, 1984b, 1987a), and frequently extending as long as 10–15 years (Brenner, 1976a, b, 1984b, 1989). The overall length of the lags is similar to those found in longitudinal epidemiological studies involving traditional risk factors, and may in fact reflect the interaction of recession and downward mobility with those risk factors. If so, the interaction of unemployment with "lifestyle" and toxicological risks may be similar to the well-documented inverse relation of SES with these risk factors.

Mechanisms Linking Recession and Health

Economic recession engenders damage to health by means of at least six mechanisms: (1) reduced financial access to health care, resulting in underutilization; (2) psychophysiological reactions to stress and loss; (3) damage to social relations and sources of social support; (4) maladaptive coping mechanisms involving high-risk consumption patterns; (5) less thorough and frequent maintenance of manufacturing plants and reduced investment in modernization, with a consequent effect on health and safety; (6) increased work stress during the economic recovery as firms that lost heavily during the recession take on more work but do not yet hire additional workers, due to a capital shortfall and uncertainty about the stability of the recovery.

First, the lack of health insurance and relative poverty of workers in the "secondary" labor market and of people outside the labor force contribute to comparatively low utilization rates of inpatient, ambulatory-curative, and preventive health services, especially among the very young and the elderly (Ford and Marlis, 1990; Rowland, 1990; Whitman et al., 1991; Ahern and McCoy, 1992; Kirkman and Kronenfeld, 1992). Even people who have health insurance are typically required to make copayments so that there is significantly lower utilization of these types of services (Shapiro et al., 1986; Anderson et al., 1991).

Second, psychophysiological coping reactions to stress and loss are known to be highly varied. They include a compromised immune system, which is less able to resist infection and malignancy (Bartrop et al., 1977; Eysenck et al., 1991); effects on the cardiovascular system (Grossarth-Maricek and Eysenck, 1991; Goldstein and Niaura, 1992; Niaura and Goldstein, 1992), including hypertension (Pickering et al., 1991; Schnall et al., 1992), myocardial infarction, angina, and acute cholesterol response (Muldoon et al., 1992); peptic ulcer (Anda, 1992); asthma (Isenberg et al., 1992); disorders of the central nervous system such as migraine, anxiety, and psychotic and adjustment disorders (American Psychiatric Association, 1987); and emotional reactions, especially depression (Paykel et al., 1969; Brown et al, 1973; Brown and Harris, 1978), aggression (Dollard et al., 1939; Miller, 1941; Berkowitz, 1962), and somatization (American Psychiatric Association, 1987).

Third, the ties of family and friendship and peer relations among coworkers have been found to moderate the impact of stress and loss (Brenner and Starrin, 1988; Turner et al., 1990). However, depressive or aggressive reactions to loss and stress can damage social networks by alienating close associates and discouraging them from providing further social support. Indeed, the impact of depression, aggression, or general tension can bring about divorce, and separation or destruction of friendship networks (Liker and Elder, 1983; Coyne et al., 1987; Krantz and Moos, 1987; Rath et al., 1989; Dew and Bromet, 1991; Rook et al., 1991).

Damage to social relations can also result directly from changes in population dynamics during and after economic recessions. According to the push-pull theory of migration, regional (and international) migration is characterized by movement away from areas of meager employment or career prospects to those that offer economic opportunity. This important demographic mechanism comes forcefully into play following nationwide recessions, when people move away from areas (or industries) that have not yet experienced economic recovery and/or expansion to those that have. Such migration therefore peaks at the high point of business cycles. This migration pattern calls for the migrating worker to adapt to a new social setting (Cassel and Tyroler, 1961; Syme et al., 1965; Marmot and Syme, 1976) and overcome social isolation. It also means that the family members and friends left behind will experience loss and long-term deprivation. Furthermore, the communities left behind will suffer a loss of population, and ultimately of economic viability; meanwhile, if migration is substantial, the regions (usually urban) and firms that receive it will also experience problems of adjustment, including weaker social ties among the local (nonmigrant) population.

Relations among different social classes and ethnic groups can suffer considerably under conditions of economic decline (Van Hook, 1990), a phenomenon that lends support to the hypotheses of "frustration-aggression" and scapegoating (Dollard et al., 1939; Miller, 1941; Berkowitz, 1962). This correlation appears to be confirmed by repeated instances of serious ethnic and civil conflict during and after periods of national economic adversity. The related assumption of a strong connection between national economic adversity and changes of government has nearly evolved to the status of a formal theory, in which "political cycles" are a central concept.

Fourth, many social scientists assume that maladaptive responses to chronic stress and adverse life events include adoption of unhealthy consumption patterns. Alcohol (Pearlin and Radabaugh, 1976) in particular has been cited as a maladaptive tool for management of stress and depression, as have tobacco and other addictive substances (Lee et al., 1991; Serxner et al., 1991). Alterations in diet, especially increases in fat and sugar consumption, are also often assumed to be coping devices (Schlundt et al., 1990).

Fifth, minimizing health risks due to occupational and chemical environmental hazards (Susser, 1985) requires considerable investment in equipment maintenance, waste

disposal, and substitution of nontoxic substances for traditional ones. Substantial health risks, therefore, accompany the depletion of investment capital, which can reduce compliance with the regulations of occupational and environmental safety agencies (Leigh, 1989).

Finally, the impact of recession and economic instability on a firm's efforts to survive may be the source of severe work stresses related to serious illness and premature mortality. During a recession, anxieties over potential job loss and threats to career and financial solvency pervade the firm (e.g., Kasl and Cobb, 1982). Recessional stresses are also likely to diminish worker autonomy, due to decreased managerial tolerance for error while the firm struggles for survival. This situation is compounded by damage to social relations (and thus social support) among workers when every worker is worried about job security and no one wants to be linked to those whose jobs are to be terminated (House et al., 1988). More influential, however, in a financially damaged firm is the perpetuation of work stress well into the period of national recovery and expansion. Such a firm will have experienced a shrinkage of working capital and will have an especially difficult time knowing when to hire in the face of growing market demand, a phenomenon known as "the signal extraction problem" (Attfield and Duck, 1985). This difficulty will result in acute and chronic shortages of staff and an extraordinary emphasis on high employee productivity. The result for employees is a situation of "high demands/low control," which has been shown to be related to cardiovascular disease and overall mortality risks (Karasek and Theorell, 1990).

The Initial 2- to 3-Year Lag of Mortality Following Economic Recessions

The initial elevation of overall mortality rates, including the major causes of death, occurs at approximately two to three years following the recession. This point of elevated mortality is then typically followed by continually heightened mortality rates for at least the next decade. Both of these phenomena are related to the initial impact of the recession.

The basis of the 2- to 3-year lag is as follows. The recession itself is the first phase of loss; the individual's response to unemployment is mental depression and probably the beginnings of other serious physiological disturbances related to chronic diseases. This is a phase of cataclysmic stress, affecting large numbers of people (thus equated with a "disaster") such that the loss of job and income is part of a national or regional phenomenon. These conditions foster social support for the affected individual.

This first phase, however, is not the critical point in the "stressful life event" associated with recession. The most important issue is whether the individual will recover his or her economic and social status, or will undergo a permanent loss. This is decided mainly during the economic recovery. At the peak of the business cycle, occurring about 2–3 years after the recession, a social distance has arisen between individuals who have not recovered and those who have. Since the highest level of employment occurs at this point, the individual who has not economically reintegrated will lose much of the social support received during the recession itself. Also at the peak of the business cycle, the economy is beginning to slow at a time of considerable overoptimism about the economic future. This is a time of great frustration, when it becomes clear that heightened expectations will be unfulfilled—which is especially acute for the individual who has not yet fully recovered from the effects of the preceding recession, but is still desperately hoping to do so.

Thus, at the peak of the business cycle, three states prevail: a maximum economic inequality between those who have recovered from the recession and those who have not; uncertainty about the economic future; and frustration over the failure to achieve the hoped for continuing recovery. All of these are particularly acute in those who suffered loss of employment and income during the preceding recession.

Macroeconomic Change and Mortality: The Economic-Change Model of Population Health

As we have seen, mortality rates in many Western industrialized societies are typically related inversely to economic growth for the population as a whole and for nearly all age groups. Also, short- to medium-term increases in mortality rates tend to be inversely related to economic recessions, with an average lag of 2–3 years; this early peak heralds the start of a long-term effect of recession, lasting 10–15 years. Epidemiological evidence based on individual-level research, and extrapolation from it to national levels via epidemic models (Doll, 1987) and diffusion theory (Rogers and Shoemaker, 1971), support the view that the major consumption-and-production risk factors (alcohol, tobacco, fats, chemical production) account for much of the hyperbolic shape of mortality trends since the Second World War. Finally, national policies providing for transfer payments (especially those for Old Age, Survivors, Disability and Health Insurance (OASDHI), education and welfare) and health-care expenditures tend to significantly modify mortality trends.

The "economic-change model of population health" helps to organize the empirical data expressing these and other variables in such a way that they can be used jointly to account for changes in mortality patterns. The economic change model (e.g., Brenner 1984a, b) includes measures of the following concepts: economic growth, economic instability (including recession), economic inequality, maladaptive responses to economic growth (excessive consumption of alcohol, tobacco, fat), chemical production, social-isolation measures such as divorce rates, and random "shocks" not predictable by other factors in the model (epidemics, abrupt population movements, military conflict, and high-impact medical technology). The final output of an analysis using this model will bring together, in the same multivariate equation, variables expressing each (or at least several) of these benefit and risk factors; relations for each variable will then cover a distributed-lag period involving at least a decade.

We begin the analysis by identifying the simplest short-term relationships for a single variable; we then examine the cumulative impact of the same variable over multiple years (distributed lag), and finally show the relations of multiple variables in a "full" time-series multiple-regression model.

The Box-Jenkins technique (Box and Jenkins, 1982) of time-series analysis, using autoregressive and moving-average terms to fit (and thereby forecast) basic trends and cycles in mortality, is useful for analyzing short-term relations. Our analysis asks, for example, whether changes in the unemployment rate, as a basic cyclic economic indicator, will help explain changes in mortality trends after the autoregressive and moving-average components of these trends have been accounted for. Previous research indicates that mortality from all causes and from major chronic diseases reaches a first peak of increase approximately 2–3 years following the bottom of a recession, as measured by the highest point in the unemployment rate (e.g., Brenner 1971a, b, 1980a, 1984b, 1987a). This is apparent in our analysis of age-adjusted mortality, both total mortality and mortality for specific sex-race groupings (see Table 7–1). For white males, a significant positive relationship between the unemployment rate and mortality is observed after a 2-year lag. For white females and nonwhite males and females, the positive relations for unemployment can be seen at 2 and 3 years.

Because, on average, economic cycles tend to be 4–5 years in length (Mitchell, 1951), this classic 2- to 3-year lag in mortality after the peak in the unemployment rate means that the first peak in mortality following recession approximately coincides with the subsequent peak in the business cycle—that is, the peak of "recovery" or "expansion." Given this observation, one should also note that the zero-lag relationship between unemployment and mortality rates is actually inverse. The *first* peak in the lagged mortality rate at 2–3 years after recession is not accidental: the delayed impact of economic distress interacts with chronic-disease mortality in such a way that the first wave of increased mortality peaks at about the same time as growth in the economic cycle. The explanation for this phenomenon involves three different populations that experience considerable loss due to recession.

The first population will have experienced loss of employment and income during the recession, and these losses may have been compounded by illness. Illness, in turn, reduces the likelihood of rapid reentry into the labor force, and substantially decreases productivity if reemployed. Over time, therefore, this population will at least remain at a lower economic level than prior to the recession, and may have a permanently decreased economic status. Meanwhile, with the subsequent expansion of employment and income, the bulk of the population will have found its economic position significantly advanced. Between the bottom of the recession and the peak of the upturn, therefore, the differences in economic position between the majority and this minority (as a specific cohort) will reach a maximum of inequality. The minority population's ensuing relative deprivation and social isolation will then expose it to its highest risk of psychological distress during the entire economic cycle.

A second population seriously harmed by recession consists of employees of firms in vulnerable industries. As we have seen, these firms frequently have difficulty adjusting to the subsequent economic upturn, due to a severe shortage of capital and the "signal-extraction problem." These difficulties result in minimal hiring despite increased market demand. Understaffing then calls for high productivity from existing employ-

Table 7-1 Box-Jenkins Model of Short-Term Time-Series Relations Between Unemployment Rate and Age-Adjusted Mortality Rate, by Race, United States 1947–1988

(Second Differences, 2- and 3-Year Lags)[a]

Variable	Lag (Years)	Coefficient	Standard Error	T Statistic
White Males				
Unemployment Rate	2	.443E-01	.151E-01	2.935
AR	1	−.525	.148	−3.542
MA	1	−.514	.163	−3.156
RBAR Sq. .592;				
Q(18) = 4.714				
White Females				
Unemployment Rate	2	.171E-01	.929	1.836
AR	1	−.915	.139	−6.563
AR	2	−.491	.139	−3.529
RBAR Sq. .480;				
Q(18) = 13.353				
Unemployment Rate	3	.165E-01	.933E-02	1.778
AR	1	−.824	.133	−6.154
AR	2	−.535	.139	−3.841
RBAR Sq. .485;				
Q(18) = 15.489				
Nonwhite Males				
Unemployment Rate	2	.733E-01	.387E-01	1.896
AR	1	−.734	.143	−5.139
AR	2	−.398	.149	−2.673
RBAR Sq. .387;				
Q(18) = 17.051				
Unemployment Rate	3	.980E-01	.326E-01	3.004
AR	1	−.774	.130	−5.957
AR	2	−.548	.130	−4.215
RBAR Sq. .447;				
Q(18) = 12.386				
Nonwhite Females				
Unemployment Rate	2	.593E-01	.259E-01	2.291
AR	1	−.751	.150	−5.012
AR	2	−.357	.151	−2.365
RBAR Sq. .322;				
Q(18) = 15.328				
Unemployment Rate	3	.706E-01	.243E-01	2.906
AR	1	−.744	.146	−5.102
AR	2	−.449	.149	−3.011
REAR Sq. .370;				
Q(18) = 10.771				

a. For white males, only the 2-year lag model.

Abbreviations: AR, autoregression term; MA, moving average; E−01, exponent to the power −.01 (e.g., .443 E−01 = .0443); RBAR Sq., adjusted coefficient of determination (i.e. adjusted explanation of variance); Q, measure of autocorrelation of residuals used in Box-Jenkins model.

Sources: Mortality data from the National Center for Health Statistics; employment data from the U.S. Bureau of Labor Statistics.

ees—a classic case of the "high-demand/low-control" situation that is gaining attention in the literature on work stress and cardiovascular disease (Karasek and Theorell, 1990). This situation would also lead to higher accident rates and, in some groups (especially the ill and impaired), feelings of hopelessness-helplessness prompted by inability to cope with severe work demands.

A third population combines features of the first two. This population loses employment and income during a recession, and is rehired possibly at a lower socioeconomic level only after economic recovery and often by firms that themselves suffered serious financial reverses during the recession. This population too may carry an additional burden of illness. That illness may have been further exacerbated in the case of the United States by loss of access to health care due to loss of income or insurance during the recession. When this ill population finds new employment during a recovery, it probably enters the economy at a time when demand for labor is high and work pressures are therefore extreme. This group, grateful to have found employment, is desperate to retain that employment despite the conflict between work pressure and illness. But if work pressure exacerbates the illness (such as cardiovascular symptoms), the individual will either be forced to quit or to work at a less intensive pace despite the requirements of the firm. In either case, inability to function "adequately" in the face of such pressure could lead to a sense of dejection and ultimately hopelessness. There is evidence that a psychological state of hopelessness-helplessness is an important factor in fatality due to chronic disease (Karasek and Theorell, 1990).

It is only the first peak in mortality, however, that occurs 2–3 years after recession. Mortality rates lag behind such economic indices as unemployment and business-failure rates by 10–15 years. To demonstrate this relationship, let us look again at the unemployment rate in its distributed-lag relation to mortality for the total population (see Tables 7–2 and 7–3). As these distributed-lag equations show, the basic cumulative temporal pattern can be measured over 2–13 years. The Shiller distributed-lag technique (Shiller, 1973) is used because it eliminates the need for a mathematically rigid functional form (as compared to the polynomial distributed lag), and because the curve-fitting method for the full distributed lag requires the expenditure of only one degree of freedom. The resulting relations, modeled in second differences (rate of change), can also be transformed to first differences (annual changes) and levels, with all variables retaining statistical significance (see Table 7–3). The statistical relationships originally modeled with both independent and dependent variables in second difference form indicate that the level of mortality rates is influenced by the level of cyclical and structural changes in the national economy.

The addition to this equation of the unemployment rate at zero-lag results in a significant distributed-lag relationship, but no longer a significant zero-lag relationship. This finding further confirms the view that, in chronic-disease mortality and in mortality from all causes, the basic short-term relation between unemployment and mortality rates peaks at a 2- to 3-year lag.

Now let us look at whether the relationship between unemployment and mortality-rate changes is still apparent after we control for the influences of other economic vari-

Table 7-2 Time-Series Regression Equation Showing Relations Between Economic and Consumption Variables and Age-Adjusted Mortality Rates for the United States, 1950–1988

(Second Differences, Shiller Distributed Lag)

Order	Economic Change	Lag (Years	Coefficient	Standard Error	T Statistic
		1950–1988			
1	Unemployment rate	2–13	.290	.569E-01	5.094
2	Per capita real disposable income	2–14	−.294E-02	.475E-03	−6.183
3	Per capita fat consumption	0–14	.152E-02	.255E-03	5.979
4	RHO		−.628	.131	−4.784
RBAR Sq. .885					
Durbin-Watson 1.941					
F Chow (3, 31) = .111					
1950–1968					
1	Unemployment rate	2–13	.240	.793E-01	3.031
2	Per capita real disposable income	2–14	−.327E-02	.662E-03	−4.941
3	Per capita fat consumption	0–14	.209E-02	.414E-03	5.044
4	RHO		−.497	.270	−1.843
1969–1988					
1	Unemployment rate	2–13	.263	.121E-01	2.168
2	Per capita real disposable income	2–14	−.305E-02	.642E-03	−4.749
3	Per capita fat consumption	0–14	.106E-02	.246E-03	3.051
4	RHO		−.822	.138	−5.965

Abbreviations: RHO, autocorrelation term; RBAR Sq., adjusted coefficient of determination (adjusted explanation of variance); Durbin-Watson, measure of autocorrelation among regression residuals; F Chow, F statistic for the Chow test of lack of equivalence of regression coefficients between the first (1950–1968) and second (1969–1988) halves of the period over which the regression equation is calculated; E−01, E−02, etc. exponents to the power −.01, −.001, etc (see footnote to Table 7-1).

Sources: U.S. Government data: unemployment rates, *Handbook of Labour Statistics*; per capita real disposable income, Bureau of Economic Analysis; fat consumption, Agricultural Statistics.

ables (such as per capita disposable income) and high-risk consumption and production factors.

First, we can observe the relation between unemployment and mortality rates, in two- or three-variable distributed lag models, including the long-term cumulative relation between per capita disposable income and a single biochemical risk factor such as fat consumption per capita (see Table 7–4).

Second, the Shiller distributed-lag procedure enables us to estimate the simultaneous impact of several predictors, including unemployment and wage rates, social-welfare and health-care expenditures, and consumption and production risk factors. These variables are entered into a step-wise multiple-regression time-series equation, again with

Table 7-3 Time-Series Regression Equation Showing Relations Between Economic and Consumption Variables and Age-Adjusted Mortality Rates[a] United States, 1950–1988

Order	Economic Change	Lag (Years)	Coefficient	Standard Error	T Statistic
(First Differences, Shiller Distributed Lag)					
1	Trend	0	−.559	.194E-02	−2.877
2	Unemployment rate	2–13	.294	.797E-01	3.687
3	Per capita real disposable income	2–14	−.295E-02	.485E-03	−6.078
4	Per capita fat consumption	0–14	.149E-02	.343E-03	4.343
5	RHO		.548	.150	3.668
RBAR Sq. .650					
Durbin-Watson 2.553					
F(4, 34) = 38.800					
Level Equation (Shiller Distributed Lag).					
1	Trend	0	−.250	.726E-01	−3.440
2	Trend square	0	.354E-02	.124E-02	2.832
3	Unemployment rate	2–13	.273	.790E-01	3.463
2	Per capita real disposable income	2–14	−.159E-02	.387E-03	−4.098
3	Per capita fat consumption	0–14	.130E-02	.127E-03	10.225
4	RHO		−.914	.104	−8.795
RBAR Sq. .998					
Durbin-Watson 1.889					
F(5, 33) = 701.490					

a. These models are algebraically transformed from the second-difference model in Table 7-2.

Abbreviations: See Tables 7-1 and 7-2.

Sources: As in Table 7-2.

a hypothesized lag of approximately 0–14 years. For total mortality rates and for the sex-race breakdown, the basic relationship can be initially estimated in second differences. (The reason for the initial second-difference modeling is that it permits necessary controls for multicollinearity, long-term linear, and quadratic trends.) It is also feasible to observe each of these sets of multivariate relations in first-difference and in level (undifferenced) form.

In the case of the level relationships, we hypothesize that the principal long-term linear trend is central both to the rate of productivity that gives rise to the standard of living and to the production of scientific knowledge, which ultimately leads to the diminution in consumption and production of factors found to be health risks. We suggest that the "factor" behind this trend is long-term investment in research and development, financed by both private sources and government. We do not yet understand, however, the empirical basis of the (relatively weak) quadratic trend that appears to increase the mortality rate. Apart from these two long-term trends, the remainder of the model remains intact, with its full set of benefit and risk factors, each variable showing statistical significance.

Table 7-4 Time-Series Regression Equation Showing Relations Between National Economic Changes and Age-Adjusted Mortality Rates[a] United States, 1950–1988

Order	Economic Changes	Lag (Years)	Coefficient	Standard Error	T Statistic
Economy					
4	Per capita real disposable income	1–14	−.841E-02	.346E-03	−24.303
7	Annual change in wage rate	1–12	−.361E-01	.593E-02	−6.090
9	Unemployment rate	0–10	.982E-01	.234E-01	4.193
11	Business-failure rate	1–7	.103E-01	.193	5.293
Policy					
14	Health Expenditures as a proportion of GNP	0–8	−24.298	11.660	−2.083
Consumption					
10	Per capita fat consumption	0–14	.203E-02	.252E-03	8.075
12	Per capita cigarette consumption	2–10	.778E-03	.240E-03	3.244
5	Per capita spirits consumption	0–13	7.648	.391	19.585
6	Per capita energy consumption	0–8	.120E-01	.575E-02	4.651
Production					
8	Per capita chemical production	0–11	.796E-01	120E-01	6.614
1	Constant		19.182	3.917	4.897
2	Linear trend		429.776	35.290	−12.178
3	Trend sq.		.206E-01	.158E-02	−13.022
13	Marriage rate	0–9	−.350	.329E-01	−10.621
RBAR Sq. .99947					
Durbin-Watson 1.840					
F(14, 25) = 33914.5					

a. These relations between *levels* of mortality (rates) and levels of economic and social variables were originally estimated in second differences, and are easily transformable algebraically into first differences (annual changes) as well.

Abbreviations: See Tables 7-1 and 7-2.

Sources: U.S. Government data: per capita disposable income, Bureau of Economic Analysis; unemployment rates, *Handbook of Labour Statistics*; business-failure rates and health expenditures, Historical Statistics of the U.S.—Statistical Abstracts; cigarette and spirits consumption, Statistical Abstracts of the U.S.

When this method of applying (1) Box-Jenkins, (2) single-variable distributed-lag, and (3) multivariate distributed-lag approaches was used to analyze overall mortality by age, sex, and major cause of death (total cardiovascular disease, coronary heart disease, malignancies, cirrhosis, suicide, and homicide) the overall findings were as follows:

1. Economic growth, as measured by real per capita disposable income (and/or by real wage rates), is inversely related to overall (and chronic-disease) mortality rates over at least 1–14 years.
2. Economic recession, as measured by the unemployment rate, and/or by the business-failure rate, i.e., bankruptcy, is positively related to mortality over 0–10 and 1–7 years, respectively. The initial peak of this relation is an elevated mortality rate 2–3 years after the recession's peak.

3. During the recessional year itself, however, overall mortality declines, followed by a sharp increase (with no lag) during the initial year of recovery. Important exceptions include suicide, homicide, and other mental-health indices and infant mortality, whose positive relation to unemployment rates *begins* at the zero year.
4. Rates of consumption of fats, cigarettes, and alcohol are positively related to total cardiovascular and cancer mortality-rate increases over at least 0–14 years.
5. Chemical production is related to increased overall mortality, and specifically to that due to malignancies, over at least 0–14 years.
6. Social-welfare and health-care expenditures per capita are inversely related to overall mortality rates over at least 0–8 years. These relations are statistically significant only after the variables representing economic change and high-risk consumption patterns are held constant.

These findings have been replicated in analyses in other countries and over different time periods.

Conclusion

The mechanisms that link SES to health also link changes in SES to changes in health. Thus, stable and equitably distributed economic growth brings about material gains (improved nutrition, sanitation, injury control, health-care technology, and access to health care), and increased potential for control of biochemical risks as a result of investments in health sciences and public education. Psychologically, stable economic growth increases self-esteem (via achievement), security, mastery, and social integration. Recession brings about the opposite effects—notably psychophysiological reactions to economic threats and losses and increased inequality—and tends to increase work stress in firms that have experienced severe economic loss.

During the recovery phase of the business cycle, illness induced by the stresses of recession may prevent reintegration of recession-displaced workers. This lack of economic recovery may be the greatest source of recession-induced health problems in some populations, combining as it does absolute loss, downward mobility, relative deprivation in relation to the majority, and probable loss of social support from peers. In combination with family instability, this set of circumstances serves to elevate the mortality rate over at least a decade following recession.

While the economy is instrumental in advancing the population's health, it is clear from the findings of this study that it does not do so through market factors alone. Market forces, in fact, often generate considerable morbidity and mortality by promoting high-risk consumption and production. Investment in the health sciences, public-health knowledge, and regulation are crucial to counterbalance these tendencies.

Trends in economic productivity and inequality have been adverse for at least the last decade and a half. Action is obviously needed to remedy the short- and long-term problems of the U.S. economy—especially relatively low investment, wage stagnation, and poverty. Perhaps debate on economic and social-welfare policy would become more pointed if it were clearly understood that the health and life expectancy of the nation are at stake.

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Class, Work, and Health

JEFFREY V. JOHNSON and ELLEN M. HALL

The inquiry into the interconnections of work, social class, and human well-being h
been a basic theme in social science since the advent of the industrial revolution in
nineteenth century. Interpreting these complex interrelationships remains a challe
however.

The association between lower social class position and adverse physical and me
health (Hollingshead and Redlich, 1958; Dohrenwend and Dohrenwend, 1969)
been reported in the United States (Antonovsky, 1967, 1968; Kitagawa and Hau
1973; Haan et al., 1987, 1989; Williams, 1990), in Great Britain (Marmot et al., 19
1987, 1991; Marmot and McDowall, 1986; Carr-Hill, 1989, Smith, 1990) and in Sc
dinavia (Vagero and Lundberg, 1989; Vagero, 1991).

Social class is a powerful determinant of a multitude of factors that affect health:
cess to social resources, such as medical care and adequate housing; the nature of
physical and urban environment; and individual resources, such as income and edu
tion, that reflect differential opportunities. Exposure to hazards and stresses of d
life are also differentially distributed along social-class lines. Those at the lower en
the social-class hierarchy are more likely to be exposed to chemical and physical h
ards in their work, to be threatened with unemployment, and to have to contend wi
deteriorating urban physical and social environment. Indeed, it has been suggested
lower social-class position is characterized by meager social and individual resour
in combination with high levels of daily stress, and that it is this imbalance, or str
that ultimately explains the higher morbidity and mortality rates of less-advantaged
cial-class groups (Haan et al., 1989).

It can be argued that social class is the primary determinant of exposure to adve
factors; however, the mechanisms by which class might influence health remain unc
specified. Biomedical researchers commonly employ three measures of social sta
that have been found to be predictive of adverse health outcomes: income, educati

Testimony of Cathy S. Woollums
Senior Vice President and Chief Environmental Counsel
MidAmerican Energy Holdings Company
Committee on Environment and Public Works
United States Senate
June 15, 2011

Thank you for the opportunity to testify today to provide you with one electric utility's perspective on the costs to comply with new Environmental Protection Agency ("EPA") regulations. My name is Cathy Woollums, and I am the senior vice president and chief environmental counsel of MidAmerican Energy Holdings Company. My comments today are not meant to represent the industry as a whole, although I believe our experiences are largely consistent with those of other U.S. electric utilities, almost all of which have spent – and continue to spend – considerable dollars and resources in planning to comply with these EPA regulations. Every utility, of course, is implementing its own unique compliance strategy based on myriad factors, including its resource base, system impacts, reliability, capital costs, operating and maintenance costs, age of its existing generation units, cost of replacement generation, and projected load growth. What I hope to do this morning is to give you a sense of how these factors translate into our utility operations' overall compliance costs.

Background on MidAmerican

MidAmerican Energy Holdings Company ("MidAmerican") is a global energy services provider serving almost 6.9 million customers worldwide. MidAmerican's five U.S. energy business platforms consist of two electric utilities, two natural gas pipelines and an independent power producer. The two regulated utilities are MidAmerican Energy Company, an Iowa-based utility providing regulated electric and natural gas service to customers in Iowa, Illinois, South Dakota, and Nebraska; and PacifiCorp, which operates as Pacific Power in Northern California, Oregon, and Washington, and as Rocky Mountain Power in Idaho, Utah, and Wyoming. The two interstate pipelines are Kern River Gas Transmission Company, providing natural gas transportation from Wyoming to Southern California; and Northern Natural Gas, which operates from Texas to the Upper Midwest. The fifth platform is CalEnergy, an independent power producer with geothermal facilities in California and cogeneration plants in New York, Arizona, Texas, and Illinois.

At the end of 2010, MidAmerican Energy Company had 7,048 megawatts of owned and contracted generating capacity. Approximately 52 percent was fueled by coal; 21 percent by natural gas and oil; 20 percent by wind, hydroelectric and biomass; and 7 percent by nuclear. PacifiCorp's generating plants have a net owned capacity of 10,623 megawatts. The company operates 78 generating facilities across the West. Approximately 58 percent was fueled by coal; 21 percent by natural gas; and 21 percent by wind, hydroelectric, geothermal, or other.

MidAmerican Energy Company and PacifiCorp are number one and number two, respectively, in the U.S. in ownership of wind-powered generation among rate-regulated utilities. As of December 31, 2010, nearly 20 percent of MidAmerican Energy Company's total owned and contracted generation capacity and nearly 12 percent of PacifiCorp's total owned and contracted

generation capacity was powered by wind. When MidAmerican Energy Company's 593 megawatts of wind capacity expansion in Iowa is complete by year-end 2011, approximately 26 percent of its total owned and contracted generation capacity will come from wind.

CalEnergy operates 10 geothermal plants with a cumulative generation capacity of 327 megawatts in California's Imperial Valley. Expansion plans call for six new plants with a total of 470 megawatts of additional geothermal capacity.

SECTION I

I. MidAmerican's Environmental Control Investments

MidAmerican has undertaken significant efforts with our permitting and regulatory agencies to ensure that our environmental control investments are timely in order to ensure compliance with existing environmental requirements, that they proceed in a reasoned fashion, and that they are coordinated with existing outage schedules to avoid additional outage time associated with equipment tie-in. These coordinated efforts reduce costs associated with replacement power and maintain system reliability.

MidAmerican has made substantial investments in pollution control equipment over the past 10 years and has budgeted for additional pollution control projects in the next 10-12 years. We began planning emission control projects targeting sulfur dioxide ("SO_2"), nitrogen oxide ("NOx"), and mercury emissions prior to 2005, when the EPA was developing its Clean Air Interstate Rule ("CAIR") and Clean Air Mercury Rule ("CAMR"). Both rules were ultimately vacated by the D.C. Circuit Court of Appeals, which directed the Agency to rework the regulatory framework underpinning both rules. Ultimately the CAIR was replaced by the Clean Air Transport Rule ("CATR") and the CAMR by the Utility Hazardous Air Pollutants ("HAPS") Maximum Achievable Control Technology ("MACT") rule. While the EPA was reworking these rules, MidAmerican continued planning various emissions control projects. Section II of this document contains a rule-by-rule overview and brief explanation of MidAmerican's compliance strategy.

Through 2010, our Midwest utility, MidAmerican Energy Company, has spent more than $370 million in capital expenditures for required pollution control equipment under these EPA rules. We estimate that the total costs for all pollution control projects (defined as capital, operations and maintenance and other costs) will exceed $1.1 billion by the end of 2020. These total costs are expected to increase annual costs to customers by $130 million per year by 2020.

Our other utility, PacifiCorp, has spent more than $1.2 billion in capital expenditures from 2005 through 2010 to comply with these EPA rules, and we estimate that total capital expenditures will exceed $2.7 billion by the end of 2022. Total costs that will have been incurred by our customers to pay for these pollution control projects during the period 2005 through 2023 are expected to exceed $4.2 billion, and by 2023 the annual costs to customers for these projects will have reached $360 million per year.

It is very difficult at this point to translate these projected costs to comply with the new EPA rules into specific percentage rate increases to our customers in all ten states in which we are subject to state public utility commission regulation, but let me give you one metric to demonstrate the magnitude of these costs. PacifiCorp's fossil steam generation units currently have a cumulative net value (after depreciation) of approximately $3.38 billion. Just compare that current value – $3.38 billion – to the estimated $1.3 billion in additional environmental control project capital costs PacifiCorp will spend between now and 2022, and that gives you a relative sense of the cost of these emissions control devices to our customers.

Due to the large number of our generating units that will be potentially affected by these new EPA regulations, deferring the installation of compliance projects places MidAmerican and our customers at risk of not having access to necessary capital, material, and labor in a compressed time frame concurrent with other utilities. For example, in the eastern United States, utilities are required to install controls under the CATR during the same 2012-2014 time frame within which they are required to comply with the HAPS MACT rule. We have already seen a dramatic rise in these pollution control costs in anticipation of the increased demand for labor and equipment. For example, MidAmerican Energy Company has just negotiated a contract for the installation of scrubbers and baghouses at two of our facilities in 2013 and 2014, and the costs are approximately 20% higher than anticipated. We have no choice, however, but to move forward, in order to ensure that we are in compliance and not subject to penalties for noncompliance or third party lawsuits.

The Department of Energy[1] estimates that between 35-70 gigawatts will shut down nationwide as a result of EPA's new rules. Similarly, a recent study by National Economic Research Associates ("NERA") estimates that 47.8 gigawatts of coal-fueled electricity capacity will likely become uneconomic and retire by 2015. Some of those facilities are also located in key transmission grid areas that provide voltage support that cannot be addressed by the fall of 2014 in order to comply with the anticipated January 1, 2015 implementation date. According to four other independent studies conducted last fall, with which I am sure the Committee is familiar (North American Electricity Reliability Council, Brattle, Credit Suisse, and Sanford Bernstein), this aggressive schedule for implementation of these and other EPA rules will likely result in closures of up to 60 gigawatts of existing U.S. coal capacity by January 2015.

MidAmerican, like many utilities, is concerned about the costs and timetables for the implementation of these EPA rules. These compliance costs will increase rates to our customers at the same time as they see increased rates for other major capital expenditures for new generation to meet increasing demands for electric service and to further diversify our generation portfolios, as well as construct billions of dollars of transmission to be able to deliver energy where it is needed. These rate increases are already occurring at PacifiCorp, with customers seeing annual rate increases, some in double-digit percentages.

Especially in this economic climate, it is critical to minimize the cost impact of these rules, which ultimately will be borne by our customers. If the timetable of the rules remains unchanged, compliance costs will be shouldered by our customers in the form of higher rates in a

[1] "EPA regulations for coal-fired power plants could force shut downs", *Bristol Herald Courier* (May 27, 2011); quoting James Wood, deputy assistant secretary for the U.S. Department of Energy.

very narrow window from 2013-2015. These increases will dramatically increase production costs for industrial plants and could result in job losses. Also, units prematurely retired in response to these EPA rules will have remaining book value issues to address.

Moreover, forcing all U.S. coal plants to comply with these EPA rules during such a short time frame will cause the costs of labor and materials for both retrofits and new generation to rise dramatically as demand for skilled labor and parts will greatly outstrip supply. A boom and bust cycle of craft labor employment created by these proposed EPA deadlines will make it challenging for firms to find, train, and retain skilled domestic craft labor.

II. MidAmerican's Environmental Compliance Planning Process

First and foremost in the decision to invest in environmental controls is our compliance obligation. If a permit or regulation requires one of our plants to reduce emissions or achieve emission limits that cannot be met with existing equipment, we examine compliance options to ascertain what equipment can be installed to achieve the emission requirements. MidAmerican also monitors state and federal rulemaking activities and legislative proposals that would have an impact on the facilities' operations. Monitoring these future requirements gives us a longer term view of the potential investments that may be required to lawfully continue operation of the facilities.

To assess the potential impacts of new environmental regulatory initiatives, the environmental groups in our business units review proposed and final regulatory requirements and actively engage in the regulatory processes at both the state and at the federal levels. We seek feedback from our environmental regulators to assess their concerns, read and analyze legislation and regulations proposed at the state and federal levels, provide feedback on legislation, and review and comment on proposed regulations. We submit written comments in regulatory proceedings and participate in public hearings on the proposals, ensuring that our concerns or support, as appropriate, are considered in these public forums. We are both well informed and engaged on these issues.

III. Compliance and Project Timing Considerations

We, like virtually all other electric utilities, examine a multitude of factors to determine the appropriate mitigation measures. For example, if a regulation prescribes a specific emissions limit, our teams review what types of controls may be available to achieve the requisite emissions limit, given the specific characteristics of each unit. We consider system impacts, reliability, capital costs, operating and maintenance costs, the life of the controls, the life of the unit itself, cost of replacement generation, and many other factors. If an emissions trading mechanism is available to achieve compliance, we compare the costs of obtaining the emissions allowances to the costs of installing and operating new equipment, considering the factors noted above.

We also examine the actual and potential compliance time frames and how those time frames may be coordinated with planned plant outage schedules. Coordinating major environmental control projects with existing outage schedules allows MidAmerican to avoid additional outage

time, thus reducing the need for replacement power, minimizing costs, and maintaining system reliability.

Pollution control projects are extremely complex and require a significant amount of evaluation and planning to bring to fruition. Moreover, state environmental agency permitting processes are required to define the technical requirements needed in order to seek competitive bidding and pricing for the work and ultimately executing the projects. The timeline for securing contracts for this type of work through project completion often has a multi-year duration.

IV. Managing Project Execution and Compliance Risk

The full and final scope of environmental regulations is not easily determined, particularly when rulemakings are often lengthy in their own right and just as often followed by extensive and lengthy litigation before the rule is finalized. Perfect foresight is not possible; the EPA has recently begun to acknowledge that its approach to regulation makes it difficult for companies with compliance obligations to make long-term decisions on compliance. In EPA Administrator Lisa Jackson's remarks prepared on the release of the HAPS MACT standards on March 16, 2011, she stated:

> The proposal and implementation of these standards will also have benefits for American utilities. For the first time in twenty years, they will have certainty about the standards they must meet. And setting national standards for mercury and air toxics will level the competitive playing field and close loopholes for big polluters. Utilities that have already put pollution control technology in place will no longer have to compete with those who have delayed those investments – a group that includes almost half of the nation's coal-fired plants, which lack advanced pollution control equipment. In fact, facilities that have already taken responsible steps to reduce the release of toxins into our air will be at a competitive advantage over their heavy-polluting counterparts. And to ensure cost-effectiveness, we have proposed flexibility in meeting the standards. The technologies being required already exist in abundance, and under the proposal, power providers have four years to comply.[2]

MidAmerican believes it would be imprudent to wait until all the regulations are considered, finalized, and quantified to install controls. Doing so would put the facilities at substantial risk of noncompliance and does not reflect the reality of the multistate operations and planning process for large utilities. Moreover, it would be imprudent to assume a large utility can install all required controls under a "just-in-time" plan. This approach to compliance poses a significant risk to MidAmerican and our stakeholders; as a practical matter, it cannot be economically achieved on a system the size of MidAmerican's utility platforms. Emission reduction projects are complex, multi-year projects. Trying to install multiple controls within the same short time frames poses a significant risk of noncompliance, with penalties that can be substantial. Even if a regulatory agency did not impose penalties for failing to achieve emission reduction deadlines,

[2] Remarks available at:
http://yosemite.epa.gov/opa/admpress.nsf/12a744ff56dbff8585257590004750b6/b7e570d651cadc03852578550057011c!OpenDocument

third parties have not hesitated to bring lawsuits against the operators of those facilities that miss deadlines or are otherwise not in compliance with permit and emission limits. Indeed, the federal Clean Air Act specifically allows for private citizen enforcement of air quality requirements.

V. Other Factors to Consider

Finally, environmental regulations and the cost of implementation are only one factor that influences whether or not to make investments in environmental projects; MidAmerican also must consider the cost of alternative generation, such as small modular nuclear reactors. Future natural gas prices, construction costs for renewable generation, and associated transmission availability and costs are also among the factors we evaluate in determining whether it is economic to install controls at coal-fueled plants.

VI. The Role of State Regulators and Stakeholder Feedback

Our state regulators are the consumers' watchdogs, and they apply standards to ensure that only those costs that are prudently incurred and useful in providing service are recovered in rates. This structure does not encourage utilities to become early movers or emission control technology developers. Those responsibilities lie with the vendor community, where the market provides greater potential rewards for successful innovation. Shareholders of these unregulated companies, not utility customers, earn the rewards of success or bear the costs of failure.

Neither utilities nor regulators have perfect foresight regarding the development of future technologies, future market conditions, or changes in environmental laws, but we make the best projections possible in our resource development decisions. We also appreciate that the American public is concerned with environmental issues, including global climate change. The significant concern for electric utilities is carbon dioxide, the byproduct of the combustion of fossil fuels. Although the primary focus has been on coal-based generation, since it produces more carbon dioxide per unit of electric energy than other fossil fuels, natural gas-fired generation also produces carbon dioxide emissions and is at risk as a continuing source of fuel due to uncertainties around climate change and carbon dioxide regulations.

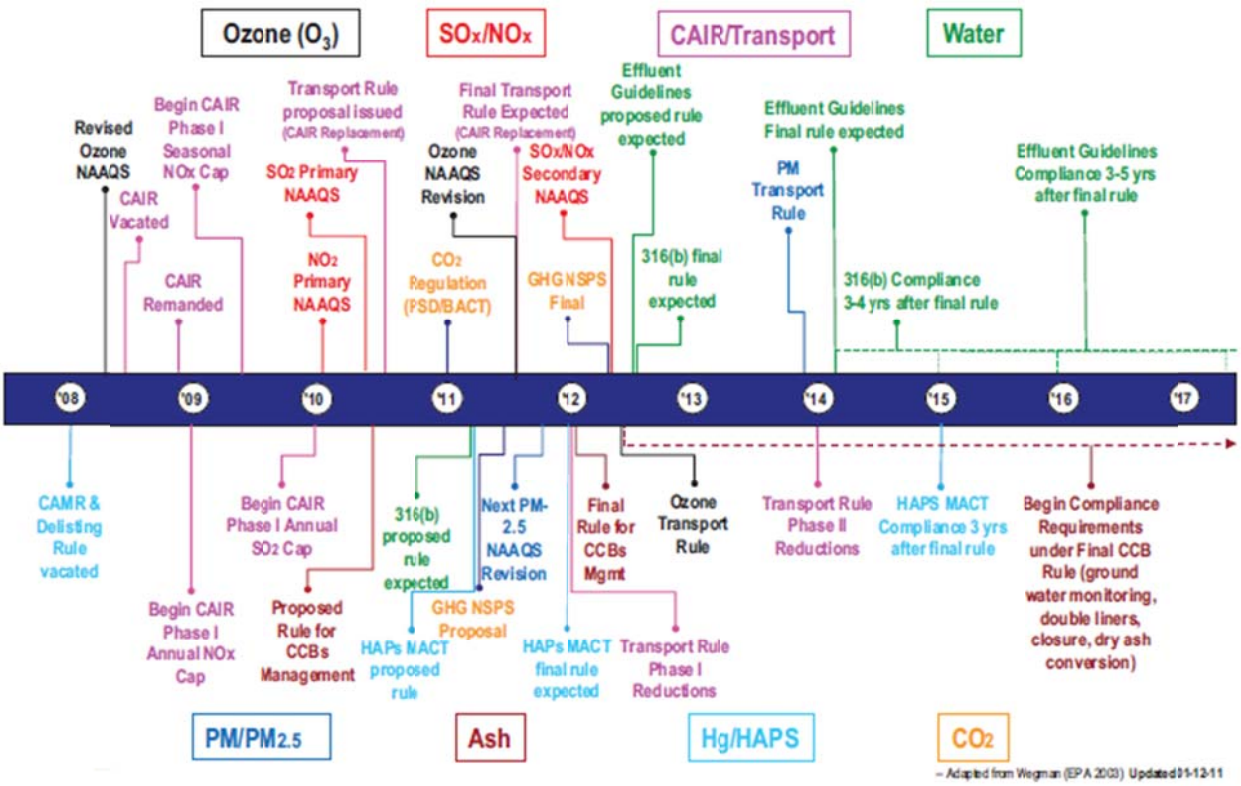
There are many different viewpoints regarding whether MidAmerican should make investments in our existing coal-fueled facilities. Our challenge is to work with these stakeholders and our regulators to come up with solutions that balance state and federal policies, ensure system reliability, maintain 100% compliance with all laws, keep the lights on, meet increasing customer loads, ensure the safety of our employees and customers, and satisfy the obligation to serve, all while maintaining reasonable rates.

SECTION II

The So-Called "EPA Regulatory Train Wreck"

Both MidAmerican Energy Company and PacifiCorp continue to pursue proactive environmental control strategies that are protective of the environment while minimizing cost impacts to our customers. There are a multitude of environmental requirements the electric industry faces over the next several years driving these investments. Figure 1 provides a timeline, referenced colloquially as the so-called "EPA regulatory train wreck" slide. It identifies some of the requirements that are currently underway or in development. There is a great deal of uncertainty associated with future environmental requirements; however, MidAmerican must comply with the requirements that exist today and prepare for the regulations that will be adopted in the future.

Figure 1 - EPA's "Regulatory Train Wreck"



The areas of regulation listed below reflect the color-coded "categories" of regulations identified within Figure 1.

1. **PM/PM2.5**
2. **Ozone (O_3)**
3. **SOx/NOx**
4. **CAIR/Transport Rule**

These first four categories are grouped together because under the Clean Air Act each of these categories is linked to one or more National Ambient Air Quality Standards ("NAAQS"). These "criteria pollutants" – particulate matter ("PM"), sulfur dioxide ("SO_2"), ozone ("O_3"), nitrogen oxides ("NOx"), carbon monoxide ("CO"), and hydrocarbons – while undesirable, are not toxic in typical concentrations in the ambient air. Under the Clean Air Act, they are regulated differently from other types of emissions, such as hazardous air pollutants and greenhouse gases.

A NAAQS by itself does not require emissions reductions from specific sources, such as power plants. Rather, the EPA and/or a state will identify various control measures that once implemented, are meant to achieve the NAAQS. A particular control measure may require emissions reductions from certain types of sources. An example of such a control measure would be the EPA's proposed Clean Air Transport Rule, discussed further below.

The Clean Air Act, which was last amended in 1990, requires the EPA to set NAAQS (40 CFR part 50) for pollutants considered harmful to public health and the environment. The Clean Air Act established two types of national air quality standards. ***Primary standards*** set limits to protect public health, including the health of "sensitive" populations such as asthmatics, children, and the elderly. ***Secondary standards*** set limits to protect public welfare, including protection against decreased visibility, damage to animals, crops, vegetation, and buildings. The Clean Air Act requires the EPA to review the latest scientific information and standards every five years. Before new standards are established, policy decisions undergo rigorous review by the scientific community, industry, public interest groups, the general public and the Clean Air Scientific Advisory Committee (CASAC).

Particulate Matter (PM) and Fine Particulates ($PM_{2.5}$): The Clean Air Act established NAAQS for particle pollution (i.e., particulate matter or "PM"). The EPA last revised the air quality standards for particle pollution in 2006. The next review is expected in 2011.

Ozone (O_3): Ozone is a gas composed of three oxygen atoms. It is not usually emitted directly into the air, but at ground-level is created by a chemical reaction between NOx and volatile organic compounds ("VOC") in the presence of sunlight. EPA last revised the NAAQS for ozone pollution in 2008 (at 75 micrograms per cubic meter), putting some counties into non-attainment and requiring states to take steps to reduce emissions to improve the ambient air concentrations. However, EPA is now reconsidering its 2008 decision and may lower the limit (to between 60 and 70 micrograms). EPA expects to make its decision by the end of July 2011.

Sulfur Dioxide (SO_2) and Nitrogen Oxide (NOx): In 2010, the EPA promulgated new "primary" one-hour NAAQS for SO_2 and nitrogen dioxide ("NO_2") concentrations, which add a temporal nature to emissions reductions necessary to improve the ambient air concentrations. New "secondary" SO_2 and NOx NAAQS are expected in 2012.

Clean Air Transport Rule ("CATR"): EPA's proposed CATR would require new reductions in SO_2 and NOx emissions from large stationary sources, including power plants, located in 31 states and the District of Columbia beginning in 2012. It is meant to help states attain NAAQS set in 1997 for ozone and fine particulate matter. This rule would replace the Bush

administration's CAIR, which was vacated in July 2008 and rescinded by a federal court because it failed to effectively address pollution from upwind states that is hampering efforts by downwind states to comply with ozone and PM NAAQS.

The EPA has been discussing the possibility of additional emissions reductions via a "PM Transport" rule (2013) or a "Transport II" rule (2014). Justification for such a rule or set of rules would be triggered by the setting of more stringent ozone or PM NAAQS. For example, a more stringent ozone NAAQS may result in an expansion of NOx emissions reduction requirements to stationary sources operating in the non-CATR states.

MidAmerican's Compliance Strategy: The Clean Air Transport Rule only impacts MidAmerican Energy Company's coal units in Iowa and CalEnergy's natural gas facilities in Texas, Illinois and New York. MidAmerican Energy Company has already completed a low NOx burner and overfire air program across its entire coal-fueled fleet. As a result, NOx emissions have dropped from approximately 40,000 tons per year to slightly over 20,000 tons per year – or nearly 50%. In addition, dry scrubbers have been installed at its Louisa and Walter Scott Energy Center unit 4 in 2007, and Walter Scott Energy Center unit 3 in 2009. Additional scrubber projects are being planned for Neal South in 2013, and Neal North units 2-3 and the Ottumwa Generating Station in 2014. Once these projects are complete, MidAmerican Energy Company's SO_2 emissions will be reduced from a baseline of over 60,000 tons per year to slightly less than 25,000 tons per year – or nearly 60%.

The EPA intends for this Rule to evolve as additional changes are made to the National Ambient Air Quality Standards for SO_2 and NOx. This could lead to significant stranded investments and cause the affected states to also expand to the western coast; if modeling shows those states ultimately contributing to a downwind attainment problem.

Regional Haze Rule: While not depicted within the EPA regulatory train wreck slide, an EPA rule meant to address visibility concerns will drive additional NOx reductions particularly from facilities operating in the Western United States. On June 15, 2005, EPA issued final amendments to its July 1999 regional haze rule. These amendments apply to the provisions of the regional haze rule that require emission controls known as Best Available Retrofit Technology ("BART"), for industrial facilities emitting air pollutants that reduce visibility. These pollutants include $PM_{2.5}$, and compounds which contribute to $PM_{2.5}$ formation, such as NOx, SO_2, certain volatile organic compounds, and ammonia. The 2005 amendments included final guidelines, known as BART guidelines, for states to use in determining which facilities must install controls and the type of controls the facilities must use. States had until December 2007 to develop their implementation plans. States were responsible for identifying the facilities that would have to reduce emissions under BART and then set BART emissions limits for those facilities. Those facilities are expected to install additional emissions controls usually within five years after the EPA approves a state's regional haze plan (2014-2017).

MidAmerican's Compliance Strategy: PacifiCorp operates 19 coal-fueled generating units; 14 of these units are BART or BART-eligible units. Between 1999 and 2014, PacifiCorp will have installed low-NOx burners at 15 units, reducing NOx emissions by 36,800 tons per year. The capital cost of these projects is $125 million; annual operating and maintenance expenses

associated with the equipment are $1.6 million. Beginning in 2014, PacifiCorp will install selective catalytic reduction ("SCR") to achieve additional NOx emission reductions. Between 2014 and 2022, five units will have SCR installed, reducing NOx emissions by 21,000 tons at a cost of $951 million; operating and maintenance costs will increase by $25.8 million annually.

Unfortunately, recent discussions with the Utah and Wyoming Departments of Environmental Quality suggest that EPA Region 8 believes it may be necessary, for purposes of Regional Haze BART requirements, to install another five SCR in Wyoming and four SCR in Utah, combined with the five planned installations, within a five-year time period—potentially requiring 14 SCR by 2017 and an additional $1.7 billion to $2 billion in costs. PacifiCorp maintains its outage schedule on a four-year cycle; major projects such as the addition of emission control require a significant outage. Installing controls during times outside of the normal outage schedule creates significant electric reliability and availability concerns and imposes significant additional costs for replacement power. The costs of controls, replacement power, and other project-related costs are reflected in increased costs to customers.

The Regional Haze program does not require that emission reductions occur on a date certain; to the contrary, the Regional Haze program is a long-term program designed to improve visibility in Class I areas with the national goal of achieving natural visibility conditions by 2064. States are required to establish reasonable progress goals to achieve the required visibility improvements. States are required, under Section 169A(b) of the Clean Air Act to consider the following when making their BART determinations:

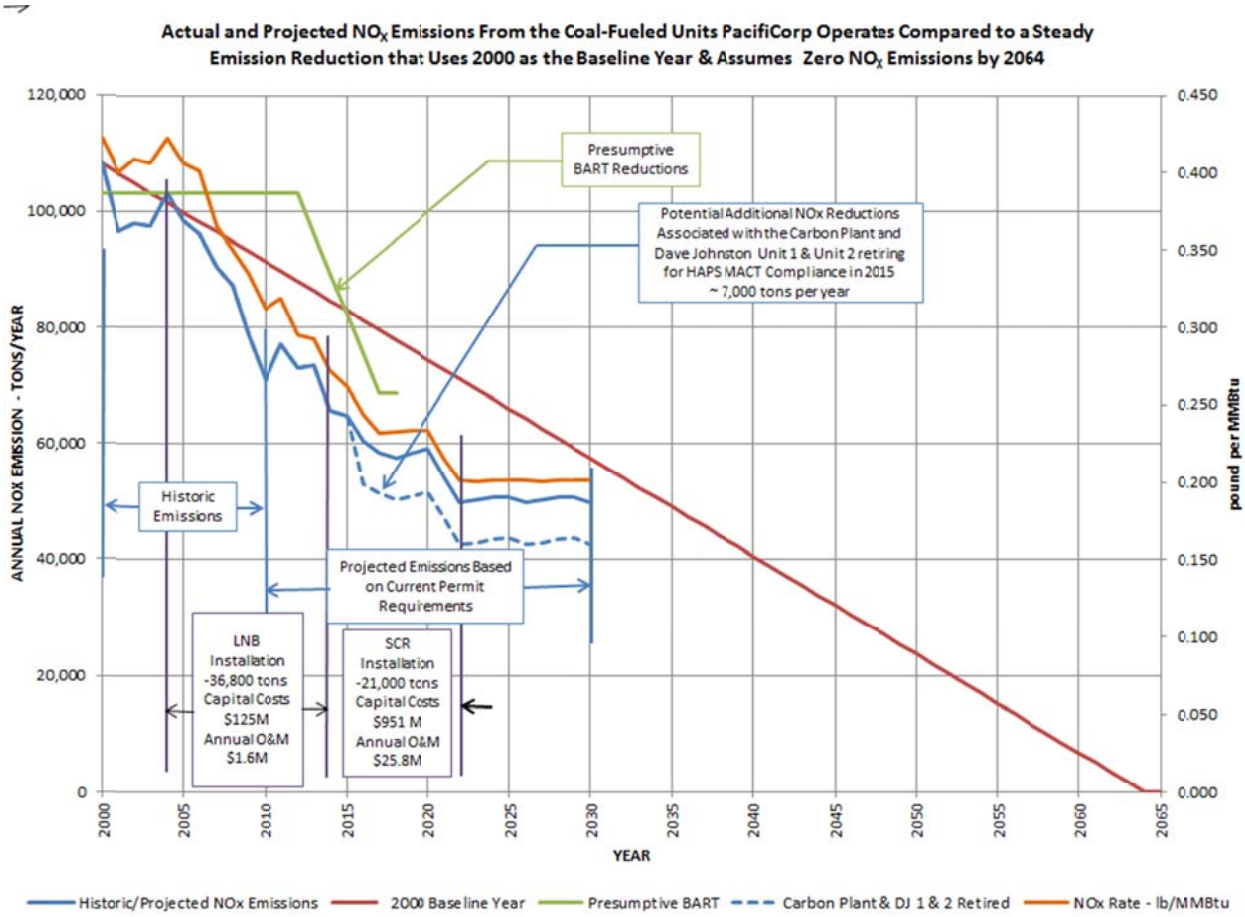
- The costs of compliance;
- The energy and non-air quality environmental impacts of compliance;
- Any existing pollution control technology in use at the source;
- The remaining useful life of the source; and
- The degree of visibility improvement which may reasonably be anticipated from the use of BART.

In considering whether the states' implementation plans are sufficient for approval, EPA appears to be focused, at best, on two criteria – the costs of compliance and the degree of visibility improvement. Effectively, EPA has indicated that any emission reductions that can be accomplished for $5,000 or less per ton at facilities that have more than a 0.50 deciview impact on a Class I area should be controlled. EPA's analysis fails to take into consideration the more robust criteria considered by the states in making their determinations, opting for more reductions sooner.

As a result of EPA's failure to take into consideration factors such as existing pollution control technology in use at the source, its cost per ton of emissions reduced is inaccurate. For example, at PacifiCorp's Jim Bridger Unit 1, low-NOx burners were installed in 2010. Rather than calculating the incremental costs associated with installation of SCR from the reduced baseline that reflects the emission reductions from low-NOx burners, EPA spreads the cost of both low-NOx burners and SCR to achieve a cost per ton removed more than $2,000 per ton lower than the incremental difference between low-NOx burners and SCR.

EPA's suggestions that it will require more emission controls in a shorter period of time is akin to jumping off a cliff, rather than achieving emission reductions in a reasonable period of time through 2064. (See Figure 2.)

Figure 2 – PacifiCorp's Regional Haze/BART Compliance Strategy



5. Hg/HAPS

In March 2005, EPA issued the Clean Air Mercury Rule ("CAMR") to permanently limit and reduce mercury emissions from coal-fired power plants under a market-based cap-and-trade program; this rule would effectively remove coal-fired power plants from the Clean Air Act list of sources of hazardous air pollutants. However, CAMR was vacated in February 2008, with the circuit court finding EPA's removal of coal-fired power plants from the list of generating sources regulated under Clean Air Act Section 112 out of statutory compliance.

On March 16, 2011, the EPA signed the proposed Utility Hazardous Air Pollutants ("HAPS") Maximum Achievable Control Technology ("MACT") rule, which sets standards for 10 non-mercury HAPS metals, mercury and acid gases. It also establishes work practices to ensure the minimization of organic HAPS such as furans and dioxins emitted by coal and oil-fueled electric generating units. The rule is standard-drive, not technology-driven and, as such, there are

multiple pathways to comply with the rule; however, it appears the EPA is encouraging utilities to: install baghouses with particulate matter continuous emission monitors for non-mercury metallic HAPS control, install sulfur dioxide scrubbers to control acid gases, and install activated carbon/reagent injection to remove mercury.

MidAmerican's Compliance Strategy: In order to meet emissions projections, MidAmerican Energy Company must complete scrubber projects planned for Neal 4 in 2013, and Neal units 2 and 3 and Ottumwa Generating Station in 2014 and add sorbent injection to Neal 1, Walter Scott Energy Center unit 1, Walter Scott Energy Center unit 2, and Riverside Generating Station. Walter Scott Energy Center unit 4 already employs an activated carbon injection system to control mercury and the remaining units with existing or planned baghouses are expected to install activated carbon injection by fall 2014. The cost of most of these projects is approximately $485 million (MidAmerican Energy Company's share). Additional activated carbon injection and sorbent injection projects at the four small coal-fueled units would require an estimated $30 million (MidAmerican Energy Company's share).

MidAmerican Energy Company's smaller coal-fueled units (Walter Scott Energy Center 1, Walter Scott Energy Center 2, Neal 1, and Riverside) may not be able to comply with the proposed HAPS MACT rule without making significant investments in control technology (unless the units are converted exclusively to fire natural gas).

For PacifiCorp, in order to meet the emission reductions anticipated by the new regulations, PacifiCorp must complete scrubber, baghouse, and mercury emissions controls projects no later than fall of 2014 in order to comply with the anticipated January 1, 2015 implementation date at a cost of approximately $1.26 billion (PacifiCorp's share). This capital cost includes installation of mercury control at all PacifiCorp units, including Carbon Unit 1 and 2 and Dave Johnston Unit 1 and Unit 2 at an estimated $12 million (PacifiCorp's share).

The units most at risk from the new HAPS MACT regulations are unscrubbed units that do not have baghouses. These units (Carbon Units 1 and 2 and Dave Johnston Units 1 and 2) may need to be idled or converted to natural gas (assuming it is available onsite) if the non-mercury metallic HAPS and acid gas HAPS limits cannot be met through dry sorbent injection, or other emergent low-cost technology solutions.

Due to the non-emission-trading nature of the proposed rule, units not meeting the unit-based HAPS MACT emission standards would be required to cease operation on or about January 1, 2015, should that date become the compliance deadline. Some of those facilities are also located in key transmission grid areas that provide voltage support that cannot be addressed by the fall of 2014 in order to comply with the anticipated January 1, 2015 implementation date. As such, we urge EPA to carefully consider potential options to develop a mechanism that avoids significant impacts to the availability, reliability and cost of electricity while balancing the need to reduce emissions.

6. Water

Cooling Water Intake Structure Rule: EPA recently released it proposed cooling water intake structure ("CWIS") rule pursuant to Clean Water Act (CWA) section 316(b) for existing steam-electric power plants. In November 2010, EPA entered into a settlement agreement with the environmental community that sets a binding timetable for a proposed rule by March 2011 and a final rule by July 2012.

MidAmerican's Compliance Strategy: All of MidAmerican Energy's coal-fueled generating facilities, except Louisa, Ottumwa and Walter Scott Unit 4, which have water cooling towers, are regulated facilities under 316(b) of the Clean Water Act and may be impacted by the outcome of the expected rulemaking. Neal 1-4, Walter Scott Energy Center 1-3, and Riverside Generating Station have once through cooling on the Missouri and Mississippi Rivers. At PacifiCorp, only the Dave Johnston plant withdraws enough cooling water to be covered by the 316(b) rule. Every other PacifiCorp facility that is potentially affected by this rule has a recirculating cooling system in place thereby meeting the likely technology requirements of the rule.

Steam Electric Effluent Guidelines: EPA announced in September 2009 that it intends to revise the existing steam electric guidelines, last updated in 1982, that set the technology-based effluent limitations for the steam electric industry. The new effluent guidelines rulemaking is likely to set strict performance standards that will force technological and operational changes at existing coal-fueled, nuclear, gas-fueled, and combined cycle facilities. The most significant impact, however, will likely be to coal-fueled facilities. The proposed rule is due in July 2012 with a final rule expected in January 2014.

MidAmerican's Compliance Strategy: MidAmerican Energy Company does not have any wet scrubbers installed in its coal-fueled fleet, and none are planned. The dry scrubbing process does not produce a significant waste water stream, as the approximate 600 gallons per minute of lime slurry water is evaporated in the process and emitted out the stack as vapor. MidAmerican, however, may face a greater challenge concerning the discharge of process water from its coal ash surface impoundments.

PacifiCorp has a number of wet scrubbers in its coal-fueled fleet which produce waste water streams. In most cases, water from these waste streams is collected and evaporated in waste water ponds. The wet scrubbers are currently installed at Hunter 1-3, Huntington 1-2, Naughton 3, Bridger 1-4, Cholla 4, Craig 1-2, and Colstrip 3-4. New wet scrubbers are planned to be placed in service at Naughton 1-2 in 2012 and 2011, respectively. In addition, the PacifiCorp coal-fueled facilities have a number of coal ash surface impoundments.

Unfortunately, there is no definitive method to ascertain the potential financial impacts of new effluent guidelines on the MidAmerican and PacifiCorp coal-fueled fleets until the actual rule requirements are proposed in mid-2012; and there are no projects budgeted to specifically address these issues. However, as the effluent discharge requirements become more and more stringent, the facilities which have discharges to waterways will likely be required to either add wastewater treatment facilities or redesign their process if possible to be a zero discharge facility. The costs to comply with such a rule are expected to be high. Wastewater treatment systems

generally range from tens of millions of dollars for a small facility, to a hundred million or more for a large facility.

7. Ash

In June 2010, EPA proposed two primary regulatory options for coal combustion residuals ("CCR") disposed of in landfills and/or surface impoundments: (1) regulation of the materials as hazardous wastes under Subtitle C of the Resource Conservation and Recovery Act ("RCRA"); or (2) regulation of the materials as non-hazardous wastes under Subtitle D of RCRA. Under both options, the proposed regulatory requirements likely would lead to the accelerated closure of all existing unlined landfills and unlined wet surface impoundments, although the agency's "D Prime" option would allow for the continued use of existing landfills and surface impoundments through their useful life as long as certain environmental and safety standards were met. Under each option, CCRs that are beneficially used would be excluded from regulation; however, the stigma associated with a hazardous waste determination would have a devastating impact on continued beneficial uses. Under the two primary options under consideration by EPA, CCR disposal practices will be impacted significantly and result in significant compliance costs, may lead to the closure of existing disposal facilities, and may threaten continued CCR beneficial use.

MidAmerican's Compliance Strategy: The regulation of CCR under either of the EPA's primary options would have a significant impact on the methods that MidAmerican Energy Company typically employs to manage its ash. With the exception of Walter Scott Unit 4 and Neal Unit 4 which handle all the coal ash dry, all of MidAmerican Energy Company's coal-fueled units sluice the boiler bottom ash to on-site surface impoundments. In addition, if CCR is ultimately designated as a hazardous waste, the beneficial use market could evaporate and eliminate the over $3 million MidAmerican Energy Company receives each year for this commodity. The loss of the beneficial use market would also increase disposal costs and dramatically increase the rate at which the monofills are filled.

Similar to MidAmerican Energy Company, the regulation of CCR under either of the EPA's primary options would have a significant impact on the methods that PacifiCorp typically employs to manage its ash. Currently, Carbon, Hunter, and Huntington do not have any wet surface impoundments at the facilities. The remaining coal-fueled units, however, sluice ash and scrubber waste to on-site surface impoundments. In addition, if CCR is ultimately designated as a hazardous waste, the beneficial use market could evaporate and eliminate the over $3.5 million PacifiCorp receives each year on average from this commodity. The loss of the beneficial use market would also increase disposal costs and dramatically increase the rate at which monofills are filled.

8. CO_2

Best Available Control Technology ("BACT") Guidelines: On November 10, 2010, the EPA published a set of guidance documents to assist state permitting authorities and industry permitting applicants with the Clean Air Act PSD and title V permitting for sources of greenhouse gases ("GHGs"). The guidance consists of a number of different documents. EPA provided a general guidance document entitled "PSD and Title V Permitting Guidance For

Greenhouse Gases," which includes a set of appendices with illustrative examples of BACT determinations for different types of facilities. There also remains ongoing concern about the application of New Source Review ("NSR") rules to GHGs. It is unclear whether owners of fossil power plants should proactively undertake efficiency improvements, lest those efficiency improvements be treated as a modification that triggers the application of NSR rules.

MidAmerican's Compliance Strategy: With respect to the GHG BACT permitting, PacifiCorp recently completed permitting for its Utah Lake Side 2 natural gas combined-cycle power plant, where the additional resources and costs required to complete the permitting effort were estimated to be between $25,000 and $50,000 for GHG-related modeling costs, consultant costs, and internal labor.

MidAmerican Energy Company recently completed its GHG BACT permitting for its George Neal South emission control project located in Iowa, but the additional work was completed internally. However, to comply with the newly proposed GHG limit, MidAmerican Energy Company demonstrated that replacing the existing turbine with a more efficient design is technically feasible and would cost approximately $20 million. We also have to test several boiler injection chemicals to determine if they improve plant efficiency. If it is determined that the chemicals are technically and economically feasible, the unit will be required to utilize them going forward.

It should also be noted, that despite claims to the contrary, there are no post-combustion technologies commercially available to control greenhouse gas emissions. Carbon capture and sequestration is likely at least 5-10 years away from becoming commercially available, and only if certain technical, legal, and liability challenges can be overcome. Additionally, the use of biomass is generally limited to certain boiler types for potential retrofit, and only a small percentage can replace the primary boiler fuel. As a result, facilities undergoing GHG BACT permitting are only left with potential efficiency upgrades / heat rate improvement projects to pursue. Since these types of projects typically result in relatively small improvements in efficiency (i.e. less than 1%-3%), an aggressive GHG BACT permit limit may not be achievable on existing units.

New Source Performance Standards: On December 23, 2010, in a settlement reached with several states and environmental groups in *New York v. EPA*, the EPA agreed to promulgate emissions standards covering GHGs from both new and existing electric generating units under Section 111 of the Clean Air Act by July 26, 2011 and issue final regulations by May 26, 2012.[3] New source performance standards are established under the Clean Air Act for certain industrial sources of emissions determined to endanger public health and welfare and must be reviewed every eight years. New source performance standards apply to new and modified sources and effectively establish the floor for determining what constitutes BACT.

In addition, emission guidelines will apply to existing sources. The emissions guidelines, issued by EPA, are used by states to develop plans for reducing emissions and include targets based on demonstrated controls, emission reductions, costs and expected time frames for installation and

[3] EPA also entered into a similar settlement the same day to address greenhouse gas emissions from refineries with proposed regulations by December 15, 2011 and final regulations by November 15, 2012.

compliance and may be less stringent than the requirements imposed on new sources. States must submit their plans to EPA within nine months after the guidelines' publication unless EPA sets a different schedule. States have the ability to apply less stringent standards or longer compliance schedules if they demonstrate that following the federal guidelines is unreasonably cost-prohibitive, physically impossible, or that there are other factors that reasonably preclude meeting the guidelines. States may also impose more stringent standards or shorter compliance schedules. Lastly, under Section 111, EPA may establish standards that rely upon market mechanisms rather than technology-specific emissions rates.

MidAmerican's Compliance Strategy: It is unclear what approach EPA will take when establishing new source performance standards covering GHGs from both new and existing electric generating units or what the guidelines will be for existing sources. The proposed settlement agreement indicates that EPA's initial evaluation of available GHG control strategies indicates that there are cost-effective control strategies for reducing GHGs from electric generating units and that it would be appropriate for EPA to concurrently propose performance standards from new and modified electric generating units, and emissions guidelines for GHG emissions from existing affected electric generating units. As noted above (p. 15), MidAmerican disagrees that there are cost-effective post-combustion control strategies for reducing greenhouse gas emissions, and only limited efficiency improvements are commercially available at this time. EPA indicated that the GHG standards are likely to apply to existing facilities starting in 2015 or 2016.

Figure 3 - Overview of MidAmerican's Environmental Control Projects

				NOx Emission Controls					SO_2 Emission Controls		Particulate Controls			Mercury (Hg) Control	CCB Compliance		
Unit	Year Installed	Regulatory Depreciation Life (non-OR)	Accredited Net MW Rating (100% Share)	Neural Network System	Low NOx Burners	Overfire Air	Selective Non-Catalytic Reduction	Selective Catalytic Reduction	Dry Scrubber	Wet Scrubber	Electrostatic Precipitator	Wet Scrubber	Baghouse	Activated Carbon Injection	(Dates shown are initial compliance projects in-service) Pond closure, wet-to-dry conversion, new facilities	Potential to Operate on Full Load Natural Gas	MEC/PAC Ownership
Neal 1	1964		135	Y	N/A	Y	Not Planned	Not Planned	Not Planned	Not Planned	Y - Hot	N/A	Not Planned	2014	2015	Y	100.00%
Neal 2	1972		295	Y	Y	Y	2014	Not Planned	2014	Not Planned	Y - Cold	N/A	2014	2014	2015	N/A	100.00%
Neal 3	1975		515	Y	Y	Y	2014	Not Planned	2014	N/A	Y - Cold	N/A	2014	2014	2015	N/A	72.00%
Neal 4	1979		644	Y	Y	Y	2013	Not Planned	2013	N/A	Y - Cold	N/A	2013	2014	Not Planned	N/A	40.57%
WSEC 1	1954		45	Not Planned	Y	Not Planned	Not Planned	Not Planned	Not Planned	Not Planned	Y - Hot	N/A	Not Planned	2014	2015	N/A	100.00%
WSEC 2	1958		88	Not Planned	Y	Y	Not Planned	Not Planned	Not Planned	Not Planned	Y - Hot	N/A	Not Planned	2014	2015	Y	100.00%
WSEC 3	1978		690	Y	Y	Y	Not Planned	Not Planned	Y	N/A	Y - Cold	N/A	Y	2014	2015	N/A	79.10%
WSEC 4	2007		800	Y	Y	Y	Not Planned	Y	Y	N/A	N/A	N/A	Y	Y	Not Planned	N/A	59.66%
Louisa	1983		745	Y	Y	Y	Not Planned	Not Planned	Y	N/A	Y - Hot	N/A	Y	2014	2014	N/A	88.00%
Riverside	1925/1961		130	Y	Y	Y	Not Planned	Not Planned	Not Planned	Not Planned	Y - Cold	N/A	Not Planned	2014	2015	Y	100.00%
Ottumwa	1981		710	Y	Y	Y	Not Planned	Not Planned	2014	N/A	Y - Hot	N/A	2014	2014	Not Planned	N/A	52.00%
Carbon 1	1954	2020	67	Not Planned	Not Planned	Not Planned	Not Planned	Not Planned	Not Planned	Not Planned	Y - Cold Side	-	Not Planned	Sorb Inj + Oxidizer	2015	Under Review	100.00%
Carbon 2	1957	2020	105	Not Planned	Not Planned	Not Planned	Not Planned	Not Planned	Not Planned	Not Planned	Y - Cold Side	-	Not Planned	Sorb Inj + Oxidizer	2015	Under Review	100.00%
Cholla 4	1981	2042	395	Not Planned	Y	Y	Not Planned	Not Planned	N/A	Y	N/A	-	Y	Coal Oxidizer	2015	Not Planned	100.00%
Colstrip 3	1984	2046	740	Not Planned	Y	Y	Not Planned	Not Planned	N/A	Y	N/A	Y	N/A	Y - Installed	2015	Not Planned	100.00%
Colstrip 4	1986	2046	740	Not Planned	Y	Y	Not Planned	Not Planned	N/A	Y	N/A	Y	N/A	Y - Installed	2015	Not Planned	100.00%
Craig 1	1980	2034	428	Not Planned	Y	Y	2014	Not Planned	N/A	Y	N/A	-	Y	Coal Oxidizer	2015	Not Planned	19.28%
Craig 2	1979	2034	428	Not Planned	Y	Y	2013	Not Planned	N/A	Y	N/A	-	Y	Coal Oxidizer	2015	Not Planned	19.28%
Dave Johnston 1	1958	2027	106	Not Planned	N	N	Not Planned	Not Planned	Not Planned	Not Planned	Y - Cold Side	-	Not Planned	Sorbent Injection	2015	Under Review	100.00%
Dave Johnston 2	1960	2027	106	Not Planned	N	N	Not Planned	Not Planned	Not Planned	Not Planned	Y - Cold Side	-	Not Planned	Sorbent Injection	2015	Under Review	100.00%
Dave Johnston 3	1964	2027	220	Not Planned	Y	Y	Not Planned	Not Planned	Y	N/A	Y - Cold Side	-	Y	Sorbent Injection	2015	Not Planned	100.00%
Dave Johnston 4	1972	2027	330	Not Planned	Y	Y	Not Planned	Not Planned	2012	N/A	Y - Cold Side	-	2012	Sorbent Injection	2015	Not Planned	100.00%
Hayden 1	1965	2030	184	Not Planned	Y	Y	Not Planned	2015	Y	N/A	N/A	-	Y	Sorbent Injection	2015	Not Planned	24.46%
Hayden 2	1976	2030	262	Not Planned	Y	Y	Not Planned	2016	Y	N/A	N/A	-	Y	Sorbent Injection	2015	Not Planned	12.60%
Hunter 1	1978	2042	430	Not Planned	2014	2014	Not Planned	Not Planned	N/A	Y	Y - Cold Side	-	2014	Coal Oxidizer	2015	Not Planned	93.75%
Hunter 2	1980	2042	430	Not Planned	2011	2011	Not Planned	2023	N/A	Y	Y - Cold Side	-	2011	Coal Oxidizer	2015	Not Planned	60.31%
Hunter 3	1983	2042	460	Not Planned	Y	Y	Not Planned	2024	N/A	Y	N/A	-	Y	Coal Oxidizer	2015	Not Planned	100.00%
Huntington 1	1977	2036	445	Not Planned	Y	Y	Not Planned	2023	N/A	Y	Y - Cold Side	-	Y	Coal Oxidizer	2015	Not Planned	100.00%
Huntington 2	1974	2036	450	Not Planned	Y	Y	Not Planned	Not Planned	N/A	Y	N/A	-	Y	Coal Oxidizer	2015	Not Planned	100.00%
Jim Bridger 1	1974	2037	530	Not Planned	Y	Y	Not Planned	2022	N/A	Y	Y - Cold Side	-	Not Planned	Sorb Inj + Oxidizer	2015	Not Planned	66.67%
Jim Bridger 2	1975	2037	527	Not Planned	Y	Y	Not Planned	2021	N/A	Y	Y - Cold Side	-	Not Planned	Sorb Inj + Oxidizer	2015	Not Planned	66.67%
Jim Bridger 3	1976	2037	530	Not Planned	Y	Y	Not Planned	2015	N/A	Y	Y - Cold Side	-	Not Planned	Sorb Inj + Oxidizer	2015	Not Planned	66.67%
Jim Bridger 4	1979	2037	530	Not Planned	Y	Y	Not Planned	2016	N/A	Y	Y - Cold Side	-	Not Planned	Sorb Inj + Oxidizer	2015	Not Planned	66.67%
Naughton 1	1963	2029	160	Not Planned	2012	2012	Not Planned	Not Planned	N/A	2012	Y - Cold Side	-	Not Planned	Sorb Inj + Oxidizer	2015	Not Planned	100.00%
Naughton 2	1968	2029	210	Not Planned	2011	2011	Not Planned	Not Planned	N/A	2011	Y - Cold Side	-	Not Planned	Sorb Inj + Oxidizer	2015	Not Planned	100.00%
Naughton 3	1971	2029	330	Not Planned	Y	Y	Not Planned	2014	N/A	Y	Y - Cold Side	-	2014	Sorb Inj + Oxidizer	2015	Not Planned	100.00%
Wyodak	1978	2039	335	Not Planned	2011	2011	Not Planned	Not Planned	Y	N/A	Y - Cold Side	-	2011	Sorbent Injection	2015	Not Planned	80.00%

American Thoracic Society

We help the world breathe PULMONARY • CRITICAL CARE • SLEEP

Statement of the American Thoracic Society
Before the Senate Environment and Public Works Committee
Presented by Alfred Munzer MD
On Wednesday, June 15, 2011

I am Dr. Alfred Munzer and I am a physician specializing in lung disease and practicing at Washington Adventist Hospital in Takoma Park, MD. I spend my days treating people with serious conditions like asthma; chronic obstructive pulmonary disease, or COPD; sarcoidosis; and a number of other serious respiratory diseases, many of which are unknown by the general public.

Through a combination of medications, interventional procedures and lifestyle modifications, I work with my patients to try to help control their respiratory disease. But there is one thing, neither I nor my patients can control and that is air pollution. Air pollution plays a major role in causing and exacerbating respiratory illnesses. From years of clinical experience, I know that when the DC area has a code orange or code red air pollution day, patients will suffer the effects. Those with asthma will experience acute exacerabations of their condition, making every breath they take more labored. And the emergency room at the hospital will be filled with patients in serious respiratory distress. In most these cases, my patients didn't do anything wrong or different: they just happened to be unlucky enough to breathe highly polluted air.

While I am not an air pollution researcher, my clinical experience of the impact air pollution on respiratory health is backed up by a countless peer reviewed studies in the United States. and abroad. The science documenting the adverse health effects air pollution has on human health is comprehensive, consistent, and compelling.

Unfortunately, that science is also under attack.

Industry regulated by the Environmental Protection Agency has started a campaign to discredit the research that is used to support EPA's regulations under the Clean Air Act. Some members of Congress appear to be taking up the mantra of discrediting or openly discounting the validity of EPA-sponsored research. This is a mistake and is a distraction from what we all should be focusing on:reducing air pollution to improve everyone's health.

The EPA's science is sound in its methodology and strong in its conclusions. But the EPA is not the only source of credible science that shows air pollution matters. Respected scientific agencies in the United States and around the globe have documented the adverse effects of air pollution.

The NIH has supported a number of studies that found that air pollution – particularly ozone and particulate matter – is bad for everyone's health. This is especially true for children.

Silverman and colleagues demonstrated warm weather patterns of ozone and $PM_{2.5}$ disproportionately affect children with asthma and appear responsible for severe attacks that could have been avoided.

Moore and colleagues in a California Air Resources Board funded study showed that current levels experienced in Southern California, ozone contributes to an increased risk of hospitalization for children with asthma.

In another study funded by the National Institutes of Health, Dr. Balmes and colleagues demonstrated that traffic related air pollution lead to measurable decreased in the lung function of adults.

The American Petroleum Institute has even contributed to the scientific literature. In an API funded study, Dr. Schelegle and colleagues demonstrated that in chamber studies exposures, ozone concentrations below 75 ppb decreases FEV1, a key measure of lung function, in healthy young adults.

I could go on about many studies supported by respected scientific agencies other than the EPA, but I hope my point is clear: air pollution impacts in a significant and negative way the health of Americans. This is not an opinion, it is a fact.

For the record, I have included a brief list of other important studies that all demonstrate that air pollution continues to be an important health issue in the United States. The most comprehensive listing of relevant studies can be found in the EPA Integrated Scientific Assessment document or the relevant the EPA criteria document.

In conclusion, the science is consistent and comprehensive and comes from multiple creditable sources, including the EPA. I hope Congress and the EPA can put to rest questions about the scientific validity of air pollution studies and start focusing our attention at the real problem: how best to move forward with eliminating the threat posed by air pollution.

I would be happy to answer any questions.

Selected References

TOPIC: Air pollution makes asthma worse, especially in children

Ambient ozone concentrations cause increased hospitalizations for asthma in children: an 18-year study in Southern California. Environ Health Perspect. 2008 Aug;116(8):1063-70. Moore K, Neugebauer R, Lurmann F, Hall J, Brajer V, Alcorn S, Tager I.

Key Finding: Ozone contributes to an increased risk of hospitalization for children with asthma living in California.

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TOPIC: Air pollution can kill you

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Key Finding: Residential ambient air pollution exposures were associated with mortality.

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TOPIC: Ozone adversely impacts lung function

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Key Finding: Air pollution alters lung function in children.

Chronic exposure to ambient ozone and lung function in young adults. Epidemiology. 2005 Nov;16(6):751-9. Tager IB, Balmes J, Lurmann F, Ngo L, Alcorn S, Künzli N.

Key Finding: Air pollution alters lung function in you adults who are near peak lifetime lung function.

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Key Finding: Ozone concentrations below 75 ppb decreases FEV1 in health young adults.